



In Collaboration with Serbian Association of Small Animal Practitioners (SASAP)

PROCEEDINGS

October 10–12, 2024

Sava Center, Belgrade

Advancing the veterinary profession in Eastern Europe

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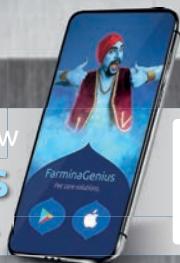
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Dear EERVC Delegate,

Thank You for being part of this year EERVC and welcome to Belgrade for the 7th Eastern European Regional Veterinary Conference.

The EERVC offers You cutting-edge international speakers, the largest regional and international trade exhibition and unparalleled opportunities for professional networking during an affordable, high-quality, 3 – day annual meeting.

EERVC Board tries to follow your suggestions when considering the speakers and their topics. Speakers have been selected by recommendation and their expertise. Lectures cover all aspects of small animal medicine and surgery, as well as topics of current interest for the profession.

On behalf of the EERVC Board, I would like to thank our exhibitors and sponsors, whose support ensures we can continue to deliver high quality and affordable CPD in this region. The trade exhibition at EERVC 2024 is a fantastic place to discover the newest products from industry, including the very latest innovations and services focused on the needs of the small animal veterinarian. We recommend that You explore the trade exhibition and make the most of the veterinary industry expertise and their products.

The EERVC Organizing Team have prepared lots of great opportunities for networking including famously known EERVC Party on Friday and more surprises. Do not miss none of them!!

My personal thanks go to the EERVC Project Board and the founding partners from the Small Animal Veterinary Associations of Croatia, Serbia and the British Small Animal Veterinary Association. The team have worked extremely hard to continue this ambitious mission of advancing the veterinary profession in Eastern Europe and beyond.

In 2016, EERVC started its journey in Belgrade and we decided to come back 8 years later. The start was challenging but all this with an overall positive emotion. One day we may write a novel about it. First and foremost, it is the people that make up the soul of Belgrade. People whose hearts remain young regardless of their birth year. People who will offer an open heart and a warm welcome to its visitors. People who proudly represent a better kind of Belgrade and will want you to remember it and come back with a smile. The kind of Belgrade EERVC will always belong to, wherever it may roam.

The EERVC Board thank every individual delegate, and we are happy to host you in Belgrade!



Denis Novak DVM MRCVS
EERVC Chairman

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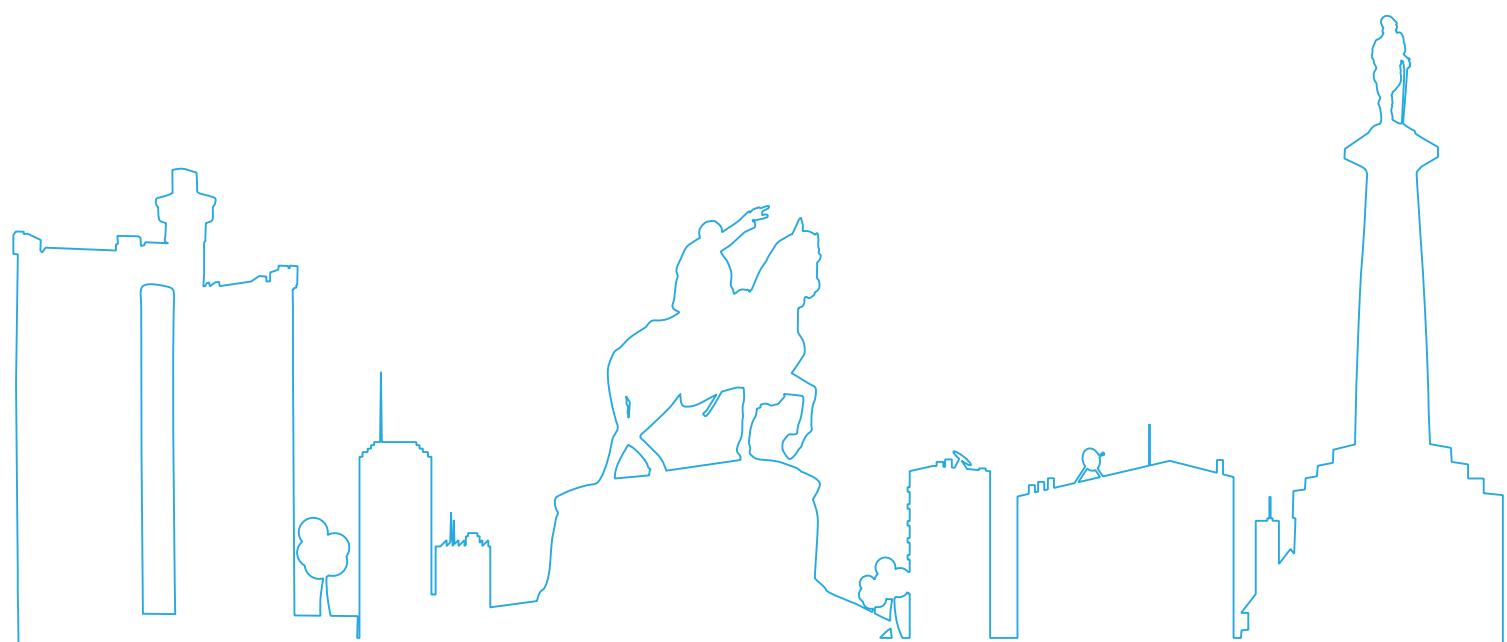
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EERVC is a not-for-profit organization that will reinvest all conference profits into the annual event and improving professional standards in the region. EERVC is managed by a Project Board joined together under a European Economic Interest Group partnership. The founding partners in this Board are the Small Animal Veterinary Associations of Croatia and Serbia, working together with the British Small Animal Veterinary Association (BSAVA). BSAVA has invested in EERVC as part of its remit as a registered charity and is offering its expertise gained from 60 years of BSAVA Congress.

ANESTHESIOLOGY





Paulo Steagall (Hong Kong)

DVM, Msc, PhD, DACVAA

EERVC 2024 Lectures

1. What's new in anesthesia and pain management in 2024?
2. I love watching cat videos: an interactive session on acute pain behaviors/assessment in cats
3. My favorite ten tricks about anesthesia in small animal practice
4. Case discussion and anesthetic protocols for common conditions in dogs and cats

Dr. Paulo Steagall is a Professor of Veterinary Anesthesiology and Pain Management at the City University of Hong Kong and the Université de Montréal. He is a board-certified specialist by the American College of Veterinary Anesthesia and Analgesia.

He is the head of a clinical research laboratory dedicated to improving pain management and animal welfare with cutting-edge research in pain assessment with emphasis on cats including the Feline Grimace Scale (www.felinegrimacescale.com). He has published over 130 scientific articles on pain management and the book "Feline Anesthesia and Pain Management" while lecturing internationally.

WHAT'S NEW IN ANESTHESIA AND PAIN MANAGEMENT IN 2024?

Paulo Steagall MV, MS, PhD, DACVAA

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This lecture will give an overview of current practical techniques and drug protocols for small anaesthesia and pain management in 2024 that are readily applicable in clinical practice. The speaker will present and evolve about the 2024 ISFM and AAFP consensus guidelines on the use of long-term NSAID in cats. The WSAVA Pain Certificate will be presented to the audience as a great opportunity to become certified in this subject or dive into pain management with a high-quality free CPD. The presentation will discuss the potential differences between gabapentin and pregabalin: is it worth the price difference? The use of gabapentinoids in clinical practice will also involve dose adjustments of gabapentin in cats with chronic kidney disease. How about new information in kittens and the use of the Feline Grimace Scale? Yes, that too. We will then move to discuss the use of opioid-free or opioid-sparing drug protocols for spay-neuter programs: is it possible or all cats/dogs will be painful without the use of perioperative opioid analgesia? The use of a new sedative vatinoxan in combination with medetomidine in dogs will be discussed – background information, reasons behind it and what the differences are when compared with dexmedetomidine, for example. Well, we could not local anaesthetic blocks – a quick review of the use of intraperitoneal, incisional and intratesticular blocks will be presented including videos. The lecture will finally present brand-new information about anaesthetic mortality in dogs and cats and novel tools to be used in practice such as monoclonal antibodies targeting nerve growth factor and AI-powered pain assessment tools.

I LOVE WATCHING CAT VIDEOS: AN INTERACTIVE SESSION ON ACUTE PAIN BEHAVIOURS/ ASSESSMENT IN CATS

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Nobody can deny the fact that watching cat videos is one of the best things in life particularly during procrastination. Well, there we go. In this lecture, we will have an interactive discussion about pain behaviours in cats, pain scoring systems (Glasgow feline composite pain scale, UNESP-Botucatu feline pain scale and Feline Grimace Scale) but we will certainly spend most of the time watching cat videos about acute pain assessment and behaviours. Please do not be surprised if you become a cat whisperer as a total of 30 videos will be shown illustrating different behaviours and what characterizes painful versus non-painful in the clinical setting, before and after surgery or the administration of analgesics. This lecture will present for the first time our new YouTube channel Cat Pain Management. (<https://www.youtube.com/@catpain-management>) providing an open-access library of these videos that anyone, anywhere in the world can have access. Examples of classic pain behaviours will include lowered head position, eye squinting, blepharospasm, head shaking, hissing, growling, repelling, depressed, non-weight bearing, abnormal gait, lying dorsoventrally with pelvic limbs extended/contracted, crouched/hunched-up position, feigned sleep, withdrawing/hiding, restlessness, attention to the wound and no attention to surroundings. Some normal and expected behaviours in pain-free cats will be also presented.

ANESTHESIA IN BRACHYCEPHALIC BREEDS: MINIMIZING COMPLICATIONS AND MORTALITY

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Oh, brachycephalic breeds...we have all got into trouble while anaesthetizing them. Is there a way to prevent complications and anaesthetic mortality in these breeds? Of course, there is. In this lecture, we will first discuss the brachycephalic obstructive airway syndrome and presenting some important data about anaesthesia and how airway surgery and its complications can affect mortality and what is the evidence

ANESTHESIOLOGY

that exists for the use of omeprazole and metoclopramide, for example. What do we do in cases of pulmonary oedema and gastroesophageal reflux (GER). By the way, should we use bicarbonate during post-GER lavage? We will dive into their susceptibility to heat stress, high vagal tone, corneal ulcerative disease and much more!

Preparation for anaesthesia is even more paramount in these breeds. What protocols should I consider? Is maropitant routinely used in these patients? Do we have to reduce doses and change routes of administration during premedication? The discussion will further present some information about anaesthetic agents and the prevalence of GER. The speaker will share his tips for anaesthetic recovery, most common complications, what to do in these cases and when to reintubate. Recovery is usually the most critical time and prevention is key to avoid issues. Several videos will be presented to illustrate specific comments and interventions during anaesthesia.

CASE DISCUSSION AND ANAESTHETIC PROTOCOLS FOR COMMON CONDITIONS IN DOGS AND CATS

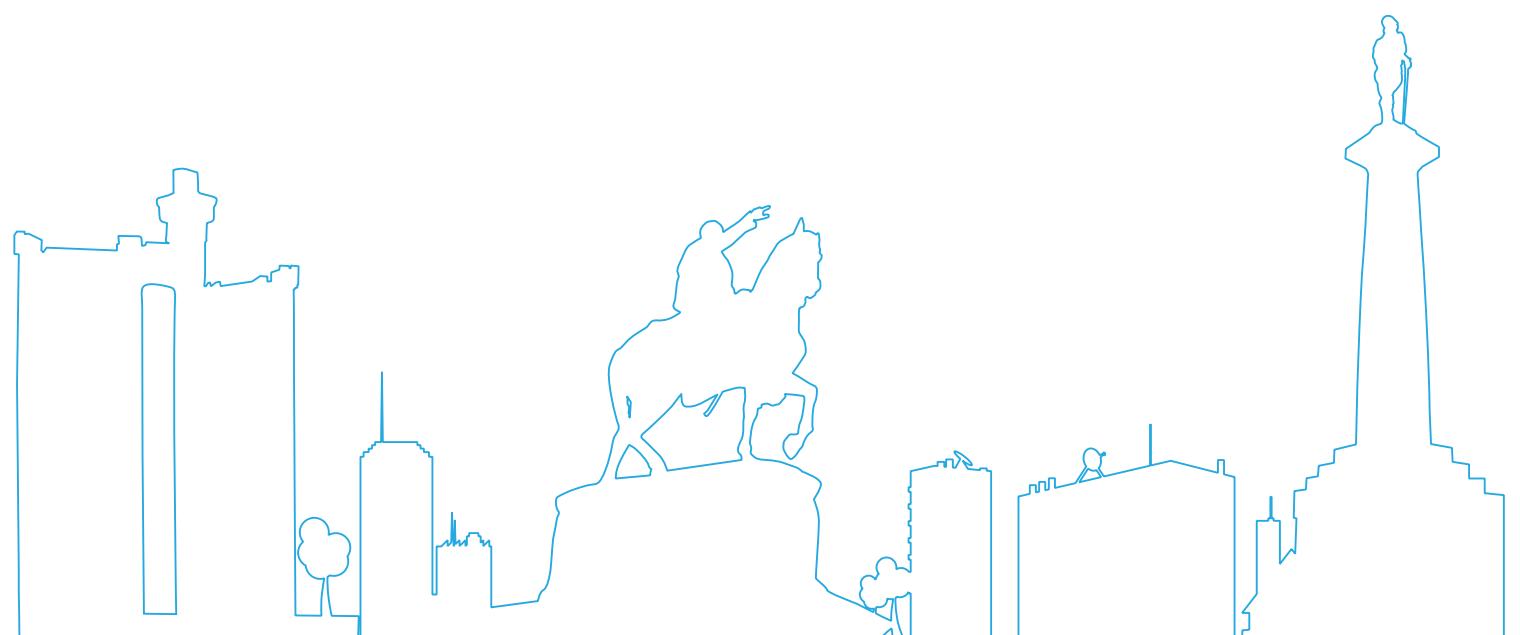
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"Please, just give me your anaesthetic protocols. I just want the doses and drug combinations. I don't want to hear your stories, she said". Well, it is true that in most of my lectures, people want to hear and take notes (or pictures) of drug protocols. However, times have evolved, and we now talk about the anaesthetic plan and a holistic approach during anaesthesia and surgery involving not only pain control but appropriate fluid therapy, control of nausea and vomiting and strategies to reduce fear and anxiety including friendly and empathetic handling of our patients. This lecture was built based on the successful FECAVA campaign on Best Basic Practices in Anaesthesia and Pain Management and its series of infographics and notes. The last infographic presents four common clinical presentations involving canine mastectomy, dentistry and neutering. One of these cases presents options for when opioids are not available. The aim of these case discussions is to help veterinarians to build up their anaesthetic protocols based on individual needs from "A to Z" including the preanesthetic preparation, monitoring, intubation, emergency, recovery and obvious all that you want: drug regimens. When to intubate? Do I need to give fluids every time? What are the basic strategies to respond to emergencies effectively? We will have an interactive and dynamic (meaning fun!) discussion on different approaches to clinical cases related to anaesthesia and pain management. We will also present basic practices that can come a long way in the clinical setting and things that veterinarians can easily incorporate in their routines. And when you do not have access to opioids, can we have alternatives such as NSAID, paracetamol, tramadol or gabapentinoids? We will present you the latest in the evidence about analgesic efficacy of these drugs.

BEHAVIOUR AND WELFARE





Gonçalo da Graça Pereira (Portugal)

DVM, MsC, PhD, Dip ECAWBM (BM), Dip ECAWBM (AWSEL)

EERVC 2024 Lectures

1. Skills for kittens and puppies
2. Cognitive Dysfunction Syndrome: can we stop ageing?
3. Are there emergencies in behaviour?
4. Noise reactivity: diagnosis and treatment
5. Dominance in dogs: myth or reality?
6. Hospitalisation and post-discharge – behavioural considerations for cats

Veterinary Degree at the Faculty of Veterinary Medicine in Lisbon. Master in Clinical Ethology and Animal Welfare at the Faculty of Veterinary from the Universidad Complutense de Madrid. PhD in Veterinary Science at the Institute of Biomedical Science Abel Salazar in O'Porto. EBVS specialist in Behavioural Medicine and Diplomate in Animal Welfare Science, Ethics and Legislation by the European College of Animal Welfare and Behavioural Medicine. Secretary of the Executive Committee from the European Board of Veterinary Specialisation (EBVS). Vice-President of the European Society of Veterinary Clinical Ethology (ESVCE). President (2017-2020) of the European College of Animal Welfare and Behavioural Medicine (ECAWBM). Founder and President (2011-2017) of PsiAnimal – Portuguese Association of Behavioural Therapy and Animal Welfare. Professor of Animal Behaviour, Welfare and Ethics both in the Veterinary Master and Veterinary Nurse Degree of the Egas Moniz School of Health and Science. Lecturer in Seminars, Conferences and Congresses national and internationally. Author, Co-author and Coordinator of several studies in animal behaviour and welfare science.

LIFE SKILLS FOR KITTENS AND PUPPIES

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INTRODUCTION

One of the most important reasons for dogs and cats relinquishment and abandonment is behavioural problems. Prevention is the most important tool to avoid this worldwide recognised problem. But a behavioural problem seen from the animal's perspective can only be a way to survive or to copy with the environment. There are dogs that immediately run away from visitors, or cats that a minimal change in the environment leads to a high stress level that start spraying. The tolerance of the caregivers is totally different among all, but repulsion behaviour ("aggression") is one of the less tolerated inadequate behaviour. And this behaviour is generally not well interpreted. The main emotional reasons for animals to show this type of behavioural manifestations are: fear/anxiety, frustration and/or pain. All these emotional motivations are known as protective emotions and the better the animal develop in early life stages, the better they can cope with them. So, having a confident cat or dog will bring to the caregiver the chance to have a good relationship with his animal, having a pet with the adequate skills for a lifetime. But how can we help the caregivers achieving this task?

TRAINING AND INFORMING

There are many different approaches in this field where we could be useful. We should work with breeders, with petshops, with animal protection associations and especially with the caregivers. The 3 first mentioned are really important, as we'll see, because they are involved in raising and first experiences of kittens and puppies. But is the caregiver that will have the chance to consolidate the learning for life. So, there are formal training sessions for caregivers (but all the other mentioned can also attend), called kitten or puppy classes. In this training the caregiver has a chance to learn about animal's behaviour and to avoid problems in the future. But it's also when starts its training. Kersti Seksel (2004) developed a program of socialization, training and early education for kittens called *Kitten Kindy*[®].¹ During this program not only the relation between cat and caregiver will increase, but there is also an habituation to veterinary environment in a positive way¹. In these classes the caregiver can learn about the normal cat's behaviour in order that they can understand and avoid future problems¹. Generally the classes focus in the appropriate way to interact and play, but also to understand body posture and cat's language¹. Nevertheless, some colleagues suggest that during these classes, as the socialization period has already passed when they start, the advantages need to be more discussed. But, at least, informative kitten sessions can be organized at the clinic, with or without the kitten. Animals that are raised by people with knowledge show appropriate social behaviours and develop less behavioural problems². Apart from this "formal" training, the vet should also pass adequate information during the first consultations and at the waiting room can have posters and leaflets about animal behaviour and their needs.

SOCIALIZATION

The socialization period (3 to 8 weeks old) in cats is earlier and shorter when compared with puppies. But it is a biological window of opportunities where the kitten can learn how to play, hunt, hygiene and biting/scratching inhibition. This learning procedure is firstly learned with the mother, but the adequate manipulation by humans will be useful to have a confident cat with humans (20mn/day, with positive experiences with women, men, children). A similar situation happens for puppies and if we don't use this period to socialize than fear reactions can happen in the future.

The separation from mother should never happen before 8 weeks (with some possible exception that should be well discussed and supported by a behaviourist), because it is really important what she has to teach to the litter. In the cats' case is well studied that those that were weaned and separated from mother too early are more reactive and more phobic, having a higher predisposition to frustration, but also have using more their mouth and paws (bite/scratch). A very similar situation can be seen in puppies that did not receive enough information from the mother and have difficulties to control themselves. The socialization, especially on puppies, with other animals (different sizes, colours, etc) and people (different ages, walking patterns, etc) is also very important during this phase. But this relation with other species is fragile and it is important that the contact is maintained along time.

JUVENILE PERIOD

The majority of kittens and puppies come to the clinic about 8 weeks to start the vaccination, but careful because the socialization period in kittens is almost gone. Nevertheless we still have many things to teach. It is during this period that the cat organizes its territory and develops the affiliative behaviours within the social group (allogrooming). So, we have to create a secure, stable and well organized territory with enough resources. At the same time the caregiver has to find some free time to play, being totally forbidden to do it with hands, feet or parts of the body. Teaching verbal cues ("sit", "lay", "give me five", etc) should start now, as this is very useful to prevent and support emotional changes in the future. When positive reinforcement and adequate training tools are used by the caregivers, the relationship with the animal improves a lot. Habituation to different type of manipulation is very important. The caregiver should associate, in a positive way, the manipulation of ears, mouth, hair and teeth brush, cut claws, but also handling and holding in different positions. Everything must be done gradually. The same should be done with different sounds, as the vacuum cleaner. The habituation to the cat carrier should be done the earliest, as possible, as well as the experience at the vet must be very positive.

CONCLUSIONS

At the end, how can caregivers prevent undesired behaviours and have confident animals? 1. Reinforcing the desired behaviour; 2. Ignoring (negative punishment) undesired behaviour; 3. Guiding the undesired behaviour (as predation) to adequate targets (as toys). Finally the animal's initiatives should be respected and reinforced (if desired). This is the reason that the caregiver needs to understand animal's body posture to clearly interpret and understand his language. To have a confident animal, the skills need to be taught early in life and there is a role for each one of us (from caregivers to veterinarians, but also the important role of the kitten or puppy's mother). All together we can work to have more confident adult animals in the future, making their life easier ensuring its welfare.

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COGNITIVE DYSFUNCTION SYNDROME: CAN WE STOP AGEING?**Gonçalo da Graça Pereira**

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INTRODUCTION

The Cognitive Dysfunction Syndrome (CDS) is a neurodegenerative pathology in older dogs and cats that is characterized by a cognitive gradual decline and increase of the brain disease.¹⁻⁸ As many pathologies, including the CDS, can present firstly behavioural signs, its early identification is important to the health and welfare of the animal. Only based in clinical signs, the CDS has been traditionally diagnosed in dogs with 11 years old or more. Nevertheless, dogs that present changes in spatial memory can be seen between 6 and 18 years old.⁸ This function is highly dependent on the frontal lobe, where there is an atrophy and accumulation of beta-amyloid substance before other cerebral areas.⁵ In cats, based in more limited data, the cognitive and motor performance start to decrease approximately at 10-11 years old, but the functional changes of nucleus caudate neurons was already observed from 6-7 years old.⁹⁻¹¹

The early diagnose offer an opportunity of treatment as well as prevention of complications. The behavioural decline can be slowed, the longevity increased and welfare assured. To diagnose CDS, veterinarians usually use the history intake and adequate anamnesis. Only with an adequate anamnesis with correct information in take through a detailed questionnaire, the diagnose can be done and found the different stages of this pathology. Several North-American associations (AAHA, 2005; AAFP, 2008) mention in their guidelines to the senior patient that an annual exam with a blood check-up should be done in average adult age dogs and twice a year in seniors. Apart from this, the guardian should always be questioned about changes that they can see both in physical and behavioural (mental) health. Only when the veterinarian show a proactive attitude and ask the guardians about specific signs, usually those are not reported as

they are seen as insignificant or not treatable. In a study with senior cats, 154 guardians from cats with 11 or more years old were asked about signs of CDS. After exclude 19 cats due "medical" problems, 35% had a diagnose of CDS; the same diagnose was found in 28% from 95 cats with ages between 11 and 15 years old and in 50% of 46 cats with more than 15 years old.²

So, the diagnose is initially based in clinical signs represented by the acronym DISHAA: a) **D**isorientation; b) **I**nteraction (changes in the interaction with people or other animals); c) **S**leep (change in sleep-aware cycle); and d) **H**ousesoiling (periuria). As the activity level can also show changes while time pass in animals with CDS, it was added an A from **A**ctivity. More recently another A, from **A**nxiety has been added to this acronym as generally these dogs have the fear/anxiety emotion-motivational system activated due all the clinical signs presented and brain functional changes. In table 1 (checklist for CDS, adaptaded from Landsberg et al.¹), can be found a summary of these signs, and this table can be adapted to a questionnaire to be filled in by every guardians while waiting for the consultation. In cats, the clinicians tend to assume that the CDS has similar signs to those observed in dogs.

Once the signs are identified, every clinical condition that can cause or contribute to these signs should be excluded. As geriatric animal have frequently concomitant multiple diseases, the diagnose of a "medical" problem does not exclude the possibility of the CDS. Although behavioural changes are very common associated with cerebral aging process or CDS, other pathologies with non-behavioural origin have to be excluded in a first phase.¹

AGING AND ITS EFFECT IN THE BRAIN

In dogs there is a decrease of the frontal volume, increase of ventricular size and there are evidences of meningitis calcification, desmyelinization, neuroaxonal degeneration and reduction of neurons number.^{6,7} There is also an increase of the activity of Mono-Amino-Oxidase B (MAOB).¹⁵ In cats there are also evidences of cerebral pathology associated to age, including neurons losing, cerebral atrophy, thickness of sulci, increase of ventricular size, but none of them are so marked as in dogs.^{2,12} A decline of the colinergic system can also be identified in dogs and cats and this can contribute to the cognitive decline and, possible as well, to the motor function, but also to the alterations seen in REM phase of sleep.^{12,15-18} Both in dogs and cats, as well as humans, there is an increase of the accumulation of diffuse plaques of beta-amyloid and perivascular infiltrates.²⁻⁵ Nevertheless, this plaques, comparing to dogs and humans, are much more diffuse in cats.^{2,3,13,19,20} But the link between the beta-amyloid plaques and the CSD in cats is still in discussion, having studies showing a positive correlation^{5,13,20} and others that don't show it.²¹

STRATEGIES TO "TREAT" CDS

Now that we saw some of the effects of brain aging, that are the origin of signs that we see in our patients with CDS, there is a specific need to see which are the strategies to delay this process. We want to improve the cognition of these animals, only delaying this process. Unfortunately the youth elixir was not yet found and we know that we will not stop this process, but only delay it.

a) Cognitive Stimulation

When we have an animal with CDS, the treatment with diet, drugs or supplements can be very useful in improving the signs and delaying the progression of this pathology and, above all, making the animal more tolerant to behavioural treatment/modification. Studies with dogs demonstrated that this is not simply an essential component in keeping the quality of life, but also an integrate part to maintain the cognitive function, using training, playing and/or exercise.²¹ This fact is similar to recent findings in humans that shown that the education, cerebral and physical exercises delay the beginning of dementia. Moreover, the more recent scientific knowledge, namely studies on cerebral neuroplasticity from the National Institute on Aging, says that the combination between senior dogs and new "tricks" is essential for the wellbeing and mental health of this animals. The brain is an organ with plasticity and can be re-trained. When talking about animal training, the development of a plan that include cognitive stimulation, simple and that involve minimal stress is a strategy to follow²⁶ not only in dogs, but in cats as well! The idea that the learning should only be requested during the early phases of the animal's development should be left behind and seen as a continuous process during the whole life independently of the specie, being detrimental in this phase of life.

b) Pharmacotherapy

Before starting any medication, an health check-up must be done and also checked if any other medication is being given (or supplementation) with which can have any lateral side effects.

There are currently no drugs approved for cats. For that reason, the possibility of showing improvement on signs should be always analysed taking in attention potential side effects. Nevertheless, has been

reported improvements in cases of CDS in cats with the use of Selegiline^{2,23} in the same dose as for dogs. The dose used vary from 0,5-1,0mg/kg, in the morning. Only some gastrointestinal signs were reported at the beginning of administration. Selegiline is an inhibitor of the Mono-Amino-Oxidase B (MAOB).^{13,22} It can increase the level of dopamine and other catecolamines in the cortex and hippocampus and has demonstrated improvements in the cognitive function (both in laboratory and clinically), having at the same time an important role in the free-radicals' accumulation decrease in the brain. There should be a specially care with the simultaneous use of other MAO inhibitors as amitraz or other drugs that increase the concentration of serotonin (due the serotoninergic syndrom!).

Propentophiline is a potent vasodilator that increase the blood output to muscles and brain. For that reason it is licenced in some countries to treat apathy, letargy and depressive behaviour in the senior dog. It has been reported improvements in cats in a dose of ¼ of a pill of 50mg per day.⁴

c) Nutritional and dietary treatment

Another strategy to delay the signs of CDS is the use of food supplements to increase the antioxidants defences and reduce the toxic effects of free radicals. In humans several studies show that the dietary management can reduce the risk or delay the beginning of dementia. For example, the high consume of fruits and vegetables, nuts, integral grains and vitamine E and C can reduce the risk of cognitive decline and dementia.^{24,25}

In humans, Alzheimer's Disease has been associated with many risk factors such as reduced cerebral glucose metabolism, deficiency in docosahexaenoic acid (DHA), chronic oxidative stress and chronic inflammation. With these factors in mind, PURINA® developed nutritional solutions (with medium chain triglycerides – MCT – and brain protection blend – BPB) that confirmed its benefits in managing clinical signs of CDS in dogs and enhancing cognitive function and slowing age-induced cognitive decline²⁷.

Depois de 2 anos, o grupo controlo (sem enriquecimento ambiental e sem a dieta em estudo) mostrou uma imento do animal, mas sim

d) Complementary Treatment

In conjunction with medication for CDS can be combined other drugs directed to specific clinical signs (always checking possible medication interactions).

In patients with changes on the sleep-wake cycle it can be importante to use an association with a benzodiazepine. Once lorazepam, oxazepam and clonazepam have not any active intermedios metabolites, they can be a good secure choice among the different benzodiazepines in patients that have alterations in the liver function.

Natural therapeutics that promote sleep and others that reduce the anxiety can be considered, including melatonine, aromotherapy and some nutraceutics, as alfa-caseine (Zylkéne®, Vetoquinol®) or a diet supplemented with this nutraceutic (Calm Diet®, Royal Canin®), the L-tryptophane combined with L-teanine (Calmex®, VetPlus®). Apart from all this, even not having studies done in this age of patients, since that pheromones help to decrease anxiety and stress, it can be a huge supportive help in these patients. The use of Feliway® (CEVA®), since its scientific proved efficacy in several studies, can be used to reduce anxiety. At the same time, reducing anxiety can promote the welfare and behavioural improvements observed in senior animals.

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DOMINANCE IN DOGS: MYTH OR REALITY?

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INTRODUCTION

In the past, much of the dog's behaviour and organization was interpreted in a very simplistic way based on the dominance hierarchy. Dogs were believed to be motivated to achieve a higher status in relation to individuals in the group, both dogs and humans, using coercive strategies such as aggression to achieve this end. At present, we know that the assumptions on which this theory is based are fundamentally false. We better understand how the brain works and how animals learn, which allows us to develop a better understanding of why certain behavioural manifestations used by dogs, such as aggression. The problem of using the concept of dominance in more complex animals became evident, as this interpretation was not consistent in different situations. In other words, while animal A is more likely to win a resource at one time, animal B might do so at another. In addition, in gregarious species, other factors appear to influence the outcome of an interaction over a resource dispute. For example, the outcome of competition for food varies with how hungry each one is. In other words, the value and availability of the resource are determining factors in the choice of strategy to be adopted and in the outcome of the confrontation. The ability to identify and learn the particular signs that can predict the behaviour of each individual in different situations will allow knowing the outcome of each encounter and how they should interact¹.

ORIGIN OF THE DOMINANCE THEORY

Since the wolf is the ancestor of the domestic dog, experts in animal behaviour suggested that, among dogs, social groups like those observed in wolves could arise, and that the formation of these groups was based on the "desire" of each individual in being the "leader" or the "alpha" of the group. The hierarchical status of an individual in the social organization of the group resulted from competitive success in direct confrontations with peers. This interpretation of dogs has become so evident and common that it has become the basis for interpreting the interactions between dogs and people, on the assumption that dogs also consider people as competitors in the struggle for social status. This assumption was used to explain behaviours that range from aggression, to seeking attention, to destruction, and even failure to respond to a call¹. If it is assumed that the dog's behaviour is motivated by a desire to control or "dominate" its guardian, it is concluded that, to face the problem, the guardian must establish "dominance" over the dog. This interpretation of the behaviour of dogs favored the development of training techniques based on positive punishment, where coercion is used to "show the dog who is the boss"².

RESULTS OF RECENT STUDIES: NEW INTERPRETATION APPROACHES

The most recent investigations on natural populations (in the wild) of wolves suggest that the grouping is based on the cooperation of operative family groups, where a breeding pair produces dogs and the other family members help in raising them³. Parents guide the litter in the development of social and hunting skills, but social organization arises through parent-litter relationships rather than supposedly competitive or aggressive encounters⁴. In these groups there is no "alpha" achieved by force or aggression⁴, and there is no evidence that wolves, as individuals, retain the title of "alpha" throughout life based on some "dominant trait"⁵. Aggressive behaviour is very rare in stable groups⁶, and when it occurs, it is flexible, depending on individual circumstances. The typical dominance hierarchy on which the social structure is supposed to be based does not seem to occur naturally in wolves (it should be noted that the studies on which this theory was based, carried out in the 1970s, were in captive wolves), therefore, the possibility of occurring in dogs, as descendants of the wolf, is a very poor argument⁷.

In addition, studies on wild dogs^{8,9} suggest that domestication significantly altered the social behaviour of this species when compared to its ancestors. In the free-living groups, feral dogs do not associate in strict family groups, there is no reproductive restriction, and apparently, they do not present any pyramidal structure based on a breeding pair and their offspring. Interactions between individuals are much more fluid, and are based more on circumstances, sexual cycles, and previous learning of the behaviour of other individuals¹.

Since neither natural groups of wolves nor groups of feral dogs appear to adopt a linear hierarchical social structure that has traditionally been attributed, the assumption that the behaviour of domestic dogs is

influenced by the desire to ascend in that structure is difficult to corroborate. In fact, recent investigations¹⁰ suggest that groups of domestic dogs do not form social groups that can be interpreted based on a dominance hierarchy.

PROBLEMS USING OUTDATED DOMINANCE THEORY

The real problem when it is assumed that the dog shows a behaviour because it has the "ultimate plan" to reach its leader status, is that it conditions the way the guardians educate and respond to their dogs. If guardians believe that their dog takes steps to "achieve status", "control" or "be the boss", they naturally tend to use coercion techniques. These techniques, used to inhibit this supposed behaviour, induce a negative emotional state (for example, fear or anxiety) that can have side effects, such as inducing unwanted behaviours and/or compromising well-being¹. Regrettably, the concept of "dominance" is too rooted in historical scientific literature and in the public consciousness, mainly due to the incorrect message conveyed by the media and some television programmes. Although most coaches and ethologists no longer think that way, there are still some authors¹¹ in the field of ethology, interpreting signs of aggression as dominance, since their definitions are based on ancient literature, tending to perpetuate this obsolete theory.

One of the most relevant characteristics of a gregarious species is the ability of individuals to communicate, both in terms of emission and interpretation of signals, which allows the adjustment of behaviour according to the situation¹². It is thus clear that social interactions within the group and their continuity depend on the communication skills of its members¹³. The assumption that dogs' behavioural responses to social interactions are innate, such as "dominance", ignores and underestimates their ability to learn and integrate complex associations. Dogs are clearly capable of learning about the concrete consequences of social interactions¹⁴, and it is important to recognize this fact when examining the development of social interactions. Each animal reads the signals of the others and learns about the possible meaning and consequences in different circumstances. In addition, every time a dog meets another, he will learn through the consequences of its own behaviour by the responses of others¹. To better elucidate this concept, the following example may be useful. Dogs go through a period of observation and evaluation when they first see an unfamiliar dog in the park. They may have had many previous experiences with dogs of all sizes, and they use all the information obtained in previous encounters, namely to communicate¹⁰. But when they are in front of a dog they see for the first time, the anxiety gradually increases because they don't know how the other will respond. Gradually they will have a more tense alert posture and show sudden jerky movements to gather information about the other. In contrast, if they already know each other, they are able to predict the likely response by reacting according to an already lived experience. Depending on what they have learned, they can go straight to play when they meet, ignore each other or even attack each other. Behaviour problems that occur among dogs are relatively common and are produced through this same learning process, which may be generalized to other individuals or contexts with similar characteristics. For example, a dog that shows aggression towards other dogs must initially have had an unpleasant experience with other dogs, and aggression is supposed to be an effective strategy to avoid the perceived threat. Likewise, a puppy who lives with an older, more tolerant dog may learn that the most effective way to achieve playful interaction is to run and jump to another dog! This behaviour will cause problems when the puppy starts interacting with other dogs that don't appreciate this greeting. Therefore, when dealing with these cases, the behaviourist must firstly obtain specific information about previous experiences, so that the reason for the development of this behaviour can be identified¹. Obviously, if dogs act with each other based on complex associative learning, there is absolutely no reason to suppose that they do it any differently with us. Indeed, a series of recent investigations¹⁵ into the dog's ability to learn and interpret specific human signals compared to its ancestral species, the wolf, reveal remarkable skills and support this theory. However, it is a mistake to think that they see people as other dogs, but as they develop into a human "family", they will learn about all the things we do with them, just as they would with other dogs. So, for example, they can learn that when a person smiles, or speaks in a certain tone of voice, it usually predicts a good outcome, and they behave accordingly (wag their tail, run, etc). But they can also learn that if certain people raise their voice, with dilated pupils and put their hand on their neck, that predicts a bad outcome. Once again, they can learn the appropriate response to resolve the situation in which they find themselves. Possible options may be to demonstrate appeasing behaviours, avoid contact by hiding, or use more aggressive manifestations to ward off the threat. Any of these options that result in success (that is, avoid the threat) is reinforced and is likely to present it on future occasions¹.

PROBLEMS ARISING FROM THE USE OF POSITIVE PUNISHMENT (TO MAKE IT EASIER, IN THIS TEXT, IT WILL BE SIMPLY CALLED AS PUNISHMENT)

Punishment tends to be an emotive word, although scientifically it only means a way of decreasing the possibility of repetition of a behaviour. Therefore, depending on the animal's characteristics and experience, and the trainer's decisions, a "positive punishment" can range from a mild "no" to an extreme aversive stimulus, such as a strangling, spikes, prong or shock collar. Punishment has been used in dog training since the beginning of domestication. Although these techniques based on inducing fear through pain have been used for a long time, it does not necessarily mean that they are the best option in terms of effectiveness and animal welfare. Indeed, training dogs using these techniques entails a series of risks¹. These are:

- Increase fear and anxiety about the situations in which it is used;
- Decrease the dog's ability to learn;
- Possibility of associating the punishment, which causes fear, with certain contexts, like the presence of other dogs or humans;
- Inhibition of behaviour, without modification of the underlying emotional response, increasing the possibility of problems in the future;
- Provoke a new avoidance response or a negative response;
- Fear prevents clear discernment of the intended response;
- Short-term or long-term evident physical pain.

CONCLUSION

There are widely used training techniques that do not require the use of punishment, eliminating the need to use techniques that affect the dogs' welfare. The comparison between the safety and effectiveness of techniques based on reward or punishment should always be taken into account¹. We must have an accurate perception of the underlying motivation of a behaviour and likewise assess the risk of an aversive experience that could actually increase the severity of the behavioural problem or induce new ones. Due to the serious risks of using punishment-based techniques, professional behaviour specialists very rarely recommend the use of these techniques for behaviour modification¹. As veterinarians, behavioural medicine specialists or not, we all share a concern and responsibility for the well-being of our patients. Therefore, it is in our duty to change the behaviour of dogs without the need to use pain or fear, recommending appropriate training techniques, as well as trainers who follow ethical guidelines and ensure the animal's welfare, achieving success in treating behavioural problems.

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HOSPITALISATION AND POST DISCHARGE: BEHAVIOURAL CONSIDERATIONS FOR CATS

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When there is a strong reason to hospitalize a cat it is important that it must be a very careful decision as being away from their territory and safe environment can be detrimental, potentially delaying their recovery! However, there is also the strong belief that it's better for their health to hospitalize cats as it increases the chance of a better diagnosis and treatment. Nevertheless, we must consider the emotional and cognitive health of the cat as part of the hospitalization plan as well. Many times we are so focused on the physical health, that when the animal leaves the hospital, it's cured, but they can take away an emotional and cognitive (due the learning that happened during the hospitalisation) problem, and this can take a long time and effort to be resolved.

If these animals are fearful and uncertain about the outcome of their experience, is it realistic to expect them to be calm, quiet and co-operative with us as veterinary practices simply because we are working for their health and welfare? In a busy practice it is not unusual to have a "house full" of patients and feel under pressure to speed up our procedures and do our "good" work quickly, even if that results in us holding these animals in uncomfortable positions and using techniques that are motivated by the desire to get the job done! Some will say that cat friendly techniques take too long, but in reality respecting the emotional needs of our patients will lead to cooperation from the patient and make the process much faster in the future. The memory of the "bad experience" can lead the animal to anticipate that it will be "threatened" the next time it comes to the practice, resulting in protective behavioural manifestations that can result in more time to finish the procedure and practice staff using more forceful handling techniques, leading to more problems.

So, let me ask you: have you ever been hospitalised before? If your answer was yes (otherwise you can ask to another person you know that passed by this undesirable experience), what are the memories you carry with you? When you were almost sleeping, did a nurse come to give you an injection, a pill or even to take your blood pressure? What about the lights? And noises?? In fact the majority of people that passed through this experience one of the things that is easily remember is the sleep deprivation. Could we say that something similar is occurring during the hospitalisation of our animals? Yes, we can! And this is the reason why the same concerns and recommendations related to the waiting or consultation room should be taken in account if the cat has to be hospitalized at the practice. So, let's review some of these recommendations.

PHYSICAL CONSIDERATIONS

If it is not a cat only practice, than the hospitalisation ward should have a specific area for cats and another for dogs. If it is not possible to have separate wards, at least visual barriers should be created so that the cats can feel more secure (also think about visual barriers between cages). A blanket, specially if it has the household scent, could cover the cage (at least partially, as some cats like to see the outside as well – and this way can choose what prefers), giving extra protection to the cat that is inside.

LIGHT: If the lights are required to be turned on the whole night, this blanket, covering the front of the cage, can also be very important to increase darkness which is very important to improve the quality of sleeping.

TEMPERATURE: Another important reason to have separate wards for dogs and cats is related with the room temperature. This is very important for cats, as they feel more comfortable in higher temperatures when compared with dogs.

SOUNDS: It is important to be aware of potentially "scary" noises that exist in the hospital setting. Apart from obvious sounds, there are many others that must be considered. Water running or a disinfectant spray can be misinterpreted by the fearful cat as another cat hissing and motorised sounds of vacuum cleaners or clippers can be so stressful that the cat reacts with an immediate protective response. Background sounds, like the sounds of nature (though note the sounds of birds that can frustrate some cats!) or white noise, can hide some of these scary noises.

SMELLS AND PHEROMONES: Between treatments/exams, make sure that a proper cleaning is done, as many alarm or attack pheromones could be present in the air. Ideally the room should be ventilated, cleaned and then synthetic feline facial pheromones sprayed on the table before the next cat is examined. Synthetic pheromone diffusers should also be plugged in (if it's a multi-species practice, there can be one for dogs and one for cats, as they are specific for each species). Remember the importance of smells in cat's perspective (in addition to from pheromone) and practicing good hygiene and using products with neutral odours is fundamental.

INTERACTIONS

It is important for the veterinary staff to be seen as the cat's best friends during the hospitalisation! So, let's give the cats what they like most. Ask the caregivers to bring the favourite treats, favourite toys and blankets with home scent, to be used by the clinic team whenever this is possible. Cats' favourite treats are often things like tuna, wet food, liquid treats in a lick-e-lix, but there are many possibilities because each cat has its own preference. Some behaviourists recommend that food should be avoided when there is already fear existing, due emotional conflict with desire/seak and fear emotional motivation, so passive offering, to allow the cat choice, should be used. Apart from this, also think about the negative association that can be made between the treat's offering and the treatment during the hospitalization. This is also the reason why new food should not be started during hospitalization. When this is not possible because there is a specific need of a new diet, have different brands or a different flavour to take home.

Unpleasant situations should always be minimized. Usually, in our busy days, we only approach the hospitalized cat to examine or treat them, forgetting about the importance (if the cat likes it) to be petted and played with, creating a positive interaction. Take it as a major rule, with exceptions dependant on the case and the physical limitations of the clinic, that all treatments and exams should not be done inside the cage. The cage where the cat is hospitalised should be the safe area. The cat's carrier brought from home, can be used for these procedures. If the cat is in a negative (protective) emotional state, such as fear or anxiety, lifting the upper top of the carrier so it is slightly open, enables you to slide a towel in over the bottom half so that the cat is totally covered by the towel. The majority of the cats hide below the towel, feeling more secure, and allow a complete examination/treatment without the need for restraint.

Towels can be the veterinarian's best friend when handling cats. A clean towel should be used for each cat when they are anxious or require handling. The towel can be lightly heated in a microwave as cats often respond more to warmth. The examination/treatment should always start with the less invasive aspects first before the more invasive, and the same should be true for the handling used. Always start with minimal handling. For example, avoiding the use of restraint before and/or after an injection, will help to prevent the cat from anticipating negative interactions in the future (which would happen if restraint is seen as a sign that something painful is coming). Always use a new needle before the injection and never forget to lubricate the thermometer. Refrigerated medication should be taken from the fridge so that they can be close to environmental temperature before being injected. The necessary equipment (cotton, needles, syringes, etc) should always be prepared and positioned in an easily accessible location before the starting the treatment so that it can be easily accessed and used.

Once a cat is sick and impaired, it will be more sensitive to the surrounding environment. Each cat should have access to a space for elimination which is separate from the resting or hiding locations. If this separation of resources is not achieved the cat will be distressed and may display unusual or unwanted behaviours such as hiding in the litter tray or eliminating in the dry food bowl. Environmental optimisation leading to cognitive stimulation is also a way to decrease stress and anxiety.

POST DISCHARGE

Finally once the animal is better it will be returning home and once again it is important to prepare for this event. If there are any other cats in the household, a reintroduction plan should be given to the client.

- Contact between the cats should never be forced.
- The other cat(s) should be given time to get used to the scent of the arriving cat, before moving on to visual contact and finally direct contact.
- The returning cat should not be simply taken out from the carrier to go and greet the other(s) and it can help for them to stay inside the carrier covered with the towel /blanket for a while, which has the scent of the household group of cats on it (the duration depending on the individual and varying from case to case), giving time to see the other(s) cat(s) reaction to the arrival from the cat.

This process, as well as medications, should be written in the discharge advice the cat is sent home with. The process and treatment must have a clear and concise plan. Otherwise no matter how good your knowledge and advice there will be little to no chance of success.

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ARE THERE EMERGENCIES IN BEHAVIOUR?**Gonçalo da Graça Pereira**

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When we talk about emergencies we immediately think of a gastropexy of a dog, having premature ventricular contractions, at 2 am; or in a diabetic cat who has received a high dose of insulin and is convulsing at 4 am; while the nurse warns nervously that a dog that has been run over and a cat that fell from the window will arrive; and all this happening while a cat has been blocked in the waiting room for more than 12 hours. It's all this that makes our catecholamines increase... yet, there are those who love working in emergencies! But for those who don't like emergencies, nor the long-term effects of catecholamines on health, is behaviour the most appropriate specialty? In the case of behaviour, catecholamines will certainly increase due to other issues, such as: the lack of resilience of the caregivers, the time it took them to seek specialized help, the things they have already done to the animal in an attempt to solve a problem, mistaken expectations from tutors that leads them to give up before they start to see results, among many other reasons. But, in fact, I'm sure some of us have already received calls to the emergency service (or even on our own cell phone) because the caregiver is locked out on the street freezing, barefoot and half-dressed, because the dog "suddenly" tried to attack him and only had time to flee into the street. Or a screaming call because the two cats "suddenly" got into a fight, with urine and feces in between. Or even a caregiver who is on the way to the hospital because when separating the two dogs who "suddenly" started fighting, he was bitten in a "protected" area of his body.

These and many other situations that are reported to us as behavioural emergencies in fact are not! In most situations there is a long history, often invisible to the eyes of caregivers (especially the cats from cats), which cannot be forgotten. There are tutors who try to cover the "sun with a sieve" in the hope of finding miraculous solutions recommended by so many people, even the neighborhood café's employee, to avoid going to a professional. But, if on the one hand the behavioural problem becomes more evident or worsens, the guardians' tolerance also changes. Therefore, when this urgency came to us, the "suddenly" is not the real truth, because it has often been several years!

Still, if there is a "sudden" behavioural change, we can be aware of one of two situations. Or this "suddenly" is not due to a behavioural issue but rather to another physical or organic situation that may be responsible for this drastic change in the animal's emotional state – all diseases that cause pain are likely to cause a change severe behavioural (such as the examples already mentioned); or there is a traumatic event for the animal's emotional health that is the trigger for this behavioural change. In the first situation, as in any other behavioural change, we will always have to rule out all diseases of non-behavioural origin that could be the cause of this problem (with the exams that are necessary for each animal). While in the second, the responsible event must be analyzed and an emotional diagnosis made, with the behavioural and medical therapy that is appropriate for each case.

However, in these emergencies, an immediate (and urgent!) intervention plan may have to be applied with changes in the environment, with fast-acting pharmacological intervention and an individual behavioural plan. In some cases, especially when the behavioural response involves manifestations of repulsion ("aggression"), we may have to carry out a risk assessment and make decisions in favor of everyone's safety. Because these behavioural emergencies are really rare, the reasons for professional burnout in the area of behavioural medicine, as mentioned before, are closely linked to many other reasons than the animal itself. Even so, our actions and our emergency plans will be essential to guarantee the well-being of both the animals and their caregivers.

NOISE REACTIVITY: DIAGNOSE AND TREATMENT

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INTRODUCTION

Anxiety disorders, fear and phobias are among the most common behavioural problems of companion dogs. These problems include generalized anxiety, separation related problems and phobias of specific stimuli such as storms, fireworks, or other noises. In each of these disorders, affected dogs exist in a state of heightened arousal and distress. They may cause damage to their surroundings or themselves as an expression of their anxiety. As such, anxiety disorders represent an important welfare issue for affected dogs and may negatively impact the human-animal bond, becoming one of the major reasons for relinquishment of animals to shelters.

There are many possible causes for anxiety disorders and reactive behaviours to different stimuli. Among other causes, the following are the most commons: a) insufficient socialization (including impossibility to express normal behaviour and/or unpredictable social interactions); b) Central Nervous System fear pathways unregulated; c) Traumatic event (including severe or frequent positive punishment); d) Genetic predisposition; e) Pain (or other medical issues) and f) Cognitive decline.

Signs of light fear (subtle and commonly not understandable as fear by the caregivers) to severe uncontrollable fear (known as phobias) are common causes of referral to behavioural practices. The most commonly reported reported reactivity is to noise (also known as sound phobia), being fireworks and thunderstorms the most common triggers. Sometimes, dogs with separation related problems, can have its origin in sound phobias.

ASSESSMENT AND DIAGNOSE

We can consider 2 different phases that each animal can arrive to our consultation. To make it easier, it will be considered phase 1 and phase 2.

Phase 1

Animals show signs of fear only when exposed to a specific noise. During this event tries to hide. Usually, predicting the events, can engage in a coping strategy to control the behavioural expression of its emotional state.

Phase 2

Fear becomes more severe and the avoiding or predicting strategies fail. The sound is now experienced in a sensitising context that will lead to more serious complications. From these complications there are 2

that are very important. The first one is the excessive sensitivity to sounds, especially the unexpected ones. The other important complication is the generalisation that can occur in this phase. The generalisation can be related to individual sounds, but also to contexts and predictive cues associated to this trigger. These complications result in disruption of the daily life of the animal (and its caregiver).

It is really important to keep in mind that any phase 1 can progress to phase 2. Unexpected exposure, altering access to hiding places, impossibility to control the exposure, are some of the factors that can influence this progression.

MANAGEMENT OF NOISE RELATED BEHAVIOUR PROBLEMS

The aim of managing this behavioural problem is supporting the animal to develop coping strategies. To make a good management it is required a not simply environmental management (guarantee always access to hiding places or refuges), but also alterations in human interaction. Caregivers should never use positive punishment or even force the animal to face the sound that develop the fear signs. Instead, caregivers should act as a role model, as not bothered with the sounds and not worried. Ignoring the fearful animal is also something inadequate, but still being recommended. Giving support and asking for an alternative and incompatible behaviour (previous trained with positive reinforcement) will help the animal to move its emotional state to a positive one.

TREATMENT

Treatment is based in 3 strategies: behavioural modification, pharmacological intervention and pheromonotherapy.

Behavioural modification

Dessensitization and counter conditioning is the basis to treat this animals. However the knowledge of clients to apply an adequate plan is usually low. Thus, making sure the client understand exactly all the steps and have someone (trustable animal trainer or a behaviourist) to support the behavioural modification plan. There are many limitations in the treatment, and every step must be well previously thought to guarantee no mistakes can happen.

Pharmacological intervention

Drugs are appropriate when there is generalization, complications, cognitive impairment, affected welfare and the inducing event may cause a relapse or worsening of the problem.

Never use acepromazine as increase the sound sensitivity and reduce escape response, but also can cause disorientation and confusion, including may disinhibit aggression. In short term medication, benzodiazepines can be recommended (being diazepam or alprazolam the most suitable). There are advantages in its use: amnesic effect, anxiolytic properties and dose related sedation. However can lead to paradoxical effect with increase of excitability, cause disinhibition and impair learning. Transmucosal medetomidine (Sileo®) has been used with very good results around the world, being a great option to be used, but with caregivers that can apply it correctly. Benzodiazepines (as alprazolam, lorazepam, among others), alfa-2-agonists (as clonidine), gabapentine, pregabalin or trazadone can also be good options to use in acute situations and combined with long term medication.

In long term medication, Selegiline or Sertraline can be used. Selegiline can be used when there are patterns of behaviour that are inhibited or avoiding. Also in cases with profound generalization or high level of sensitivity. Sertraline is indicated also with high level of sensitivity. However when predominant feature is anxiety or significant panic elements.

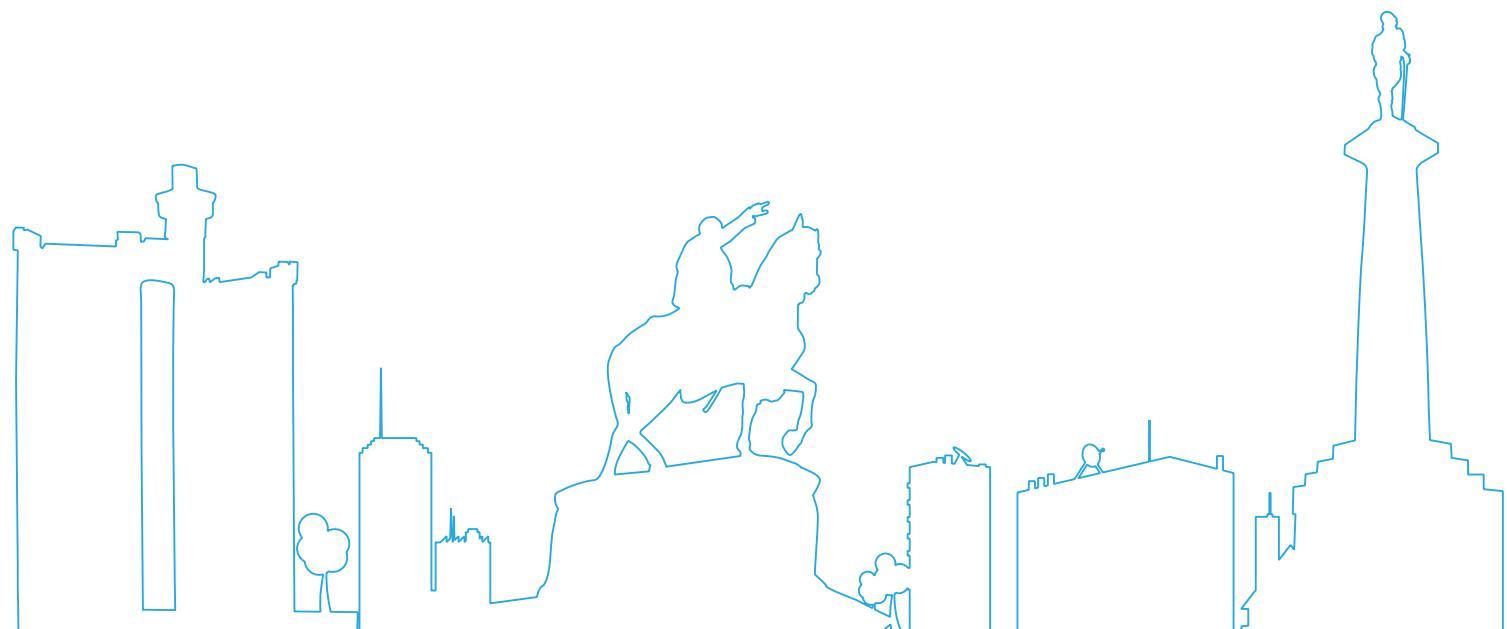
Pheromonotherapy

Pheromones can play a long term in long term treatment approaches. Adaptil® or Feliway® can increase the appeasing qualities of the environment supporting the behavioural modification plan.

CONCLUSIONS

From welfare perspective and quality of life, these animals deserve appropriate management and/or treatment. When left without intervention, these conditions can get worsen.

EMERGENCY AND CRITICAL CARE



EERVC 2024 Lectures

1. Emergency and critical care basics
2. Pathophysiology, clinical suspicion and diagnosis of shock... things you might have forgotten
3. The initial treatment of shock patients: Fluids (and beyond)
4. How can point of care ultrasound help you in shock patients
5. How can point of care ultrasound help you with gastro intestinal tract disorders
6. Septic peritonitis in companion animals: what we've learned from human medicine
7. Different types of dyspnea: Lessons learned from a good clinical exam
8. How can point of care ultrasound help you with dyspneic patients: Part A the pleural space
9. How can point of care ultrasound help you with dyspneic patients: Part B the pulmonary parenchyma
10. How can point of care ultrasound help you with dyspneic patients: Part C the focused cardiac ultrasound
11. The initial treatment of the dyspneic patient
12. Advanced treatment of the dyspneic patient: The basics from high flow to mechanical ventilation

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Kris Gommeren graduated from Ghent University in 2002, where he completed an internship and residency in internal medicine. In 2009, he became a diplomate of the European College of Internal Medicine and worked briefly in a referral practice before moving to the University of Liège, where he developed the ECC service. Since then, the emergency and intensive care service of the University of Liège has grown into a service with a team of 12 veterinarians, including 2 specialists and 4 residents in training, supplemented by 6 full-time ECC veterinary assistants, and assisted by rotating interns. The service sees more than 3,500 emergency rooms every year, and offers an average of 8 intensive care patients the best care every day whilst trying to provide students with the necessary basic knowledge about this field. In 2017, Kris became a diplomate of the European College of Veterinary Emergency and Critical Care and in the meantime completed a PhD on the effects of systemic inflammation on the cardiovascular system. Kris is former president of the European Society of Emergency and Critical Care (EVECCS), founding member of the Veterinary Emergency and Critical Care UltraSound (VECCUS) interest group, and co-founder of the Animal Blood Bank Benelux. His main areas of interest are point-of-care ultrasound, assessment of the cardiovascular system in critically ill patients, fluid therapy, and the assessment of volume status and fluid responsiveness.

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Dr. Céline Pouzot-Nevoret is a professor in Emergency and Critical Care, and the head of the SIAMU, the ICU of the Veterinary School of Lyon. She graduated from this School in 2002 and joined SIAMU the following year and completed a PhD on acute respiratory distress syndrome. She became an European board-certified specialist (Dip. ECVECC) in 2018. She is actively involved in European ECC as she is the European Emergency and Critical Care society (EVECCS) past-president and was the chair of the Congress organisation committee for several years. She is now member at large in the European College of Emergency and Critical Care (ECVECC). Her main fields of interest are the management of respiratory distress patient, use of POCUS in the ICU and haemorrhagic shock.

EMERGENCY AND CRITICAL CARE BASICS

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****Introduction****

Emergency and critical care in companion animals, particularly cats and dogs, is a challenging yet rewarding field that requires a thorough understanding of species-specific needs, rapid decision-making, and effective communication with pet owners. This proceeding will cover the fundamental principles clinicians should follow to ensure the best possible outcomes in emergency situations.

****Key Point 1: Species Differences in Disease Presentation and Treatment****

One of the foundational aspects of emergency and critical care in companion animals is recognizing that cats and dogs are distinct species with different physiological responses, disease susceptibilities, and therapeutic requirements. For example:

- **Disease Variability**: Cats are more prone to conditions like hypertrophic cardiomyopathy, while dogs frequently suffer from dilated cardiomyopathy. Both conditions affect the heart but may require different diagnostic and treatment approaches.
- **Symptom Presentation**: A similar disease may present differently between species. For instance, cats with respiratory distress often show open-mouth breathing, whereas dogs might present with a more overt cough.
- **Therapeutic Approaches**: The use of certain drugs, dosages, and treatment protocols may vary significantly between species. Clinicians must be aware of these differences to avoid adverse outcomes and tailor treatment plans appropriately.

****Key Point 2: Prioritizing Patient Survival in Emergency Situations****

In emergency care, the primary objective is to ensure the patient's survival. This often requires accepting the inherent risks associated with certain procedures or therapies. Clinicians should conduct a risk-benefit analysis when deciding on the best course of action. The analysis involves weighing the potential benefits of an intervention against the possible risks. For example:

- **Life-saving Procedures**: Administering a blood transfusion in a patient with ongoing bleeding may involve risks but is necessary to prevent death. Even if ideal conditions like crossmatching aren't met, the immediate need for intervention takes precedence.
- **Risk Acceptance**: Clinicians must be prepared to undertake procedures with known risks if these are the only options available to keep the patient alive. This might include administering medications off-label or performing emergency surgeries under less-than-optimal conditions.
- **Delay not life threatening condition evaluation and treatment**: Clinician must triage the most life threatening condition in the patient, and accept or the deterioration of one condition to save the life of the patient (ex : increase azotemia with diuretics in a cat with concomitant chronic kidney disease and acute congestive heart failure, or to postpone complementary exam for a non-life threatening condition).

****Key Point 3: Managing Uncertainty and Decision-making****

In emergency situations, clinicians often have to make decisions with limited information, where waiting for absolute certainty could result in harm or death. One approach to manage this uncertainty is to aim for decisions based on a 90% certainty threshold:

- **Gray-zone Approach**: This involves identifying a diagnostic "gray zone" where clinicians can confidently rule in or rule out a disease with a high degree of certainty. For example, in assessing whether a patient is suffering from a disease, there may be a point where one can say with 90% certainty that the patient does have the disease (high specificity), and another point where one can say with 90% certainty that the patient does not have the disease (high sensitivity). Between these two points lies a gray zone where the clinician must make informed decisions based on the available evidence and the patient's condition, or look for other markers that could provide clearer insight into the situation.

****Key Point 4: Flexibility and Rule-bending in Emergency Care****

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Emergency care often requires flexibility and a willingness to deviate from standard protocols:

- ****Breaking the Rules**:** Clinicians might need to exceed the recommended blood transfusion volumes or forego pre-transfusion crossmatching in life-threatening situations. The key is understanding the pathophysiology behind the patient's condition and using this knowledge to justify necessary deviations from standard practices.

- ****Clinical Judgment**:** The ability to "bend the rules" effectively requires a deep understanding of the patient's condition and the confidence to apply this knowledge in practice.

****Key Point 5: Honest Communication with Pet Owners****

Open and honest communication with pet owners is crucial. Clinicians must:

- ****Inform Owners**:** Clearly explain all available therapeutic options, including the potential benefits and risks, to allow owners to make informed decisions about their pet's care.

- ****Adapt Strategy to Owner Preferences**:** If an owner has financial or other limitations, clinicians should adapt the treatment plan accordingly, always ensuring that the chosen approach is in the best interest of the pet. This might mean accepting higher risks in treatment but doing so transparently and with the owner's written informed consent.

- ****Defend the Patient's Best Interests**:** Clinicians should advocate for the pet's well-being, ensuring they are comfortable with the agreed-upon treatment plan and that it aligns with the pet's best interests.

****Key Point 6: Recognizing and Respecting Professional Limitations****

Emergency care often involves complex cases that may be beyond a clinician's expertise or resources. In such situations:

- ****Referral to Specialists**:** Clinicians should not hesitate to refer patients to specialized centers if they believe the patient would benefit from advanced care. This is not a failure but a crucial aspect of ensuring the best possible outcome for the pet.

- ****Initial Stabilization**:** Even when planning to refer a patient, there are often critical interventions that can be performed immediately to stabilize the patient. For example, administering fluids to a hypovolemic patient or performing a thoracocentesis for pleural space disease can be life-saving measures that prepare the patient for transfer to a specialist.

****Conclusion****

Emergency and critical care for companion animals demands a combination of species-specific knowledge, rapid decision-making, flexibility in clinical practice, and strong communication skills. By prioritizing patient survival, managing uncertainty, and being willing to bend the rules when necessary, clinicians can navigate the challenges of emergency care effectively. Furthermore, recognizing one's limitations and working within a network of professionals ensures that each patient receives the best possible care, even in the most challenging circumstances.

PATHOPHYSIOLOGY, CLINICAL SUSPICION AND DIAGNOSIS OF SHOCK... THINGS YOU MIGHT HAVE FORGOTTEN

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Understanding Shock in Veterinary Medicine

****Shock**** is a life-threatening condition characterized by inadequate tissue perfusion and oxygen delivery to meet the metabolic needs of the body. This imbalance leads to cellular dysfunction, organ failure, and if untreated, death. In veterinary medicine, recognizing and managing shock in dogs and cats is critical for successful outcomes.

Shock is classified into four main types based on the underlying pathophysiological mechanisms:

1. **Hypovolemic Shock**:

- **Cause**: Severe fluid loss due to hemorrhage, vomiting, diarrhea, or severe dehydration.
- **Pathophysiology**: Decreased circulating blood volume leads to reduced venous return, decreased cardiac output, and ultimately inadequate tissue perfusion.

2. **Cardiogenic Shock**:

- **Cause**: Primary cardiac failure, such as severe dilated cardiomyopathy, valvular heart disease, or arrhythmias.
- **Pathophysiology**: The heart's pumping ability is compromised, leading to decreased cardiac output and poor tissue perfusion despite adequate blood volume.

3. **Distributive Shock**:

- **Cause**: Severe vasodilation often due to sepsis, anaphylaxis, or neurogenic causes.
- **Pathophysiology**: Widespread vasodilation leads to relative hypovolemia and pooling of blood in the peripheral circulation, reducing venous return and cardiac output.

4. **Obstructive Shock**:

- **Cause**: Physical obstruction to blood flow, such as in cases of pericardial tamponade, gastric dilation and volvulus, tension pneumothorax or pulmonary thromboembolism.,
- **Pathophysiology**: Obstruction impedes venous return to the heart or outflow from the heart, leading to decreased cardiac output and compromised tissue perfusion.

Early recognition of shock is based on clinical assessment of perfusion parameters. Key findings in patients suspected of being in shock include:

- **Mucous Membrane Color**: Pale, cyanotic, or hyperemic gums indicate poor perfusion or abnormal blood flow.
- **Capillary Refill Time (CRT)**: Prolonged CRT (>2 seconds) suggests poor perfusion, while a shortened CRT (<1 second) may indicate hyperdynamic states like sepsis.
- **Heart Rate**: Tachycardia is common in shock as the body attempts to compensate for decreased cardiac output. Bradycardia may be seen in terminal stages or specific types of shock (e.g., neurogenic shock).
- **Pulse Quality**: Weak, thready pulses are indicative of poor cardiac output, while bounding pulses may be seen in early distributive shock.
- **Temperature Extremities**: Cool extremities suggest poor perfusion, while warm extremities may be seen in hyperdynamic distributive shock.

The Role of Arterial Pressure Measurement in Shock Diagnosis and Monitoring

Arterial pressure measurement is an essential tool in the diagnosis and management of shock. It helps determine the severity of shock and guides therapeutic interventions.

- **Direct Arterial Pressure Measurement**:

- **Benefits**: Provides accurate and continuous monitoring of blood pressure, allowing for real-time assessment of treatment efficacy. Direct measurement is particularly useful in critical care settings.
- **Challenges**: Requires invasive catheterization of an artery, which can be technically challenging and associated with complications such as infection or thrombosis.

- **Indirect Arterial Pressure Measurement**:

- **Benefits**: Non-invasive, easier to perform, and widely available. Techniques include Doppler and oscillometric methods.
- **Problems**: Less accurate than direct measurement, especially in patients with hypotension, arrhythmias, or very small body size. In such cases, the readings may underestimate or overestimate true blood pressure, leading to potential mismanagement. For shock patients the Doppler technique is typically preferred as oscillometric devices may perform poorly in case of severe tachycardia, weak signals and as a consequence may overestimate the actual blood pressure.

The Importance of Lactate Measurement and Kinetics in Shock

Lactate is a byproduct of anaerobic metabolism, and its elevation is a marker of tissue hypoxia. That said, other rare conditions may also cause a mild to moderate rise in lactates. Measuring blood lactate levels provides crucial diagnostic and prognostic information in shock.

- **Diagnostic Value**:

- **Elevated Lactate Levels**: Suggests tissue hypoperfusion and is often used to confirm the diagnosis of shock. Lactate levels above 2.5 mmol/L in dogs and 2.0 mmol/L in cats are generally considered abnormal.

- **Lactate Clearance**: A decrease in lactate levels over time indicates improving tissue perfusion and a positive response to therapy. Failure of lactate levels to decrease or a further increase suggests ongoing or worsening shock.

- **Prognostic Value**:

- **High Initial Lactate Levels**: Associated with increased mortality in both dogs and cats. Studies have shown that dogs with lactate levels above 6 mmol/L have a significantly higher risk of death. That said, the clinician should never cast any judgement on an initial lactate measurement.

- **Lactate Kinetics**: Serial lactate measurements are useful in monitoring the progression of shock and the effectiveness of treatment. Rapid clearance of lactate is associated with a better prognosis, while persistent hyperlactatemia is a poor prognostic indicator. Clinicians should always first attempt to address the shock state, prior to taking treatment decisions.

Scientific Evidence Supporting the Use of Arterial Pressure and Lactate Measurement in Shock

- **Arterial Pressure**:

- Several studies have demonstrated the importance of maintaining adequate mean arterial pressure (MAP) in shock management. A MAP of 60-70 mmHg is generally targeted to ensure organ perfusion in dogs and cats.

- Indirect arterial pressure measurement methods, while less accurate, have been shown to provide valuable trends in blood pressure changes, which can still guide therapy effectively when direct measurement is not feasible.

- **Lactate Measurement**:

The use of lactate as a prognostic tool is well-supported by veterinary research. Several papers found that dogs with septic shock and lactate levels above 8 mmol/L have a significantly lower survival rate. That said, other studies reported that lactate clearance within the first 6 hours of resuscitation was associated with improved outcomes in critically ill dogs.

Conclusion

Shock in dogs and cats is a complex and multifactorial condition requiring prompt recognition and aggressive management. Understanding the different types of shock and the associated clinical signs is essential for early diagnosis. Arterial pressure measurement, whether direct or indirect, plays a crucial role in monitoring and guiding therapy, despite some limitations in accuracy. Lactate measurement provides invaluable diagnostic and prognostic information, with lactate kinetics serving as a key indicator of treatment efficacy. By staying informed about these critical aspects of shock diagnosis and management, veterinarians can improve outcomes for their canine and feline patients in shock.

THE INITIAL TREATMENT OF SHOCK PATIENTS: FLUIDS (AND BEYOND)

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Understanding Shock and Its Treatment

Shock in canine and feline patients is a critical condition that requires prompt and appropriate intervention. Shock can be classified into four main types according to Weil's classification: hypovolemic, cardiogenic, distributive, and obstructive. Each type of shock has distinct underlying pathophysiology, requiring specific treatment approaches. This proceeding will discuss the initial treatment of shock in dogs and cats, focusing on fluid therapy, vasopressors, positive inotropes, and other interventions tailored to each type of shock. Additionally, the importance of thorough monitoring throughout the treatment process will be emphasized.

Treatment Recommendations for Each Type of Shock

1. Hypovolemic Shock: Hypovolemic shock results from a significant loss of blood or fluid, leading to decreased venous return, reduced cardiac output, and insufficient tissue perfusion.

****Fluid Administration:****

- **Dogs:** Administer crystalloids at an initial shock dose of 90 mL/kg, delivered incrementally in boluses of 10-20 mL/kg over 10-15 minutes. The patient's response should be reassessed after each bolus.
- **Cats:** Administer crystalloids at an initial shock dose of 60 mL/kg, with boluses of 5-10 mL/kg over 10-15 minutes. Reassess after each bolus, as cats are more sensitive to fluid overload.

****Additional Interventions:****

- **Colloids:** Can be used in refractory cases or when crystalloids are insufficient. Administer 3-5 mL/kg boluses in both dogs and cats, up to a total of 20 mL/kg in dogs and 15 mL/kg in cats.
- **Blood Products:** Indicated in cases of hemorrhagic shock. Administer packed red blood cells (PRBCs) or whole blood at 10-20 mL/kg.

2. Cardiogenic Shock: Cardiogenic shock occurs due to primary cardiac dysfunction, such as severe heart disease or arrhythmias, leading to inadequate cardiac output.

****Positive Inotropes:****

- **Pimobendan:** Administer 0.15 mg/kg orally or IV in dogs (off-label use in cats) to improve cardiac output through its inotropic and vasodilatory effects.
- **Dobutamine:** Administer at 5-15 mcg/kg/min IV. It enhances cardiac contractility and is especially beneficial in dogs and cats with systolic dysfunction. That said, it may increase cardiac oxygen requirements, and thus should be considered in specific cases.
- **Anti-arrhythmics:** In cases of shock caused by arrhythmias, anti-arrhythmic drugs such as lidocaine (dogs: 1-2 mg/kg IV; cats: 0.25-0.5 mg/kg IV) may be indicated.

****Fluid Administration:****

- **Dogs and Cats:** Administer small fluid boluses of 5-10 mL/kg over 10-15 minutes whenever the patient also presents any sign of hypovolemia. That said, be cautious to avoid fluid overload. Patients with congestive heart failure should not receive fluid boluses!

****Vasopressors:**** Vasopressors will increase the afterload, although they may also have a positive effect on preload. The increase of the afterload is however likely to increase the cardiac workload... and hence these molecules will typically be avoided in patients with cardiogenic shock.

- **Dopamine:** Initiate at 5-10 mcg/kg/min IV in dogs and cats. This drug increases cardiac output and blood pressure by stimulating dopamine and beta-adrenergic receptors.
- **Norepinephrine:** Start at 0.1-1 mcg/kg/min IV. It is particularly useful in cases where hypotension persists despite adequate fluid resuscitation.

3. Distributive Shock: Distributive shock, often caused by sepsis, anaphylaxis, or neurogenic factors, is characterized by severe vasodilation, leading to relative hypovolemia and inadequate tissue perfusion.

EMERGENCY AND CRITICAL CARE

Fluid Administration:

- **Dogs:** Administer crystalloids at 90 mL/kg, in boluses of 20-30 mL/kg over 10-15 minutes, reassessing after each bolus.
- **Cats:** Administer crystalloids at 60 mL/kg, with boluses of 10-15 mL/kg, and reassess frequently.

Vasopressors:

- **Norepinephrine:** Start at 0.1-1 mcg/kg/min IV to counteract vasodilation and increase systemic vascular resistance.
- **Epinephrine:** In anaphylactic shock, administer 0.01 mg/kg IM or SC (IV administration in severe cases) to rapidly reverse the vasodilatory effects of histamine release.

Positive Inotropes:

- **Dobutamine:** Start at 5-15 mcg/kg/min IV in cases of septic shock where myocardial depression is evident.

Additional Interventions:

- **Corticosteroids:** In anaphylactic shock, administer dexamethasone (0.1-0.2 mg/kg IV) or prednisolone (1 mg/kg IV) to reduce inflammation and histamine release.
- **Antibiotics:** Broad-spectrum antibiotics should be administered immediately in cases of septic shock, based on culture and sensitivity when possible.

4. Obstructive Shock: Obstructive shock results from physical obstruction to blood flow, such as in gastric dilation-volvulus (GDV), pericardial tamponade, or pulmonary thromboembolism.

Fluid Administration:

- **Dogs and Cats:** Administer crystalloids at 20 mL/kg boluses, reassessing after each bolus. Fluids help to maintain perfusion despite the obstruction.

Vasopressors:

- **Norepinephrine:** Use 0.1-1 mcg/kg/min IV to support blood pressure when obstruction limits cardiac output.

Additional Interventions:

- **GDV:** Immediate decompression of the stomach is critical, followed by surgical correction.
- **Pericardial Tamponade:** Pericardiocentesis is necessary to relieve pressure on the heart.
- **Pulmonary Thromboembolism:** Anticoagulants like heparin (75-100 IU/kg SC every 8-12 hours) are indicated to prevent further clot formation.

Monitoring the Shock Patient

Effective monitoring is crucial for reassessing the shock patient and guiding ongoing treatment. Key monitoring parameters include:

1. Clinical Perfusion Markers:

- **Mucous Membrane Color and CRT:** Monitor changes in color and capillary refill time to assess peripheral perfusion.
- **Heart Rate and Pulse Quality:** Regularly assess to monitor the response to therapy and detect any worsening of the condition.

2. Blood Pressure:

- **Indirect Measurement:** Useful for trending changes in blood pressure, although less accurate in hypotensive patients.
- **Direct Measurement:** Preferred for accurate, continuous monitoring in critical cases.

3. Lactate Levels:

- **Initial Measurement:** High lactate levels indicate poor tissue perfusion and the severity of shock.
- **Lactate Clearance:** A decreasing trend in lactate levels suggests improving perfusion and a positive response to treatment.

4. Point of Care Ultrasound (POCUS):

- **Cardiac Ultrasound:** Assess cardiac function, including contractility, chamber sizes, and presence of pericardial effusion.
- **Abdominal Ultrasound:** Identify free fluid, or other abdominal causes of shock. Assess volume status by assessing the caudal vena cava, or its ratio to the aorta.
- **Thoracic Ultrasound:** Evaluate for pleural effusion, pneumothorax, pulmonary consolidations, or pulmonary edema.

Conclusion

The initial treatment of shock in dogs and cats requires a tailored approach based on the type of shock, with careful administration of fluids, vasopressors, and positive inotropes as appropriate. Interventions specific to the underlying cause of shock must be promptly initiated, and continuous monitoring is essential to guide therapy and reassess the patient's response. Through a comprehensive and systematic approach, clinicians can effectively manage shock and improve outcomes for their canine and feline patients.

HOW CAN POINT OF CARE ULTRASOUND HELP YOU IN SHOCK PATIENTS

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Introduction

Cardiovascular shock is a critical condition that demands immediate and precise intervention. Point of care ultrasound (POCUS) has emerged as an invaluable tool for veterinarians, offering rapid, non-invasive assessment and aiding in the management of shock in companion animals. This proceeding will explore how POCUS can help clinicians diagnose, guide treatment, and monitor the progress of dogs and cats presenting with shock.

Key Point 1: Definition of Cardiovascular Shock and Its Subcategories

Cardiovascular shock is defined as a state of insufficient blood flow to the tissues, resulting in inadequate oxygen delivery and subsequent cellular and organ dysfunction. According to Weil's classification, shock is broadly categorized into four main subtypes:

- **Hypovolemic Shock:** Caused by a significant loss of blood or fluids, leading to decreased circulating volume and, consequently, reduced cardiac output.
- **Cardiogenic Shock:** Results from the heart's inability to pump blood effectively, often due to myocardial infarction, severe valvular disease, or cardiomyopathy.
- **Distributive Shock:** Characterized by inappropriate vasodilation, as seen in conditions like sepsis or anaphylaxis, leading to relative hypovolemia.
- **Obstructive Shock:** Caused by physical obstruction of blood flow, such as in cases of pulmonary embolism, cardiac tamponade, or tension pneumothorax.

Key Point 2: Definition of Point of Care Ultrasound (POCUS) in Veterinary Medicine

Point of care ultrasound (POCUS) refers to the use of ultrasonography at the bedside or within a clinical setting, performed by the clinician at the time of patient evaluation. In veterinary medicine, POCUS is increasingly utilized to provide immediate insights into a patient's condition, facilitating rapid decision-mak-

ing and improving outcomes, particularly in emergency situations. The veterinarian is however supposed to formulate closed questions that she/he wants to answer with POCUS. In other words, it should not be regarded as a full comprehensive ultrasound, neither a fishing expedition...

Part 1: POCUS in Diagnosing the Cause of Shock

POCUS is a powerful diagnostic tool for identifying the underlying cause of shock, allowing clinicians to assess the presence of major pathologies and evaluate the patient's volume status and cardiac function.

- **Identification of Major Pathologies**:**

- **Free Fluid**:** POCUS can quickly detect the presence of free fluid in the abdomen (ascites) or thorax (pleural effusion), which might indicate hemorrhage, infection, or neoplasia. Identifying free fluid helps in diagnosing conditions like hemoperitoneum or septic peritonitis, which can cause or contribute to shock.

- **Pulmonary Lesions**:** POCUS can be used to identify pulmonary edema, consolidation, or pleural effusion. These findings may suggest conditions like congestive heart failure, pneumonia, or pulmonary contusions, guiding the clinician towards the appropriate subtype of shock.

- **Volume Status and Fluid Responsiveness**:** POCUS allows for the assessment of the caudal vena cava (CVC) diameter and its size compared to the aorta (at the sublumbar site) or collapsibility (at the subxiphoid site), providing insights into the patient's volume status or fluid responsiveness.

- **Cardiac Function**:** POCUS enables the clinician to evaluate cardiac contractility, chamber sizes, and the presence of pericardial effusion, which can help in diagnosing cardiogenic shock, indicating volume status, or diagnose obstructive shock due to cardiac tamponade.

Part 2: POCUS-Guided Therapeutic Interventions

POCUS is not only a diagnostic tool but also a guide for therapeutic interventions, improving both safety and accuracy.

- **Guiding Pericardiocentesis**:** In cases of cardiac tamponade, POCUS can be used to visualize the pericardial effusion and guide needle placement for pericardiocentesis, reducing the risk of complications and ensuring effective fluid removal.

- **Thoracic and Abdominal Drain Placement**:** POCUS aids in identifying the optimal site for thoracocentesis or abdominocentesis, ensuring the drainage of fluid or air is both safe and effective.

- **Central Line Placement**:** POCUS can guide the placement of central venous catheters, ensuring correct positioning and minimizing the risk of complications. This is especially beneficial in shock patients with difficult venous access, or those requiring invasive hemodynamic monitoring.

Part 3: Monitoring Treatment Effectiveness with POCUS

POCUS allows for real-time monitoring of the effects of treatment, enabling clinicians to make informed adjustments based on the patient's response.

- **Reassessing Volume Status and Fluid Responsiveness**:** After administering fluids or other therapies, POCUS can be used to reassess the patient's volume status and fluid responsiveness. This ensures that the treatment is achieving the desired effect without causing fluid overload or other complications.

- **Monitoring Cardiac Function**:** Ongoing evaluation of cardiac function with POCUS can help assess the impact of treatments such as inotropes or vasopressors. Adjustments can be made based on changes in contractility or the development of new cardiac issues.

Part 4: Monitoring Disease Progression with POCUS

POCUS also plays a critical role in monitoring disease progression, providing insights into how the patient's condition evolves over time.

- **Pulmonary Patterns**:** POCUS can track changes in pulmonary patterns, such as worsening or improvement of pulmonary edema, allowing clinicians to adjust treatments accordingly.

- **Accumulation of Free Fluid**:** By regularly assessing for free fluid in the thorax or abdomen, POCUS helps monitor disease progression, such as active hemorrhage or infection-related effusions, prompting timely interventions.

Conclusion

POCUS is a highly valuable tool in the management of dogs and cats in shock. It aids in the rapid diagnosis of the underlying cause of shock, guides therapeutic interventions, and allows for ongoing monitoring of treatment efficacy and disease progression. Incorporating POCUS into the clinical approach significantly enhances the clinician's ability to deliver timely, accurate, and effective care, ultimately improving patient outcomes.

HOW CAN POINT OF CARE ULTRASOUND HELP YOU WITH GASTRO INTESTINAL TRACT DISORDERS

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Since the first publication in 2004 of the use of point-of-care ultrasound (POCUS) to detect abdominal free fluid in trauma patients, the applications for abdominal POCUS has grown rapidly. Among them, gastrointestinal (GI) evaluation in the emergency setting has much potential.

Gastrointestinal POCUS in the ER

The acute abdomen is a common emergency presentation in dogs and cats with several possible primary GI etiologies. Beyond detection of free fluid, POCUS can aid diagnosis of some primary GI etiologies such as intestinal intussusception, GI foreign body obstruction, and GI perforation. However, no veterinary study has compared GI POCUS with abdominal ultrasonography or computed tomography. POCUS can be incorporated into the initial physical exam, particularly in unstable patients where secondary diagnostics (such as radiographs) could be detrimental. Abnormalities detected on POCUS can improve treatment decisions, but lack of POCUS abnormalities does not rule out primary GI etiologies.

GI perforation

Gastrointestinal perforation, a major cause of septic abdomen, is a medical and surgical emergency. POCUS can be used to detect abdominal free fluid and free air that can be found during GI perforation.

It is important to consider patient positioning – Free fluid will accumulate in dependent regions; free air will accumulate in the non-dependent regions.

Free fluid will be detected as an anechoic triangle shape structure, around the abdominal organs. If free fluid is detected, it is important to tap and analyze it.

Identification of abdominal free air requires identification of reverberation artifacts from the parietal peritoneal lining, associated with an enhanced peritoneal stipe sign. Other POCUS abnormalities supportive of pneumoperitoneum include thickened intestinal loops or air reverberation artifacts within peritoneal free fluid.

GI obstruction

Gastrointestinal obstruction can be suspected based on the presence of segmental intestinal luminal distension and ineffective GI hypermotility - typically manifesting as to-and-fro motions of luminal contents with or without segmental hypo- and hypermotility. These findings should prompt further investigation for obstructive causes such as a foreign body, intussusception, or neoplasia.

- Foreign bodies appear as static intraluminal structures that produce gas-shadowing artifacts.

Intussusception can present as a target shape composed of multilayered concentric rings with signs of obstruction. Transcutaneous manipulation of intussusceptions by POCUS has also been described. This technique involves applying pressure with one hand to the intestine aboral to the intussusception followed by pushing of the intussusception orally with the other hand. Reduction is then confirmed using POCUS.

Pain and palpation can help localize a mass in dogs and cats presented with an acute abdomen. Placing the probe in the region of pain or the mass could help identify the lesion.

Use of gastrointestinal POCUS in the critically ill patient***GI motility evaluation***

Gastric or intestinal dysmotility due to illness or medication is common in critically ill patients and can lead to complications such as vomiting, regurgitation, large gastric residual volumes or abdominal discomfort, aspiration pneumonia and disruption of effective delivery of nutritional support. Assessment of GI motility will help to guide prokinetic administration and identify post-operative complications.

Ultrasound in dogs and cats with normal gastrointestinal motility showed 4-5 contractions per minute in the stomach and proximal duodenum and 1-3 contractions per minute in the jejunum. Several protocols have been described for GI evaluation, with the patient standing or in lateral recumbency and should be adapted to the patient's condition and operator preference.

The evaluation of the gastric antrum in humans showed a dilated static antrum with solid and/or liquid contents corresponded to increased gastric contents. Cross-sectional area of the gastric antrum has been used in cats and dogs to evaluate gastric emptying. Further studies are needed to evaluate the reliability of gastric suctioning via a feeding tube compared with ultrasound assessment of gastric residual volume and the clinical consequences of this evaluation. The author will present preliminary data of a study conducted in dogs and cats hospitalized in the ICU.

Use of POCUS for feeding tube placement

Feeding tubes are frequently placed in sick, hospitalized patients. Misplaced feeding tubes can lead to significant morbidity and mortality, thus radiographs are typically performed to confirm correct placement in the GI tract. But moving and positioning critically ill patients for radiographs can be challenging or even contraindicated. In humans, ultrasonography has been shown to be an efficient method for verification of nasogastric tube placement, although there is insufficient evidence supporting its use to identify *incorrect* gastric tube placement. Several techniques are described in human medicine for this purpose: direct visualization of the tube with transverse or longitudinal views of the cervical esophagus during placement, direct visualization of the tube in the stomach using the subxiphoid POCUS view, and visualization of air or liquid filling the stomach via the feeding tube from the same subxiphoid view. An ultrasound-guided percutaneous dilatational esophagostomy tube placement technique has been described in dog cadavers with the benefit of minimizing oral contamination of instruments. In canine and feline neonates, ultrasound guided orogastric tube placement is described. More studies are needed in critically ill patients to evaluate ease and risks of POCUS guided feeding tube placement.

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SEPTIC PERITONITIS IN COMPANION ANIMALS – WHAT WE’VE LEARNED FROM HUMAN MEDICINE

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Septic peritonitis represents a critical emergency in veterinary medicine, requiring a high degree of suspicion, rapid diagnosis, and prompt intervention to optimize patient outcomes. This condition is most frequently triggered by the use of non-steroidal anti-inflammatory drugs (NSAIDs) in dehydrated patients or those with pre-existing gastrointestinal (GI) issues, potentially in combination with steroids. As prevention is always preferable to treatment, veterinarians should carefully evaluate the necessity of NSAIDs for each patient and inform pet owners of potential adverse effects. Any GI signs in patients on NSAIDs warrant immediate cessation of the medication and close monitoring.

While NSAIDs are a common culprit, other causes of septic peritonitis include GI tract rupture secondary to foreign objects or neoplasia, urogenital or hepatobiliary tract ruptures, and penetrating trauma. These proceedings focus primarily on the rapid diagnosis and treatment of septic peritonitis, as the prognosis is significantly better for stable patients compared to those who have progressed to shock.

It is important to recognize that not all patients with septic peritonitis present in shock. A retrospective study from the Royal Veterinary College revealed that more than half of referred patients diagnosed with septic peritonitis had lactate levels within the normal range, indicating they were still cardiovascular compensating for the septic event.

Pearl 1: “POCUS in All Unstable Patients!”

When a patient presents with abdominal distension and a positive fluid wave, free fluid should be suspected. However, septic peritonitis often presents with only moderate fluid accumulation, accompanied by a rapidly deteriorating general condition. Point-of-care ultrasound (POCUS) is invaluable in identifying free fluid, and should be performed in all unstable patients, even if septic peritonitis is not initially suspected. Early detection can significantly improve survival rates.

In early cases, the amount of free fluid may be minimal, making detection challenging. However, the presence of an enhanced peritoneal stripe sign on ultrasound suggests free gas in the abdomen, which, in the absence of recent surgery, is indicative of bacterial gas production or organ rupture. POCUS is also useful for monitoring fluid accumulation over time, especially after stabilization.

Pearl 2: “Perform Centesis and Analyze the Fluid!”

Once free fluid is identified, it should be aspirated and analysed. Ideally, this includes cytological examination, glucose concentration measurement, and lactate concentration measurement. Septic effusions are typically exudates, rich in proteins and cellular content. A refractometer can estimate protein content, while cytology can assess cellularity. The presence of intracellular bacteria on cytology confirms a diagnosis of septic peritonitis.

Glucose and lactate measurements can further support a diagnosis. In septic peritonitis, glucose levels in the fluid will be lower, typically more than 20 mg/dL below blood levels, while lactate levels will be higher, typically more than 2 mmol/L above serum concentrations. Portable devices for measuring glucose and lactate are recommended for general practice, as they can provide rapid, critical information.

Pearl 3: “Administer Antibiotics Early!”

EMERGENCY AND CRITICAL CARE

Early administration of systemic antibiotics is crucial once septic peritonitis is diagnosed or highly suspected. Evidence from human medicine underscores that early appropriate antibiotic therapy significantly impacts survival rates in septic patients, even more so than early fluid administration. However, antibiotic stewardship is essential to avoid resistance. The choice of antibiotics should be guided by the suspected source of infection and the patient's overall condition. In Europe, amoxicillin clavulanic acid is the most common antibiotic of choice. Always perform culture and sensitivity testing to guide and potentially adapt antibiotic therapy.

Pearl 4: "Stabilize Prior to Surgery!"

Before surgical intervention, ensure the patient is stable enough to tolerate anaesthesia. Key indicators of stability include normal clinical perfusion parameters (mentation, heart rate, pulse pressure, mucous membrane colour, capillary refill time, and temperature), supported by normal arterial systolic blood pressure ($>80\text{-}90 \text{ mmHg}$) and lactate levels ($<2.5 \text{ mmol/L}$).

If these parameters are not met, stabilize the patient with fluid boluses of isotonic fluids. In cases where further stabilization is not achievable with basic interventions, consider this the optimal condition for surgery, but inform the owner that the prognosis is more guarded. Other types of fluids, as well as vasopressors might further improve patient stabilisation, and if not available to you in your practice, the patient might be better referred to a specialist centre nearby.

Pearl 5: "Perform a Rule of 20: Assess All Organ Systems!"

Septic peritonitis can impact all organ systems, necessitating a comprehensive treatment plan tailored to the individual patient. Kirby's Rule of 20 is a valuable tool, prompting clinicians to assess 20 vital parameters daily, including fluid status, oncotic pressure, oxygenation, immune function, pain management, and more.

For example, regular pain assessments should guide the analgesic plan, and monitoring urine output and weight can help tailor fluid therapy. While not directly part of initial management, these considerations significantly influence outcomes and reflect the complexity and diligence required in managing septic peritonitis.

Conclusion

Managing septic peritonitis in companion animals is a complex, multi-faceted process that demands early diagnosis, appropriate use of antibiotics, stabilization, and a holistic approach to patient care. By applying lessons from human medicine, veterinarians can improve the prognosis and survival rates of their patients facing this serious condition.

DIFFERENT TYPES OF DYSPNEA: LESSONS LEARNED FROM A GOOD CLINICAL EXAM

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Introduction

The respiratory system can be divided anatomically into six main regions; upper respiratory tract (nasal passages, pharynx, larynx, and trachea), lower airways (bronchi and bronchioles), lung parenchyma, pleural space diaphragm, and chest wall (ribs and intercostal muscles). During each breath, air is inspired and moves via the upper and lower airways to the lungs through an active process involving the diaphragm and intercostal muscles. The diaphragm contracts and flattens and the intercostal muscles contract causing the ribs to move up and outwards. This creates a negative pressure gradient from the nose to the thoracic cavity causing air movement and air expansion of the lungs. Expiration is passive, elastic recoil of the lungs secondary to the relaxation of the diaphragm that expels air from the lungs. The chest wall is not actively involved in expiration, but the abdominal wall muscles may assist when there is increased expiratory effort (1).

Dyspnea, or difficulty breathing, is a common clinical sign in both dogs and cats. It can be caused by a variety of underlying conditions, ranging from respiratory to cardiovascular diseases. A thorough clinical examination is crucial for accurately diagnosing the type and cause of dyspnea, which in turn guides effective treatment strategies.

Dyspnea can be classified based on the phase of respiration affected and the underlying cause:

- Inspiratory Dyspnea: Characterized by difficulty during inspiration. Common causes include upper airway obstructions such as laryngeal paralysis or tracheal collapse, and is often associated with a noisy respiration
- Expiratory Dyspnea: Difficulty during exhalation, often seen in conditions like chronic bronchitis or asthma.
- Asynchronous, inverse, and paradoxical breathing: This occurs when the chest wall moves inward during inspiration and outward during expiration, opposite to the normal pattern. It is often a sign of severe respiratory distress and can indicate conditions such as pleural disease, diaphragmatic fatigue or flail chest.
- Mixed Dyspnea: Involves both inspiratory and expiratory difficulty, typically seen in severe cases of pulmonary edema or pleural effusion.

This lecture will describe several abnormal respiratory patterns, in order to help the clinician to recognize rapidly any modification.

1. Inspiratory dyspnea

Upper respiratory obstruction can be readily identified due to the presence of loud inspiratory noises without the need for a stethoscope (2). A stertor, a low-pitched sound, is associated with nasopharyngeal pathology, and stridor, a high-pitched sound, is most commonly associated with laryngeal pathology (3).

Upper airway involvement results in a dynamic or static reduction of the airway lumen, causing an increase in respiratory effort. Clinically, this obstruction manifests as noisy breathing and often marked dyspnea. When the obstruction is **extrathoracic** (laryngeal region, extrathoracic trachea), dyspnea will be **inspiratory**. When the obstruction is **intrathoracic** (intrathoracic trachea, large bronchi), dyspnea is **expiratory**. In both cases, dyspnea is noisy (2).

Specific sites commonly associated with upper airway obstruction include the nasal cavities, larynx (e.g., paralysis or neoplasia), nasopharynx (palate or pharyngeal tissue abnormalities), trachea, and large bron-

chi. Brachycephalic dogs are at increased risk of upper airway obstruction due to their abnormal anatomy, and large breeds are more predisposed to laryngeal paralysis, while Yorkshires are prone to tracheal collapse. Thus, the epidemiological context is crucial in differential diagnosis.

Early recognition of the upper respiratory tract as a cause of respiratory distress is crucial as this pathology is associated with hyperthermia and may substantially worsen with handling; therefore, these patients usually require cooling interventions and early administration of anxiolytic or mild sedatives before a full physical examination is performed. Therefore, management includes:

- Oxygen therapy (flow-by, nasal catheter, nasotracheal tube)
- Sedation (butorphanol, which is also an antitussive, can be beneficial in this case)
- Corticosteroids: Upper airway obstruction causes inflammation, which further increases the obstruction. Corticosteroids, at anti-inflammatory doses, can quickly reduce this inflammation. The preferred corticosteroid is dexamethasone (0.1 mg/kg IV SID) due to its strong anti-inflammatory effect.
- Temperature management: Upper airway obstruction often leads to hyperthermia, worsening the clinical picture, as the animal is no longer able to regulate its body temperature.
- Intubation: In severe obstruction, intubation is life-saving. It is rarely impossible in dogs and cats. Only lesions compromising the patency of the upper airways, such as severe jaw fractures, tracheal crushing, or laryngeal/pharyngeal trauma, may necessitate emergency tracheotomy or the placement of a trans-tracheal catheter. However, often a few hours of intubation, combined with corticosteroid therapy and temperature management, allow for a calm recovery and passage through the acute crisis.

2. **Expiratory dyspnea**

Expiratory dyspnoea with noticeable abdominal “push” and elevated respiratory rates are most commonly detected in patients with lower airway disease, and although associated with lower airway disease, wheezes are less consistently auscultated in these patients (2, 4, 5).

Other symptoms include coughing, associated with primarily expiratory distress.

Respiratory distress in this case is due to an obstruction of expiratory airflow caused by bronchoconstriction, inflammation (reduction in bronchial diameter), and the accumulation of bronchial secretions. During inspiration, the increased pressure inside the airways allows them to open. During expiration, the increased intrathoracic pressure and decreased pressure in the bronchi lead to bronchial collapse, resulting in increased expiratory effort, expressed as an “expiratory push” during physical examination (abdominal contraction during expiration). Pulmonary auscultation will reveal expiratory wheezing, indicating reduced airway diameter.

Management includes oxygen therapy, bronchodilators, sedation and corticosteroids.

3. **Asynchronous, inverse, and paradoxical breathing**

Asynchronous, inverse, and paradoxical breathing are terms often used interchangeably to describe the outward movement of the chest and abnormal inward movement of the abdomen during inspiration; however, there are inconsistencies in the definition of inverse and paradoxical breathing in the veterinary literature, and therefore, it is recommended in an attempt to avoid confusion and keep it simple, the term **asynchronous** is used; the chest and abdominal wall are moving asynchronously.

An asynchronous breathing pattern is a very sensitive indicator of pleural space disease but has lower specificity; in other words, the absence of this breathing pattern can make you less suspicious of pleural space pathology, but it should not be used alone to diagnose pleural space disease. This breathing pattern, alongside the presence of reduced lung sounds on auscultation, can further support a diagnosis of pleural space disease (2,6, 7).

The presence of pleural effusion, pneumothorax, or a mass in the pleural space will cause respiratory distress by compressing the lungs and thus limiting lung expansion. The pleural space can be filled with fluid (pure or modified transudate, exudate), air, or a mass (neoplasia, abdominal organs, etc.).

Initial stabilization is the same (oxygen, sedation), and pleural space involvement can be easily confirmed by performing thoracic point of care ultrasound (POCUS). Once pleural space involvement is confirmed, thoracocentesis is both a therapeutic and diagnostic procedure.

4. Mixed dyspnea

This one is the trickiest one. Both expiration and inspiration are modified, at different level.

Mixt dyspnea is more related to **pulmonary parenchymal involvement**, including cardiogenic or non-cardiogenic edema, bronchopneumonia, pulmonary contusions/hemorrhages, and neoplasms.

These conditions are characterized by decreased lung compliance. The lungs become more "rigid" and require higher inspiratory pressures to achieve the same tidal volumes. Clinically, this results in both inspiratory and expiratory efforts, leading to mixed dyspnea. Clinical signs are also related to decreased efficiency of pulmonary gas exchange, resulting in hypoxemia. This is due to the presence of fluid in the alveolar space, causing alveolar collapse, or thickening of the alveolar-capillary membrane, which reduces the efficiency of gas exchange.

Your next challenge will be to differentiate between cardiac and non-cardiac dyspnea.

Cardiac auscultation is very helpful for diagnostic orientation, as well as. Pets with congestive heart failure (CHF) typically have both an inspiratory and an expiratory increase in effort; Hypothermia is more consistent with CHF, whereas hyperthermia is more consistent with respiratory disease; however, temperature can also be normal. The heart rate is a critical parameter; with CHF, the sympathetic nervous system is in overdrive, so these patients should be tachycardic. The presence of sinus arrhythmia, an indicator of high vagal tone, essentially rules out CHF!

Other physical examination findings that would increase the suspicion of CHF include the presence of a loud (\geq grade 4/6) left apical systolic murmur in a small breed dog, consistent with more significant degenerative valve disease, the presence of tachyarrhythmias (i.e., premature beats or a very fast, irregular rhythm concerning for atrial fibrillation), or a gallop sound in a large breed dog. Findings that would decrease the suspicion for CHF would include the absence of a (loud) heart murmur in a small breed dog, a cough that only occurs on tracheal palpation, or a stable patient with crackles. A crackle is the sound of an alveolus popping open after it has collapsed. Alveoli can collapse with a variety of diseases – not only CHF but also pneumonia, hemorrhage, neoplasia, and fibrosis. Generally, if there is enough cardiogenic fluid in the lungs to cause crackles, the patient is in significant distress. A relatively stable and happy patient with diffuse crackles is unlikely to be in CHF.

Cats are more challenging, as heart murmur is not often present in case of CHF. A recent study (8) described an interesting algorithm to aid in the diagnostic orientation of dyspnea in cats. Thus, a temperature below 37.5°C, a heart rate above 200 bpm, and a respiratory rate above 80 bpm were indicative of congestive heart failure. Furosemide is the drug of choice for congestive heart failure (1-2 mg/kg SC or IV in cats, 2-4 mg/kg in dogs)

When cardiac auscultation is normal, history is important: the presence of vomiting before the consultation suggests aspiration bronchopneumonia, trauma suggests pulmonary hemorrhages. Etiological treatment should then be implemented (IV antibiotics for bronchopneumonia, tranexamic acid (10 mg/kg IV TID) for pulmonary contusions).

Thoracic POCUS will be of great help to differentiate between all these diseases and will be described in several specific lectures.

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HOW CAN POINT OF CARE ULTRASOUND HELP YOU WITH DYSPNEIC PATIENTS: PART A THE PLEURAL SPACE

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Introduction

Point of care ultrasound (POCUS) is an essential tool in the evaluation of dyspneic dogs and cats, and can rapidly allow to identify a pneumothorax or pleural effusion, two life threatening conditions that are encountered frequently in our companion animals. The Calgary PLUS protocol provides a systematic approach to identify the boundaries of the thoracic cavity and assess the presence of normal and abnormal findings. This proceeding will describe the key anatomical landmarks of the thoracic cavity, the identification of normal lung sliding, and the recognition of abnormal findings such as free fluid and free air in the pleural space.

Understanding the boundaries of the thoracic cavity is crucial for accurate POCUS interpretation:

- ****Cranial Boundary**:** The cranial boundary of the thoracic cavity is defined by the front limb, and this creates a thickening of the muscle layer overlying the thoracic cavity becomes. The cranial extent of the thorax can be challenging to visualize due to the presence of the scapula and associated musculature, which can obscure deeper structures.

- ****Dorsal Boundary**:** The dorsal boundary is formed by the hypaxial muscles, which are located along the vertebral column. These muscles provide a clear demarcation between the thoracic cavity and the paraspinal musculature. As air rises, screening this region will be mostly important when screening for a pneumothorax in a patient in ventral recumbency.

- ****Ventral Boundary**:** The ventral boundary is composed of the sternum, where the thoracic cavity can be seen curving away from the ultrasound probe. This boundary helps in visualizing the heart and mediastinal structures, as well as differentiating between thoracic and abdominal contents. The ventral aspect is mostly screened when the clinician wants to rule out pleural effusion.

- ****Caudal Boundary**:** The caudal boundary is characterized by the ****curtain sign****, which marks the transition from the thoracic cavity to the abdominal cavity. The curtain sign is observed as the diaphragm moves cranially and caudally with respiration, creating a curtain-like appearance on ultrasound. The thoracic air (whether within the lungs, or free in the thoracic cavity, makes it impossible to see any deeper lying structures, and hence the thoracic cavity seems to create a curtain that is pulled over the recognisable abdominal content during inspiration. That said, in dyspneic patients, aerophagia may cause the stomach to be airfilled, and thus on the leftside, clinicians should be cautious to distinguish the pleural line from the gastric wall. The normal curtain sign should be synchronous with respiratory movements.

- ****Pericardiophrenic Recess**:** Located at the caudoventral aspect of the thoracic cavity, the pericardiophrenic recess is an area where both the heart and liver can be visualized simultaneously. This area indicates the most caudoventral spot of the thoracic cavity, and hence is the most sensitive spot to identify free pleural fluid. That said, it can also be pivotal to help to distinguishing between pleural and pericardial effusions.

The diaphragm, which separates the thoracic cavity from the abdominal cavity, can only be directly visualized when scanning from the abdominal side.

Normal Pleural Space Findings: Lung Sliding

- ****Lung Sliding**:** Lung sliding refers to the dynamic movement of the pleural line as the lungs expand and contract during respiration. This movement appears as a shimmering or sliding motion of the pleural line

on ultrasound and indicates that the visceral and parietal pleurae are in contact and moving synchronously. The presence of lung sliding rules out pneumothorax in the scanned area, as it confirms that the lung is fully expanded against the chest wall.

Abnormal Pleural Space Findings: Free Fluid and Free Air

- ****Free Fluid in the Pleural Space**:** Free fluid within the pleural space, also known as pleural effusion, typically appears as an anechoic (black) area between the lung and the chest wall. Pleural effusions can be localized in different parts of the thoracic cavity and can vary in volume, but tends to sit in the most gravity dependent sites. Although typically hypoechoic, pleural fluid can be more hyperechoic in patients with a hemothorax, or especially with a pyothorax. Pleural effusion can be differentiated from pericardial effusion by scanning the caudoventral aspect of the thorax or using a subxiphoid view. In pleural effusion, fluid will be seen surrounding the lungs but not the heart, while in pericardial effusion, fluid is confined within the pericardial sac around the heart. By scanning the caudal lung fields, the clinician can identify whether the fluid extends towards the diaphragm, or curves around the heart.

- ****Free Air in the Pleural Space (Pneumothorax)**:** Free air in the pleural space, or pneumothorax, can be identified by several key ultrasound findings:

- ****Abnormal Curtain Sign**:** In a normal thorax, the curtain sign is synchronous with respiratory movements. However, in cases of pneumothorax, this sign may become asynchronous or may appear as a double curtain sign, indicating air in the pleural space.

- ****Lung Point**:** The lung point is a pathognomonic sign of pneumothorax, representing the point where the lung intermittently contacts the chest wall as it moves during respiration. Identifying a lung point on ultrasound confirms the presence of a pneumothorax.

- ****Ruling Out Pneumothorax**:** A pneumothorax can be ruled out by identifying a normal lung sliding or the presence of a lung pulse (a subtle pulsation of the pleural line synchronous with the cardiac cycle, seen when lung sliding is absent but the lung is still against the chest wall).

In cases where there is doubt regarding the presence of a pneumothorax, or if ultrasound findings are inconclusive, a diagnostic thoracocentesis can be performed. This procedure involves inserting a needle or catheter into the pleural space to aspirate air (or fluid) and confirm the diagnosis. Thoracocentesis can also be therapeutic, providing immediate relief in cases of tension pneumothorax.

Conclusion

Point of care ultrasound is a powerful tool for assessing the pleural space in dyspneic dogs and cats. By understanding the boundaries of the thoracic cavity and recognizing normal lung sliding, clinicians can confidently diagnose and differentiate between pleural effusions and pneumothorax. In cases of uncertainty, particularly with suspected pneumothorax, diagnostic thoracocentesis can be a valuable adjunct to POCUS, helping to ensure accurate diagnosis and prompt treatment.

HOW CAN POINT OF CARE ULTRASOUND HELP YOU WITH DYSPNEIC PATIENTS PART B THE PULMONARY PARENCHYMA

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Introduction

Point of care ultrasound (POCUS) has become an essential tool for veterinarians in the assessment of dyspneic dogs and cats, particularly when evaluating the pulmonary parenchyma. The Calgary PLUS protocol provides a structured approach for using POCUS to assess the pleural space and lungs, enabling clinicians to rapidly identify abnormal findings. This proceeding will explore the POCUS findings related to the pulmonary parenchyma in dyspneic patients, emphasizing normal and abnormal sonographic patterns.

Normal Pulmonary Findings Using the Calgary PLUS Protocol

When performing POCUS on the pleural space and lung in dogs and cats, several normal findings are expected:

- **Bat Sign**: This is the typical appearance of the thoracic wall and ribs on ultrasound. The ribs cast acoustic shadows, resembling the wings of a bat, while the pleural line represents the bat's body.
- **Rib Shadow**: The ribs create an acoustic shadow below them due to their high density, which is a normal finding during a thoracic ultrasound.
- **Pleural Line**: This is a hyperechoic line seen just below the rib shadows, representing the interface between the chest wall and the lung. The pleural line should normally be a thin and smooth line, which moves smoothly with respiration, a phenomenon known as lung sliding.
- **A-lines**: These are horizontal, hyperechoic reverberation artifacts seen beneath the pleural line when the probe is held perpendicular to the chest wall. A-lines are indicative of normal aerated lung tissue and should be the predominant finding in healthy animals.
- **B-lines**: These are vertical, hyperechoic lines extending from the pleural line to the bottom of the ultrasound screen without fading. B-lines move with respiration, and are not influenced by the presence of A-lines, which they tend to obliterate. While an occasional B-line may be seen in healthy animals, especially in the caudal lung fields, they should be sparse. Literature suggests that B-lines in normal dogs and cats are usually rare, with only a few (1-2) seen per hemithorax or intercostal space in healthy patients. Evidently the exact incidence will depend on patient characteristics such as age, BCS, and the definition of "healthy", as well as environmental factors such as housing conditions (dust, smoke), as well as the probe used and the amount of lung surface being assessed.

Abnormal Pulmonary Findings Using the Calgary PLUS Protocol

In dyspneic dogs and cats, POCUS can reveal several abnormal findings in the pulmonary parenchyma that may indicate underlying pathology:

- **Increased B-lines**:
 - **Definition**: An increase in the number of B-lines, beyond the sparse presence in normal lungs, is often indicative of pulmonary pathology such as edema, interstitial pneumonia, or lung contusions.
 - **Normal vs. Abnormal**: In healthy dogs and cats, 0-2 B-lines per intercostal space may be observed. An abnormal finding is typically defined as three or more B-lines per intercostal space. Studies suggest that even a single B-line is considered significant in the cranial and middle lung fields, especially in cats. Some describe an increase expressed as more than 3 per hemithorax. The context and the protocol used will impact this interpretation.
 - **Coalescent B-lines**: When B-lines are so numerous that they coalesce into a continuous echogenic area, this suggests severe interstitial or alveolar disease, such as advanced pulmonary edema or fibrosis. This is also sometimes referred to as "white lung".
 - **Consolidations**:

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- **Shred Sign**: This occurs when there is a partial consolidation of lung tissue, and the interface between consolidated and aerated lung appears irregular or "shredded" on ultrasound. This can be seen in conditions like pneumonitis, pneumonia or atelectasis. It is however only very rarely described in cardiogenic pulmonary edema, which thus should be considered unlikely when consolidations are visualized.
- **Nodule Sign**: Hypoechoic nodules within the lung parenchyma may indicate neoplasia, granulomas, or abscesses. These are often surrounded by aerated lung, which appears as a sharp smooth boundary around the nodule.
- **Wedge Sign**: This refers to a wedge-shaped area of consolidation, seen in pulmonary infarcts or certain types of pneumonia. The wedge sign is however only rarely reported in companion animals and the occurrence may be a species specific finding.
- **Translobar Consolidation**: This is a large, homogenous area of hypoechoic tissue representing a fully consolidated lung lobe, often seen in severe cases of pneumonia, pulmonary hemorrhage, or atelectasis.

Assessing the distribution and pattern of these sonographic findings is crucial for accurate diagnosis:

- **Pattern Distribution**:
 - **Localized vs. Diffuse**: The distribution of abnormal findings helps in differentiating between localized conditions (e.g., focal pneumonia, pulmonary neoplasia) and diffuse processes (e.g., cardiogenic pulmonary edema, acute respiratory distress syndrome).
 - **Cranioventral vs. Caudodorsal**: In conditions like aspiration pneumonia, consolidations are often cranioventral, whereas cardiogenic pulmonary edema typically affects the caudodorsal lung fields.
- **Holistic Approach**:
 - **Signalment and History**: The patient's age, breed, and history (e.g., recent trauma, known cardiac disease) should guide the interpretation of POCUS findings.
 - **Clinical Correlation**: The findings from POCUS should be integrated with physical examination, auscultation, and other diagnostic tests (e.g., radiography, blood work) to form a complete clinical picture.
 - **Decision-making**: POCUS is a dynamic tool that provides real-time information. It should be used alongside other clinical data to make informed decisions about the diagnosis, treatment, and monitoring of dyspneic dogs and cats.

Conclusion

Point of care ultrasound is an invaluable tool in assessing the pulmonary parenchyma of dyspneic dogs and cats. Understanding the normal findings, such as the bat sign, A-lines, and occasional B-lines, is crucial for recognizing pathological changes like increased or coalescent B-lines and lung consolidations. By evaluating the distribution of these patterns and considering the overall clinical context, POCUS can significantly enhance the accuracy of diagnosis and the effectiveness of treatment in these critical patients.

HOW CAN POINT OF CARE ULTRASOUND HELP YOU WITH DYSPNEIC PATIENTS PART C THE FOCUSED CARDIAC ULTRASOUND

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Introduction

Point of care ultrasound (POCUS) is a critical tool in the assessment of dyspneic dogs and cats. There is very interesting data that indicates POCUS can rapidly distinguish between dyspnea of cardiac or non cardiac origin. A basic cardiac ultrasound allows for rapid evaluation of the heart to identify underlying cardiac disease, which can be crucial in guiding treatment decisions. Be aware that the aim is not to obtain an exact diagnosis regarding the underlying cardiac disease. Rather cardiac POCUS should enable the clinician to demonstrate the phenotypical disorder such as leftsided congestion, rightsided congestion, or poor contractility.

This proceeding will discuss the three basic cardiac views: the right parasternal short-axis transventricular (mushroom) view, the right parasternal short-axis left atrial and aortic (Mercedes and whale) view, and the right parasternal long-axis four-chamber view. Key findings relevant to dyspneic patients, including left atrial to aortic ratios, pulmonary hypertension, and ventricular contractility, will be highlighted.

The Three Basic Cardiac Views

1. **Right Parasternal Short-Axis Transventricular (Mushroom) View**:

- **Description**: The transventricular view is obtained by placing the probe on the right side of the chest, perpendicular to the long axis of the heart, and rotating it slightly to align with the short axis of the ventricles. This view resembles the cross-section of a mushroom and allows visualization of the left and right ventricles.

- **Clinical Relevance**: This view is essential for evaluating right-sided heart disease. It enables assessment of ventricular size, wall thickness, and function. In patients with suspected pulmonary hypertension, this view can reveal changes in the right ventricular size and shape.

Pulmonary Hypertension Screening:

- **10-Point Score by Lyssens**: This scoring system involves five key parameters: right ventricular size, right ventricular free wall thickness, septal flattening, tricuspid regurgitation velocity, and pulmonary artery size. A higher score increases the likelihood of pulmonary hypertension. Gray zone cut-offs help rule in or out pulmonary hypertension.

- **Eccentricity Index by Lekane**: This index measures the ratio of the anterior-posterior to septal-lateral diameters of the left ventricle, which changes with increased right ventricular pressure. An increased eccentricity index could be an easy marker for pulmonary hypertension.

2. **Right Parasternal Short-Axis Left Atrial and Aortic (Mercedes or Whale) View**:

- **Description**: This view is obtained by slightly sliding and angling the probe towards the base of the heart from the transventricular position. It provides a cross-sectional image of the left atrium, aorta, and the right atrium, resembling a Mercedes-Benz logo (the aorta) and a whale (the enlarged left atrium). The normal left atrial to aortic (LA/Ao) ratio is typically less than 1.5 to 1.6 and higher than 1.0.

- **Clinical Relevance**: An increased LA/Ao ratio is a hallmark of left-sided congestive heart disease (L-CHF), although obviously this can also be induced by overzealous fluid administration. Studies have shown that dyspneic cats and dogs with cardiogenic pulmonary edema tend to have significantly higher LA/Ao ratios (roughly 2.0) compared to those with non-cardiogenic causes of dyspnea (typically 1.1). This view is thus crucial in differentiating the cause of dyspnea and performs as well as an NT-proBNP snap test, with less confounding factors. As many dyspneic patients, especially cats, can be intolerant to be placed in lateral recumbency, a study was conducted to confirm that values obtained in standing or sternal position are similar than those in lateral recumbency.

3. **Right Parasternal Long-Axis Four-Chamber View**:

- **Description**: This view is achieved by rotating the probe from the short-axis position along the long axis of the heart, allowing visualization of all four chambers (left and right atria, left and right ventricles).

Clinical Relevance:

- **Left Ventricular Contractility**: This view provides a subjective assessment of left ventricular contractility. Hypokinesis or akinesis of the left ventricle can indicate systolic dysfunction, often seen in conditions such as dilated cardiomyopathy.

- **Left Ventricular Filling**: The size of the left ventricular lumen can indicate diastolic function. A small, underfilled left ventricle may suggest hypovolemia, while a dilated ventricle may indicate dilated cardiomyopathy or severe volume overload.

- **Phenotype Screening**: This view allows differentiation between hypertrophic and dilated cardiomyopathy phenotypes. Hypertrophic cardiomyopathy (HCM) will show thickened ventricular walls with a small lumen, while dilated cardiomyopathy (DCM) will show a dilated lumen with thin walls and poor contractility.

Drainage of the pulmonary vasculature and pleural sheets

Dogs with Left-Sided Congestive Heart Disease (L-CHF) typically present with increased B-lines on lung ultrasound, indicating interstitial or alveolar edema. The pleural line remains regular and smooth. Right-sided heart disease in dogs may result in pleural effusion. This condition is often associated with signs of systemic venous congestion, such as ascites.

In cats, both left-sided and right-sided heart disease can lead to pleural effusion, which appears as an an-echoic (black) space between the lungs and chest wall on ultrasound.

Conclusion

A basic cardiac ultrasound using POCUS is invaluable for the assessment of dyspneic dogs and cats. The three basic cardiac views—transventricular (mushroom), left atrial and aortic (Mercedes/whale), and four-chamber—provide critical information about cardiac structure and function. The LA/Ao ratio is especially important in distinguishing cardiogenic from non-cardiogenic causes of dyspnea. The evaluation of right-sided heart disease through the transventricular view, particularly with tools like the 10-point score and eccentricity index, enhances the clinician's ability to diagnose pulmonary hypertension.

THE INITIAL TREATMENT OF THE DYSPNEIC PATIENT

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Managing an animal with severe respiratory distress is one of the most challenging tasks for emergency clinicians. Effective management requires a keen awareness of the fragility of dyspneic patients. The stress from life-threatening conditions, combined with transport and the unfamiliar environment of a noisy emergency clinic, should never be underestimated. Even a brief evaluation can be fatal, especially for cats, so the initial major body system assessment may need to be done in stages. All dyspneic animals should be given supplemental oxygen immediately using the least stressful method available. Additionally, before performing any diagnostic tests, the risks of any procedure should be carefully weighed against the potential benefits. To understand the importance of this, consider the following: in your experience, what is the most common cause of death in dyspneic cats? Do they die spontaneously, or do they die during interventions? Unfortunately, it is usually the latter, indicating that we must be extremely cautious with this patient group. Many cases will stabilize to some extent with oxygen and stress reduction alone. Therefore, our greatest challenge with these patients is to have the confidence to do nothing other than provide oxygen for a while, even though our instincts urge us to take action.

1. Oxygen supplementation

At presentation, a patent airway should be assured. If the airway is not patent, then attempts should be made to clear the airway and intubate the patient. If an obstruction prevents intubation, then a trans-tracheal catheter or emergency tracheostomy should be performed if it will by-pass the obstruction and assure a patent airway. To provide perspective, it is extremely rare that an emergency tracheostomy is required. Most patients can be intubated, and the tracheostomy then performed in a more controlled way. Immediate oxygen supplementation should be provided while assessing for a patent airway. Our first choice is via mask or flow-by. This is convenient, inexpensive, and provides an opportunity to examine the patient as well as do procedures if necessary. Other modes of oxygen supplementation include an enriched oxygen environment (oxygen cage or tent), nasal oxygen, high flow oxygen therapy and intubation with positive pressure ventilation. Each has its advantages and disadvantages that will be discussed during the lecture.

2. Stress release

All procedures causing stress should be avoided when admitting a dyspneic animal. Therefore, the method of oxygen therapy should be as stress-free as possible, and additional examinations are often deferred. In many cases, sedation is useful. The most suitable drugs for sedating a dyspneic patient are:

- **Opioids**, which have a significant sedative effect, pulmonary vasodilator properties, analgesic effects (allowing increased respiratory movement amplitude during pain), and reduce respiratory rate (improving pulmonary exchange). The most commonly used are:

- Butorphanol: 0.1 to 0.3 mg/kg SC, IM, or slow IV: This is the most suitable molecule for managing a dyspneic animal. If the animal is not in pain, butorphanol should be the first choice for sedation.
- Morphine: 0.1 to 0.3 mg/kg SC, IM (beware of induced vomiting)
- Fentanyl: 1 to 3 µg/kg IV: It has good sedative properties and excellent analgesic properties. It is very useful in cases of severe pain and dyspnea. However, it only acts for 10-15 minutes, and a continuous infusion should be set up to maintain its analgesic effect (1-3 µg/kg/h).
- Methadone: 0.1 to 0.3 mg/kg SC, IM, IV. Good sedative properties, excellent analgesic properties. It can sometimes cause intense polypnea in dogs, which is harmful in cases of respiratory distress.
- Buprenorphine: It has an onset time of about 45 minutes, making it unsuitable for emergency management of an animal and should not be used as a first-line treatment.
- Dexmedetomidine: Used at low doses (0.5-2 µg/kg IV), it offers excellent sedative properties while limiting cardiovascular effects. It is very useful when the animal is highly stressed but does not have cardiovascular failure (hypovolemia). It can also be used in continuous infusion (0.5-3 µg/kg/h), with the dose adjusted according to the animal's response. It is increasingly used in intensive care, particularly for managing stress-associated dyspnea in brachycephalic breeds.
- Acepromazine: Its use should be reserved for hemodynamically stable animals, as it causes vasodilation, which can be dangerous. It is used in small doses (0.01 to 0.05 mg/kg SC, IV).

It is important to titrate these drugs (starting with the smallest dose, then repeating the dose until the desired effect is achieved), as patient reactions to a given dose are difficult to predict.

3. Triage: take the most of your physical examination

Triage is the process of 'sorting out'/prioritizing treatment necessities based on urgency. It is a very quick procedure and should be done immediately on patient arrival at the clinic.

Performing diagnostic tests (radiographs, blood tests, etc.) can pose a serious danger to a dyspneic animal. Observing the respiratory rate and pattern, along with auscultation of the lung fields, usually allows localization of the respiratory disorder's origin and the initiation of effective emergency treatment. Thus, an upper respiratory tract involvement will result in inspiratory dyspnea associated with significant inspiratory noises audible without a stethoscope, a lower respiratory tract involvement will result in expiratory dyspnea (mainly in cats) or mixed dyspnea, and pleural or diaphragmatic involvement will result in discordance. In most cases, dyspnea is characterized by an increased respiratory rate. A decrease in this respiratory rate should direct us towards muscular or neuromuscular disorders, or extreme fatigue of the respiratory muscles.

This examination allows, within a few minutes, to have a good idea of the lesion's location and to adapt the specific management steps.

4. Specific Stabilization Techniques

Upper Airway

These cases are usually easy to identify because you can hear them without a stethoscope as they have loud stertor or stridor. Fixed airway obstructions (mass, non-moving foreign body) will result in increased effort and prolongation of both the inspiratory and expiratory phases of the respiratory cycle. Dynamic airway obstructions (tracheal collapse, laryngeal paralysis) will have increased effort and prolongation of either the inspiratory or expiratory portion of the respiratory cycle. Inspiratory dyspnea suggests that the problem is outside of the thorax and expiratory dyspnea suggests it is something associated with the intra-thoracic trachea or large airways. In severe cases of distress, intubation and/or tracheostomy may be necessary. Both interventions will only alleviate proximal airway obstructions. In most situations, it is possible to obtain endotracheal intubation even if a smaller than normal tube or the use of stylet is necessary. While tracheostomy may be needed for long-term management of some conditions, it is rarely a truly emergent procedure as most of the time some other airway access can initially be achieved. Paying careful attention to body temperature and overheating is also important, especially in dogs. Hyperthermia will profoundly increase respiratory drive, which is detrimental in cases of upper airway obstruction, and often worsens airway narrowing in cases of dynamic obstruction. Hyperthermic patients will require active cooling in addition to sedation before their respiratory effort lessens.

Potential Causes

Brachycephalic airway syndrome, nasopharyngeal polyps, nasopharyngeal foreign bodies/infection, nasopharyngeal edema, laryngeal paralysis, inflammatory laryngeal disease, tracheal collapse, tracheal stenosis/stricture, tracheal foreign body, upper airway neoplasia, etc.

Hallmarks

Stertor/stridor that you do not need a stethoscope to hear, often have prolonged or dyspneic inspiratory or expiratory phase or both depending on whether the obstruction is dynamic or fixed. In severe cases abdomen will pull inward during inspiration.

Lower Airway Disease

Cats with asthma are probably the most frequent example of this localization. Lower airway disease is typically manifested as a prolonged and difficult expiratory phase with an expiratory push. Auscultation may reveal expiratory wheezes or harsh lung sounds. In the short-term bronchodilators, in addition to sedation, are often helpful in alleviating clinical signs.

Treatment associate corticosteroids and bronchodilators:

- **Terbutaline** (β_2 -agonist): 0.01 to 0.05 mg/kg SC, IM, IV. Improvement occurs within 15 to 30 minutes. Avoid in cats with HCM as it induces tachycardia.
- **Salbutamol** (β_2 -agonist): 2 puffs administered via an inhalation chamber, allowing the animal to breathe for 10 to 15 cycles.
- **Ipratropium bromide** (Anticholinergic): same dosage as salbutamol. It is beneficial to combine salbutamol and ipratropium bromide as their actions have been shown to be synergistic.
- **Reducing inflammation:**
 - **Dexamethasone**: 0.1 mg/kg SC, IV. Preferred over prednisolone.
 - **Prednisolone**: 0.5-1 mg/kg IV.

Potential Causes

Feline asthma, allergic airway disease, eosinophilic bronchopneumopathy, pulmonary infiltrates with eosinophils, parasitic pulmonary disease.

Hallmarks

Expiratory difficult with expiratory push, lung sounds may be diffusely harsh to wheezes.

Parenchymal Disease

These patients can typically move air readily but are dyspneic because they cannot exchange oxygen. Typically, they will not exhibit more apparent inspiratory vs. expiratory dyspnea but will show increased work of breathing (labored) overall. Auscultation will generally reveal harsh lung sounds or crackles. There are many possible causes of parenchymal disease, so paying attention to other findings is also necessary to try and determine the cause. Fever may be more indicative of pneumonia. A history of pulling on the leash, seizures, electrocution, or strangulation may indicate non-cardiogenic pulmonary oedema. Congestive heart failure (CHF) may be suspected based on signalment, history of known cardiac disease, appearance of increased left atrial:aortic diameter on thoracic ultrasound, or the presence of an arrhythmia. Nearly all dogs with CHF will also have an auscultable murmur, typically grade III/VI or higher. Unfortunately, the presence or absence of a murmur does not rule in/out CHF in cats. Patients with CHF will also tachycardic (unless they have a pathologic bradycardia causing their failure, i.e., 3rd degree AV block). Hypothermia is common in CHF as well. Classic patterns seen on x-rays include interstitial to alveolar patterns, sometimes with characteristic distribution depending on underlying disease. Severe parenchymal disease may require intubation due to respiratory fatigue or to provide higher levels of respiratory support (oxygen, +/- PEEP); both of which will then necessitate mechanical ventilation for a period of time.

In case of high suspicion of CHF, diuretic therapy should be started:

- **Furosemide**: 1-2 mg/kg SC or IV in cats, 2-4 mg/kg in dogs.

- Then in continuous infusion: 0.5 (cat) – 1 (dog) mg/kg/h for 6 hours.
- If continuous infusion is not possible, it is better to give small regular doses (1 mg/kg q4h) rather than the same dose all at once.

When cardiac auscultation is normal, the history will be important: the presence of vomiting before the consultation will suggest aspiration bronchopneumonia, and trauma will suggest pulmonary haemorrhages. Etiological treatment should then be implemented (IV antibiotics for bronchopneumonia, tranexamic acid (10 mg/kg IV TID) for pulmonary contusions).

Potential Causes

Contusions, congestive heart failure, fluid overload, non-cardiogenic pulmonary oedema, acute lung injury/acute respiratory distress syndrome, aspiration pneumonia, other infectious pneumonias, neoplastic infiltration.

Hallmarks

B-lines (focal or diffuse) in lung fields, increased lung sounds (harsh to crackles), can be associated with heart murmur.

Pleural Space Disease

These patients will typically display rapid, shallow respirations, and with severe effusions can display a dysynchronous respiratory pattern with paradoxical abdominal movement. Animals with severe thoracic wall trauma (rib fractures, flail chest) can also present similarly and will only voluntarily take short shallow breaths. In these cases, aggressive analgesia is typically hugely helpful to allow them to take more normal breaths and alleviate their respiratory distress. For animals with true pleural space disease, auscultation will reveal quiet or decreased lung sounds. For pneumothorax this will be most pronounced dorsally and for effusions this will be most prominent ventrally. Thoracic POCUS is highly sensitive and specific for identifying effusions in the thoracic cavity. While it may be helpful, it is much less reliable when detecting pneumothorax, but the absence of a glide sign does correlate to the presence of a pneumothorax. The definitive treatment for pleural space disease is thoracocentesis and it is diagnostic as well in the case of effusion. Thoracocentesis is a relatively low-risk procedure so if physical exam findings support the suspicion of pleural space disease you should feel confident in performing at least a diagnostic tap prior to performing radiographs.

Potential Causes

Hemothorax, pneumothorax, chylothorax, pyothorax, diaphragmatic hernia, neoplastic effusion, right heart failure (dogs), left heart failure (cats), thoracic wall disease (rib fracture, flail chest).

Hallmarks

Rapid and shallow respirations may have paradoxical abdominal motion as well. Decreased lung sounds, dorsally for pneumothorax, ventrally for effusions.

Hypoventilation

Hypoventilation can sometimes be apparent, but in many cases it is not. Patients demonstrating hypoventilation will have a normal to increased PCO₂ in the face of hypoxemia, which can be measured on a venous or arterial blood gas. Fish-mouthing with little chest-wall motion or abdominal breathing can be overt signs of hypoventilation. These are more often seen with failure of the muscles of respiration due to central nervous system or lower motor neuron disease. These patients are also often hypoxic due to their hypoventilation and oxygen supplementation will correct the hypoxemia. However, it will not improve their ventilatory function and they will still progress to respiratory arrest unless their underlying condition can be rapidly corrected, or they are given assisted ventilation. Respiratory fatigue is a common cause of hypoventilation and can occur in animals that appear to be making significant efforts to breathe. The muscles of respiration are composed of striated muscle like other voluntary muscles, and with extreme use they can reach a point of fatigue where they are no longer able to function adequately. Clinically, they often appear as though they cannot get comfortable even in oxygen—they may fall asleep standing or sitting up or be unable to lay down, and they simply do not look like they are breathing comfortably.

In case of severe hypoventilation, mechanical ventilation will be the only option.

Potential Causes

Sedation due to drugs, caudal brainstem disease, cervical myelopathy, lower motor neuron disease, severe airway obstruction, severe pleural space disease, severe thoracic wall disease, respiratory fatigue.

Hallmarks

Elevated or sometimes normal PCO₂ in the presence of hypoxemia, breathing looks uncomfortable and patient is unable to rest despite supportive measures.

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ADVANCED TREATMENT OF THE DYSPNEIC PATIENT: THE BASICS FROM HIGH FLOW TO MECHANICAL VENTILATION

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Oxygen administration is frequently used in veterinary medicine to treat hypoxia. Noninvasive or conventional oxygen administration methods are readily available in most hospitals or clinics and don't require specialized equipment or skills to administer to the patient. Conventional oxygen therapies include nasal cannula, oxygen cage, hood oxygen and flow by/mask administration. With conventional oxygen therapy (COT), FIO₂ is limited to 30–75% and no significant pressure support is provided. In patients with severe hypoxemia (PaO₂ < 60 mm Hg) who fail COT, more aggressive treatment is warranted. The purpose of this lecture is to describe advanced oxygen therapy techniques that could be used in small animal patients. Advanced oxygen therapies can be divided into two groups:

- Advanced invasive oxygen therapies requiring intubation.
- Advance noninvasive oxygen therapies: advanced oxygen delivery methods that do not require intubation.

1. Conventional oxygen therapy

Conventional oxygen therapy can be supplied from various sources (eg, centralized in-house oxygen, portable oxygen tanks, anesthetic machines) and in many different ways, depending on the severity of respiratory distress, the need to handle the patient while providing oxygen, the duration supplementation is needed, the available equipment, and the clinical experience and skills of the clinician. Pro and cons of the different COT are described in the table below.

Oxygen administration technique	Oxygen concentration attainable	Advantage	Limitations
Flow-by	25-40%	<ul style="list-style-type: none"> • Easy to implement • Keep patient accessible for examinations and procedures 	<ul style="list-style-type: none"> • Might stress patient • Limited FiO₂ • Requires someone to hold tubing • Only useful short-term
Face mask	50-60%	<ul style="list-style-type: none"> • Easy to implement • Higher FiO₂ • Keep patient accessible for examinations and procedures 	<ul style="list-style-type: none"> • Might stress patient • Can cause rebreathing of CO₂ if mask is too tight
Elizabethan collar	30-40%	<ul style="list-style-type: none"> • Higher FiO₂ • Limited equipment • Keep patient accessible for examinations and procedures 	<ul style="list-style-type: none"> • Might stress patient • Humidity, CO₂, and temperature can increase in the hood
Nasal	Up to 50% with bilateral catheters	<ul style="list-style-type: none"> • No operator required • Allows transport, examinations and care without discontinuing oxygen 	<ul style="list-style-type: none"> • Not tolerated in all patient • Difficult in brachycephalic • Panting can reduce effectiveness • Nasal irritation
Oxygen cage	Up to 100%	<ul style="list-style-type: none"> • Minimizes patient stress, allowing stabilization • Higher FiO₂ 	<ul style="list-style-type: none"> • Limited to no patient access • Expensive to purchase equipment and use • Oxygen concentration rapidly fall when door is opened

Fortunately, COT provide adequate oxygen support for the majority of our patients.

When COT fails to improve respiratory distress in dogs and cats, advanced oxygen therapy technique can be used, as mechanical ventilation or high flow oxygen therapy.

2. Mechanical ventilation: indication and cares

Patients are placed on a ventilator for three main reasons:

- Severe hypoxemia despite oxygen supplementation (PaO₂ < 60 mmHg with oxygen or SpO₂ < 90%)
- Severe hypoventilation despite therapy (PCO₂ > 60 mm Hg)
- Excessive respiratory effort with impending respiratory fatigue or failure.

It has been suggested that patients requiring short-term ventilation have better outcomes than patients requiring long-term ventilation¹. Multiple complications associated with long-term ventilation have been documented, many of which are related to nursing care issues². These include oral and corneal ulceration, tracheal tube occlusion or dislodgment, and gastric distension requiring decompression. The importance of nursing care for ventilator patients in human medicine is highlighted by an increased risk of late- (but not early-) onset ventilator-associated pneumonia (VAP) with lower nurse staffing level³. Ideally each patient on the ventilator should have a dedicated veterinary technician at all times.

a. Airway management

Airway management and monitoring is of high importance to avoid VAP and VILI (ventilator induced lung injury). Airway management consists of endotracheal tube (ET) care, humidification of the airways and airway suctioning.

i. Care of the endotracheal tube

Patients undergoing mechanical ventilation need intubation with either an ET or a tracheostomy tube. Ide-

ally the ET tube should be sterile and have a low-pressure cuff and the intubation process performed with sterile gloves. Whenever the airway is being handled after intubation, hand hygiene should be performed first, and examination gloves used. To help prevent tracheal necrosis, it has been suggested to deflate the cuff and reposition it every 4 hours in veterinary medicine.

ii. Humidification of the airways

Lack of humidification leads to increased mucus viscosity and volume, which can cause ET tube occlusion, tracheal inflammation, and depressed ciliary function. There are two major methods of airways humidification: heat and moisture exchangers (HME) and hot water humidifiers. The choice to use one method instead of the other depends on both technical and economic considerations. HMEs are more often used nowadays because they are simple to use and cost-effective. Their performances are for several models comparable to those of a heated humidifier. They are usually changed after 24 h of use. It has recently been shown that some HMEs can be changed only every 48 h and that at least one HME can be changed only once a week in some patients⁴. Humidification could also be done with nebulization of saline, synchronized with the inspiration. Other nebulized medications that could be used in ventilated patient are hypertonic sodium chloride (...%) (mucolytic action, increased muco-ciliary function, anti-inflammatory), antibiotic when infectious disease is suspected (e.g.: Gentamicin), and heparin (specially in case of ARDS and ALI induced by smoke inhalation).

iii. Airway suctioning

Suctioning of the airway is of key importance to help prevent ET tube occlusion with airway secretions. In the awake patient, coughing helps clear secretions; however, the cough reflex is blunted or absent in the anesthetized patient. Suctioning can be performed by either an open or closed system suction method. It should be performed every 4 hours or more frequently on an as-needed basis; and in a pre-oxygenated pet.

Specific techniques of chest physiotherapy can help expectoration of secretion in ventilated patient and will be developed during the lecture.

b. Specific organ cares

Oral, eye, urinary, gastrointestinal and recumbent patient care is mandatory. Patients anesthetized for prolonged periods such as ventilated patients can develop a significant number of complications involving these organs⁵. The eyes should be kept well lubricated to prevent ulceration. The mouth should be regularly cleaned of secretions and flushed with an oral rinse (e.g., Chlorhexidine) and a glycerin solution should be applied to the tongue to help prevent lingual drying and damage. Ideally, urinary tract care should involve a urinary catheter being placed to keep the patient clean and dry (as well as providing the added benefit of being able to monitor hydration status and kidney function). Just be aware that it carries the risk of development of bacteriuria either from true urinary tract infection or colonization of the catheter. So, urinary catheter care should be performed every 8 hours. Nutritional support of ventilator patients can be quite challenging as they are usually sedated, if not fully anesthetized, and unable to protect their airway making them high risks for regurgitation and aspiration.

Furthermore, anesthetic ileus can be an issue if enteral feedings are to be administered. Gastric distention should be monitored for closely and gastric residual volumes should be assessed if possible prior to feedings. Enteral nutrition in mechanical ventilation carries risks and benefits. Enteral feeding may be delivered via a nasogastric, gastrotomy, or jejunostomy tube⁶. In light of these previously stated concerns, these patients become good candidates for parenteral nutrition instead.

Body positioning should be changed every 4-6 hours and adequate padding should be provided on ventilator patients because they are at high risk for pressure sores and nerve damage. Passive range of motion exercises should also be part of the patient care as these patients are prone to muscle atrophy especially if they are on the ventilator for longer than 48 hours.

c. Monitoring

Other parameters that should be monitored on ventilator cases include electrocardiograph monitoring, pulse oximetry, capnography, arterial blood pressure and continuous temperature. The different probes should be replaced every 4 hours to avoid tissue damage. When possible, these patients should have an arterial catheter for routine arterial blood gas sampling and invasive pressure monitoring. Blood pH, glucose, lactate and electrolytes should be serially monitored as well, especially if parenteral nutrition is being administered.

3. Noninvasive advanced oxygen therapy: focus on high flow oxygen therapy

High flow oxygen therapy (HOT) is a nasal oxygen delivery system that has been used in people for over 10 years but is relatively new to veterinary medicine. High flow nasal cannula accurately delivers humidified and heated oxygen to the patient.

HOT is carried out using an air/oxygen blender, active humidifier, single heated tube, and specific nasal cannula. Those specific nasal cannulas should ideally be 50% or less the diameter of the nares.

Able to deliver adequately heated and humidified medical gas at higher flows than COT, HOT is considered to have a number of physiological advantages, including reduced anatomical dead space, PEEP, constant FIO_2 , and good humidification. Initial studies looking at the use of HFNC in veterinary patients have been encouraging.

A pilot study looking at healthy dogs showed a significant increase in PaO_2 in dogs receiving HOT at 20 and 30 L/min compared to dogs receiving conventional nasal oxygen at 100 ml/kg/min⁷. A retrospective study looking at 6 dogs undergoing HOT for hypoxemia not responsive to conventional oxygen therapies showed that hypoxemia resolved in 4/6 dogs with HOT⁸. Moreover, this study showed that HOT was well tolerated and with few adverse effects. Our team published a prospective study on the use of HOT in dyspneic dogs, showing a significant increase in PaO_2 and SpO_2 with HOT compared to nasal oxygen therapy. Tolerance was excellent⁹. We also describe the use if HOT in cats, with the same benefits, despite the fact that placing the nasal canula can be more challenging¹⁰.

Usual flow rate for conventional oxygen therapy is 150 ml/kg/min for nasal cannula (e.g. 1.5 L/min for a 10 kg dog). With HOT, the recommended flow rate is 1 to 2 L/kg/min¹¹. So, for our 10 kg dog, the initial flow rate will be of 10 to 20 L/min. The flow rate could be slightly adjusted based on the patient comfort. With this flow rate, FIO_2 can be precisely determined. Usually, FIO_2 is started at 100%, then oxygen is mixed with air to decrease FIO_2 and avoid oxygen toxicity.

HOT has also been described during bronchoscopy in dogs and cats, reducing the risk of hypoxemia¹².

Conclusion: Prompt recognition of hypoxemia and institution of oxygen therapy is important in correcting tissue hypoxia. Equally important is recognizing the need for escalation of oxygen therapy in patients not responding to conventional oxygen delivery methods. In veterinary patients, intubation is associated with higher morbidity and mortality. High flow oxygen therapy is a new and promising advanced oxygen therapy technique.

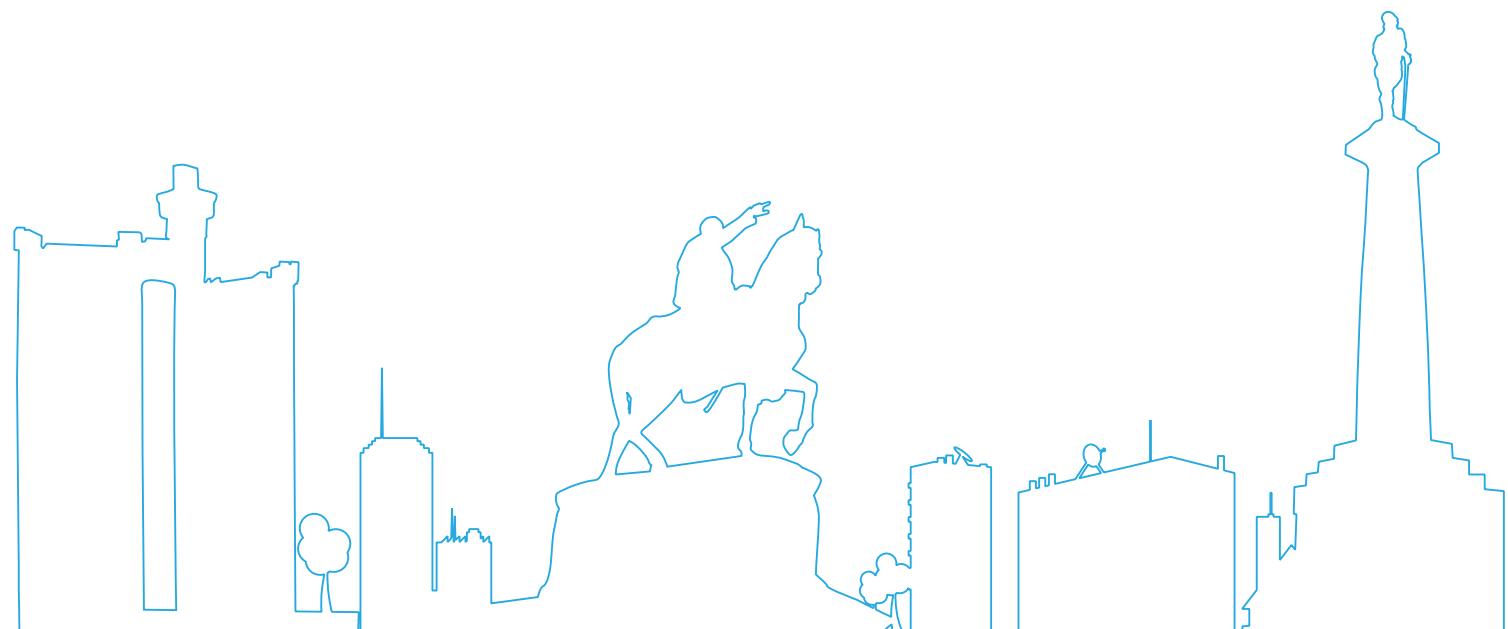
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EXOTICS



**Norin Chai (France)**

DVM, MSc, MScVet, PhD, Dipl.ECZM

EERVC 2024 Lectures

1. Pain management in exotic medicine
2. Dermatology in small mammals
3. Ophthalmology in exotic medicine
4. Update in anaesthesia and analgesia of birds and reptiles
5. Reproductive surgery in exotic medicine
6. Primate medicine for practitioners

Graduated from the National Veterinary School of Alfort, Norin completed two Masters of Science in 1995, one in "Tropical Animal Pathology" and one in "Tropical Animal Production". In 1996, he completed his veterinary thesis on "Ecology and ethology of the leopard (*Panthera pardus*)". In the meantime, he began his career as the director of the Manda National Park (Chad) from 1995 to 1996. In 1997, he joined the National Museum of Natural History (France) as a research engineer, deputy director and Head vet of the Parc de la Haute Touche (zoological park in central France). In 2000, Norin returned to Paris as a veterinarian at the Ménagerie du Jardin des Plantes. In 2008, he obtained his PhD on amphibian medicine. In 2013, he became Diplomate of the European College of Zoological Medicine (Zoo). From 2005 to September 2020, he held the position of headvet and deputy director of the Menagerie. At the same time, Norin was the veterinary manager of all the transgenic animal research facilities of the National Museum, president of the NGO Elefantasia and co-founding president of the Regional Wildlife Monitoring Laboratory. He left his position at the zoo in October 2020 to follow his own personal conservation and humanitarian projects, in particular with his association Yabouumba, founded in 2000. He is regularly requested for expertise on medical and surgical cases in wildlife and on conservation projects around the world.

Currently, Norin consults in a referral veterinary clinic specialized in exotic animals (Spenac Argos, Paris), in a zoo-rescue Center (La Tanière Zoo, Chartres), in other several rescue centers (Costa Rica and Asia) and has a business coaching activity.

He has published numerous articles in indexed veterinary journals, national magazines and given hundred lectures in national and international conferences.

PAIN MANAGEMENT IN EXOTIC MEDICINE

Norin Chai, DVM, MSc, MScVet, PhD, Dipl. ECZM

Pain is a complex and multifaceted experience that serves as a critical biological warning signal, indicating potential or actual harm to an organism. It is both a sensory and emotional experience, influenced by a variety of factors including the nature of the stimulus, individual perception, and the context in which it occurs. Untreated pain can lead to chronic stress, immunosuppression, worsening of chronic conditions, delayed healing and recovery. Alleviating pain improves the animal's quality of life and encourages normal behaviors. Pain management in exotic animals presents unique challenges due to the vast diversity of species, each with distinct anatomical and physiological characteristics.

This lecture will first cover various signs of pain in exotic species, including behavioral and physiological indicators, and highlight the subjectivity and variability in pain perception across different species. The lecture will then discuss pharmacological pain management, detailing not only the use of classical analgesics such as opioids, NSAIDs and local anesthetics but other drugs. We will address species-specific considerations, including variations in drug metabolism and sensitivity, supported by case studies of common exotic animals. Additionally, non-pharmacological pain management strategies will be discussed, encompassing environmental modifications, physical therapies, laser therapy and nutritional support.

DERMATOLOGY IN EXOTIC MAMMALS

Norin Chai, DVM, MSc, MScVet, PhD, Dipl. ECZM

Exotic mammals, particularly four-toed hedgehogs, ferrets, rabbits, and rodents, often present unique dermatological challenges in veterinary practice. This lecture will focus on the most common dermatologic abnormalities in these species, emphasizing the importance of accurate diagnosis and effective management.

In four-toed hedgehogs, Acariasis, dermatophytosis, and cutaneous neoplasia are the most prevalent skin conditions. The hedgehog mite, *Caparinia tripilis*, is easily identified by the three long setae on the third pair of legs. Transmission of *Trichophyton* infections from hedgehogs to humans necessitates strict hygiene practices. Cutaneous neoplasia is frequently observed, with diagnostic approaches beginning with fine-needle aspiration and often requiring biopsy for definitive diagnosis.

Ferrets commonly experience alopecia due to adrenal gland disease, hyperestrogenism, seasonal alopecia, fleas, and neoplastic conditions. Pruritus in ferrets can result from infectious diseases as well as endocrine and neoplastic conditions, including hyperadrenocorticism, lymphoma, and mast cell tumors. The lecture will also cover Blue Ferret Syndrome, a benign condition where the skin acquires a bluish tint after fur clipping, resolving without treatment. Zoonotic skin diseases in ferrets, such as sarcoptic mange, leishmaniasis, and dermatophytosis, will also be discussed.

In rabbits, ectoparasites are the primary cause of pruritus. The use of fipronil is contraindicated due to toxicity risks. Serologic tests for syphilis in humans are applicable for diagnosing rabbit treponematosis. Additionally, domestic rabbits are highly susceptible to myxomatosis.

Dermatologic conditions in pet rodents are a frequent concern, with acariasis, dermatophytosis, bacterial infections, endocrinopathies, neoplasia, and environmental and husbandry-related issues often presenting as multifactorial problems. Successful management requires a thorough history, comprehensive husbandry review, and diagnostic evaluation.

OPHTHALMOLOGY IN EXOTIC ANIMAL MEDICINE

Norin Chai, DVM, MSc, MScVet, PhD, Dipl. ECZM

The eyes represent an essential organ system that is often not extensively evaluated during routine physical examinations. While ocular exams are common, they are frequently cursory. A thorough ophthalmic examination can provide valuable insights not only into the ocular system but also into the overall health of the patient, revealing conditions such as sepsis or hypertension. Exotic pets can develop many of the same ophthalmic diseases seen in domestic species. However, vision loss in exotic pets, particularly prey species such as rabbits and rodents, can significantly impact their welfare and quality of life. Therefore,

veterinarians must perform comprehensive ophthalmic examinations and be proficient in recognizing various ophthalmic diseases in exotic pets. This lecture reviews the ophthalmic anatomy and common disease conditions in exotic pets, alongside potential treatments.

In amphibians, the most frequently described condition is corneal lipidosis in frogs, with other issues including traumatic and infectious corneal ulcers and uveitis. Chelonians and other reptiles are particularly susceptible to hypovitaminosis A, and conjunctivitis, which can result from ocular diseases, dietary deficiencies, environmental irritants, infections, or foreign material. Reptiles can also suffer from periocular masses and spectacle-related disorders (in snakes), with cataracts becoming more common as husbandry improves and lifespans increase.

In birds, trauma-induced conjunctivitis, including third eyelid lacerations, is common and often inflicted by cage mates. Bacterial and parasitic conjunctivitis also frequently occur. Corneal ulcers in birds typically result from trauma and are treated similarly to other species. Raptors are particularly prone to intraocular inflammation, hyphema, vitreal hemorrhage, and retinal detachment due to trauma.

Domesticated rabbits commonly suffer from blepharitis, which may be primary or secondary to nasolacrimal duct obstruction, often due to dental disease or rhinitis. Bacterial infections, notably from *Pasteurella multocida* and *Staphylococcus aureus*, are also very common. *Encephalitozoon cuniculi* in rabbits can cause various ocular signs, including masses in the iris, ocular hypotension, cataracts, and secondary glaucoma. Unilateral exophthalmos in rabbits is typically linked to orbital abscesses, primarily due to dental disease, but can also arise from orbital neoplasia, parasitic cysts, or other conditions. Ocular disorders are less frequently reported in pet rats and mice, but conjunctivitis caused by *Chlamydia caviae* is common in laboratory colonies and has also been observed in pet guinea pigs.

By understanding these conditions and their treatments, veterinarians can significantly improve the welfare and quality of life of exotic pets through effective ophthalmic care.

UPDATE IN ANAESTHESIA AND ANALGESIA OF BIRDS AND REPTILES

Norin Chai, DVM, MSc, MScVet, PhD, Dipl. ECZM

Reptiles

Reptiles especially aquatic animals, commonly exhibit species and individual differences in response to anesthetic drugs, dosages, and protocols. Thermoregulation in a healthy animal is accomplished by behavioral adaptations; however, sick animals often fail to effectively regulate their body temperature. The response to anesthetic drugs, as well as cardiopulmonary performance during anesthesia, is optimal if the patient is maintained within the species-specific preferred optimal temperature range. Most anesthetic drugs have cardiovascular depressant effects. Most agents, especially when used alone at high dosages, cause pronounced cardiopulmonary depression, prolonged induction and recovery times, and poor muscle relaxation during maintenance of anesthesia. Ketamine HCl is used in Reptiles for both immobilization and induction of anesthesia. It has a wide margin of safety and can be administered IM for immobilization and IV for induction of anesthesia. Ketamine alone provides poor muscle relaxation, minimal analgesia and, if used at high dosages, prolonged recovery times. Ketamine is rarely used alone because high dosages are required in most species to produce immobilization. Most commonly, ketamine is combined with benzodiazepines (i.e., diazepam, midazolam), opioids (i.e., butorphanol, buprenorphine) or the α₂-adrenergic agonist medetomidine or dexmedetomidine. A combination will reduce the ketamine dose; this results in more rapid and smoother induction and recoveries, improves muscle relaxation, and provides analgesia. Tiletamine/zolazepam (Telazol) can be used for immobilization and induction of anesthesia. At high dosages (greater than or equal to 6 mg/kg IM) it is associated with prolonged recovery (greater than or equal to 48–72 hrs), especially in African spur-thighed tortoises (*Geochelone sulcata*), and is not recommended. To facilitate handling of large chelonians (i.e., to gain access to a peripheral vein) a low dose of tiletamine/zolazepam (2–4 mg/kg IM) can be used. However, induction with propofol is then recommended. Medetomidine facilitates handling and concurrent administration of synergistic agents results in a plane of sedation effective for short procedures such as abscess debridement, shell repair procedures, and collection of diagnostic samples. It is most commonly combined with ketamine and an opioid (e.g., butorphanol). For reversal, atipamezole is administered IM at 5 times the medetomidine dose. Propofol has been used in a variety of reptile species for both induction and maintenance of anesthesia. It must be administered IV and the induction dosage depends on the health status of the chelonian and the type and amount of premedication. Propofol causes systemic hypotension, decreased myocardial contractility, and respiratory depression. Propofol (2 to 5 mg/kg IV) induces general anesthesia, especially following premedication with midazolam (0.5 to 1 mg/kg IV). Alphaxalone, is for the author a very useful drug ready for use. The author has found that 10 mg/ kg with IM injection give a very good sedation,

fast, enough for quick procedures or intubation. Anesthetic induction with inhalational agents alone often results in prolonged induction times, especially in aquatic animals capable of prolonged breathholding. Isoflurane results in short recovery times and minimal cardiopulmonary depressant effects, and has limited effects on renal and hepatic function.

Injection of sedative and anesthetic drugs in the caudal body-half should be avoided if possible, since some drugs may undergo a hepatic first pass effect, which may be more important for most anesthetic drugs, compared to the more often discussed renal first-pass effect. Plasma levels and efficacy of anesthetic drugs can be greatly reduced and may require repeated drug administration.

Historically the intramuscular route has been recommended as the route of choice for non-vascular administration. However, the subcutaneous route provides a suitable alternative, in particular in animals with reduced muscle mass or if larger volumes of drugs need to be administered.

During anesthesia, respiration and heart rates are recorded regularly. Supportive care depends on preanesthetic clinical and laboratory findings, the size of the chelonian, and the procedure to be performed. Fluid therapy is accurately administered, even to small animals, with a syringe pump; most require maintenance fluid administration of a balanced electrolyte solution at 5 to 10 ml/kg per hour. The patient is kept within the preferred optimal body temperature range for the particular species. The anesthetic plane is frequently assessed for the presence or absence of reflexes such as righting, palpebral, cloacal, and limb and tail withdrawal reflexes. At a surgical plane the righting and palpebral reflexes are usually absent. Absence of the corneal reflex and no response to a surgical stimulus indicates a deep plane of anesthesia. Electrocardiography (ECG) is performed with leads attached in a conventional manner and detects changes in heart rate such as tachycardia, bradycardia and arrhythmias. However, it does not evaluate mechanical performance of the heart. The use of pulse oximeters in Reptiles to monitor heart rate, arterial oxygen saturation (SpO_2), and diagnose hypoxemia during anesthesia is of limited value because all devices are calibrated based on the human oxygen hemoglobin dissociation curve. However, at present, pulse oximetry may be more useful to detect trends in arterial oxygen desaturation.

Our knowledge on reptile analgesia continues to grow. In general most mu-opioid receptor agonists have been shown to provide analgesia in turtles and tortoises as well as lizards and crocodilians. Drugs such as morphine, hydromorphone, fentanyl and tramadol are most commonly used.

Birds

Anesthesia and analgesia are an important part of the daily routine associated with avian veterinary practice. These procedures differ from mammal medicine primarily because of different physiologic composition and different anatomical structures, which are described when relevant to anesthetic management. Injectable anesthetic agents are typically metabolized in the liver and eliminated through the kidneys. Patients that are diagnosed with hepatic and/or renal disease may have reduced drug elimination, a long anesthetic recovery period, and concurrent cardiopulmonary depression. A surgical plane of anesthesia using injectable anesthetic agents is typically possible for up to 30 minutes, which can be used primarily for short surgical procedures, sedation for diagnostic purposes, and sampling during field studies. Local anesthesia is not commonly used in avian patients because small doses of local anesthetic drugs may have toxic effects and, most importantly, the patient remains conscious during a very stressful procedure. Ketamine should not be used as a single anesthetic agent, but may be used in combination with an alpha-2-agonist drug. In the same way, as alpha-2-agonists has cardiopulmonary depressive action, they should not be used as a monoanesthetic agent. Actually, midazolam is the most common drug used for sedation of pet birds and has a wide safety margin. Midazolam has sedative, muscle relaxing, anxiolytic, amnestic, and appetite-stimulating properties in birds. The injectable form of midazolam (midazolam hydrochloride, 5 mg/mL) or a more concentrated form (50 mg/mL) can be administered intranasally and/or intramuscularly without side effects. Dosages of midazolam commonly used in pet birds range from 0.5 to 3 mg/kg. The author routinely uses 2 mg/kg of midazolam in pet birds, if administered intranasally and as the sole sedative agent. Butorphanol is the most commonly used opioid analgesic in birds. Besides its analgesic effects, butorphanol also has sedative effects, which are potentiated by benzodiazepines (e.g., midazolam and diazepam). The combined administration of midazolam and butorphanol is recommended in birds for which midazolam alone provides an insufficient level of sedation or which require deeper sedation for certain clinical procedures (e.g., radiographic positioning). Butorphanol can be combined with midazolam into a single syringe and administered intramuscularly or intranasally. No side effects of intranasal administration of butorphanol at a dose range of 1 to 3 mg/kg have been reported in psittacines. The dose routinely used for intranasal or intramuscular administration is a combination of butorphanol (1 to 2 mg/kg) with midazolam (1 to 2 mg/kg) to pet birds.

When possible, inhalation is the method of choice for anesthesia in birds. Modern inhalation anesthetic agents have a low blood-gas solubility, which results in a rapid induction as well as a rapid recovery. Therefore, the concentration of administered anesthetic gas can be adjusted easily.

Basic considerations are similar to small mammal medicine and anesthetic circuits include nonrebreathing systems, pediatric circles, and adult circle systems. If necessary, the bird can be ventilated using volume- or pressure-controlled application systems, the latter having major advantages because they are patient-size independent. The main risk of inhalation anesthesia, especially in smaller birds, is hypothermia because of the large surface area of the air sac system.

Butorphanol (1-3 mg/kg IM) is the current recommendation for opioid analgesia in parrots. Buprenorphine at 0.1 mg/kg IM in African grey parrots did not show an analgesic effect when tested by analgesimetry. However, clinical use of buprenorphine suggests it has an analgesic effect.

REPRODUCTIVE SURGERY IN EXOTIC MEDICINE

Norin Chai, DVM, MSc, MScVet, PhD, Dipl. ECZM

Neutering small mammals

One of the main reasons for neutering exotic pet mammals is to control reproduction. However, medical and behavioral indications are also important factors to consider. In most species, castration makes male mammals less aggressive both to other animals and to their owners. The urine of many intact male mammals has a potent odor and is used for territorial marking. The odor and behavior can often be controlled by neutering the animal. In many species, mammary neoplasia is influenced by the presence of estrogen and ovarioectomy can decrease the occurrence of mammary cancer. Performing ovariohysterectomy effectively prevents pyometra and other uterine diseases. Because ovarian hormones influence most uterine diseases, ovarioectomy is expected to be nearly as effective at preventing female reproductive diseases as is ovariohysterectomy.

The testes of rabbits move freely between the abdomen and the scrotum through the function of a well-developed cremaster muscle. The inguinal canal is open in rabbits; however, the intestine does not herniate because of the large epididymal fat pad which fills the inguinal canal when the testes are within the scrotum and the inguinal fat pads within the abdomen. The proper ligament of the testis which attaches the tunica vaginalis to the scrotum is quite strong in rabbits. There is no fasting, to avoid hypoglycemia and paralytic ileus. Castration with uncovered testicles is the easiest and quickest surgical technique to perform. The spermatic and vascular cords are cut. Hemostasis is controlled. The cord is reintegrated into the vagina. The inguinal rings being open, closure of the vaginal ring is obligatory. It is made with a cross stitch. The scrotum is closed using surgical glue.

The uterus of young rabbits is found just dorsal to the bladder. The uterus and ovaries are generally easy to exteriorize; however, they are more fragile than those of dogs and cats. The uterus is bicornuate and each horn has its own cervix. There is no distinct uterine body. The mesometrium of rabbits is a site of fat storage. In obese rabbits this can make surgery more challenging, as it is often difficult to definitively identify the ovarian and uterine vessels for ligation.

The inguinal ring remains open throughout the life of the rodent. This anatomy determines the operating techniques used for the castration of these animals.

The testicles can migrate from an abdominal position to a scrotal position and vice versa. The practitioner during the intervention can change the position by simple pressure on the abdomen or scrotum. Castration with exposed testicles is preferred. The skin incision is made in the ante-scrotal or scrotal region depending on the species. There vaginal is incised and the testicle exteriorized without this envelope for its excision. Closure of the vaginal opening is essential to prevent any risk of inguinal hernia. This technique can be used in all rodents. It has the advantage of being quick and not exposing the abdominal cavity. A low risk of infection exists due to potential contamination of the wound near the anus and the urinary meatus.

Surgical intervention in birds

Surgical salpingohysterectomy or endoscopic salpingohysterectomy may be indicated in specific patients that are plagued with chronic egg laying problems. Salpingohysterectomized birds still retain their ovary, and hence may still be predisposed to estrogenic behaviors, hyperestrogenism, cystic ovarian disease, internal ovulation, and egg yolk peritonitis.

Unlike the ovary, birds have two testicles. Thus, surgery will need to be bilateral unless the contralateral testicle cannot be visualized. The testes are positioned cranioventrally to the cranial lobes of the kidneys and

are held in place by a mesorchium. The size of the testes increases dramatically during the reproductive period and it is recommended to wait for testicular involution or to induce it using hormones before planning castration. Indications generally include the prevention and treatment of behavioral problems, aggression, diseases linked to male hormones, testicular tumors, and sterilization in bird collections or for hybrids.

In avian medical practice, counter-hormonal therapies, including leuprolide acetate (Lupron[®]) and deslorelin, seem to be the more common treatments recommended for prevention as well as intervention for recurrent reproductive problems, and targeted behavioral or antecedent arrangement strategies seem to be less commonly implemented.

Reptiles

The vast majority of reproductive surgeries are for therapeutic purposes. In all cases, the realization requires perfect knowledge of the topography of the organs. The testes are close to the adrenal glands, and are connected to the anterior pole of the kidney by the mesorchium. The right testicle is always located more cranially than the left. The epididymis is absent in snakes, but very large in turtles where it can measure the entire length of the testicle. The genital tract finally opens into the cloaca, at the level of the urogenital sinus. Squamates have two hemipenis, embedded at the base of the tail. Turtles have a unique penis. Reptile ovaries are saccular structures that easily reveal follicles. We are talking about ovarian clusters. The ovaries are connected to the dorsal wall by the mesovarium, in continuity with the equivalent of the mesosalpynx which surrounds the oviducts. The oviducts are connected to the dorsal wall by the mesosalpynx. The left oviduct is sometimes atrophied or even absent in certain squamates. Most often in snakes, the right oviduct is the one that contains the developing eggs. As in birds, the oviduct can be subdivided into several parts: the infundibulum, the uterine horns, the isthmus, the uterus and the vagina. Mainly, indications for surgery are dystocia, population management, neoplasia.

PRIMATE MEDICINE FOR PRACTITIONERS

Norin Chai, DVM, MSc, MScVet, PhD, Dipl. ECZM (ZHM)

The nonhuman primates encountered in practice include few apes or prosimians. The overwhelming majority are simians weighing 10kg or less. Therefore, the author will often refer specifically to these monkeys in this presentation.

Management and preventive medicine

Primates are characterized by a large brain compare to other mammals, as well as an increased reliance on visual acuity. They have opposable thumbs although some such as colobus monkeys (*Colobus sp.*) have vestigial thumbs. Non-Human Primates (NHP) include a wide range of species with a wide range of characteristics.

Environment and psychological well-being care are then the first very important point to analyse and upgrade for NHP preventive medicine. Stereotypical behaviours or depression can be minimized with an enriched environment and with training. The program of enrichment should be appropriate for the species and continually evolving. Novel, stimulating challenges should be introduced, otherwise boredom will reemerge. Enrichment devices should be rotated often so that the novelty does not wear off. Many different forms of enrichment can be provided to encourage exercise and mental stimulation. Swings, perches, tunnels, hammocks, infant safe toys, mirrors, noise-makers, and water-play toys are staples in primate enrichment. Whenever possible, foraging behavior should also be encouraged; food items can be hidden throughout the cage. There are a multitude of homemade busy boxes and foraging devices that can be constructed, limited only by the imagination.

As with any other animals, the ability to successfully maintain many NHP in good health in captivity depends in a great part on providing diets that closely resemble the gross composition of wild diets. There are many commercial formulations (that should form the basis of the diet, 80 - 90% of the daily intake) available that can be supplemented with fresh produce (fruits...) or specialized foods such as nectars, gums or live insects. In general, nonhuman primates have a minimal 7% to 10% protein requirement (DM basis), and pregnant or lactating animals require 12.5% protein. Commercially prepared diets vary from 16% to 26.1% protein (lower protein diets are used for most nonhuman primates and higher protein ones for New World monkeys). All NHPs require vitamin C supplementation. New World primates require vitamin D3, while Old world primates can metabolize vitamin D2.

Physical examination should be proceeding everyday. Several sites are accessible for collecting blood samples. The femoral vein or artery in the region of the femoral triangle is the easiest site from where samples can be obtained in most primates. The posterior tibial vein, or small saphenous vein, running up the posterior aspect of the hindlimb, is an ideal site for intravenous injections and indwelling catheter placement. However, like the cephalic vein, it tends to collapse easily in small animals, making it suitable for collecting only small amounts of blood (typically < 1 millilitre). Jugular veins may be used for blood collection and catheter placement in anesthetized animals.

Annually, thorough clinical examination (ophthalmologic exam, gynecologic exam, ultrasound of the heart and reproductive track), dental examination, CBC, serum chemistries, fecal/rectal culture, TB testing should be scheduled. The tuberculin test site (eyelid) should be observed at 24, 48, and 72 hours for erythema and induration and described using standard criteria. Test interpretation may be complicated by falsepositive, false-negative, and nonspecific responses.

Because of the frequency of enteric and diarrheal diseases in NHP, fecal examination is one of the most commonly employed diagnostic tests. This should include direct examination of fresh thin fecal smears, as this is especially useful for the detection of protozoa such as ameba. Several techniques should be used: routine flotation examination (for helminth larvae or ova) and fecal sedimentation examination (detect the heavy ova of parasites such as *Prosthenorhynchus sp.* that are not detected by routine fecal floatation). Flagellates are frequently observed on direct fecal smear examinations of diarrheic feces. Every 6 months, fecal wet mount/flotation are followed by deworming treatment.

Restraint, anesthesia and analgesia

In general, animals weighing less than 5 kg can be restrained safely by one person, while larger animals often require either a second person or chemical restraint. As usual preoperative examination of the patient is mandatory to evaluate the anesthetic risks, although this is often restricted to a visual appraisal for many species. Fasting should be in accordance with body size and feeding habits. The amount of time necessary to withhold food prior to anesthesia varies by species and range from 3 to 4 hours in the ruffed lemur (*Varecia variegata*), a frugivorous species, and up to 24 hours in eastern lesser bamboo lemurs (*Hapalemur griseus*) and Coquerel's sifaka (*Propithecus verreauxi coquereli*), species that are highly folivorous. Inhalation anesthetics can be used not only to maintain anesthesia, but to induce it as well. Small New World primates can be manually restrained and induced using a facemask. In general, ketamine in combination with (dex) medetomidine is typically used, with dose rates varying between species. The loss of heat during anesthetic procedures is well documented and small animals are at a greater risk than larger animals. Closely monitor body temperature and hydration status during the entire procedure. Analgesia should be considered in all painful procedures, and meloxicam and buprenorphine are recommended, especially following major surgical procedures. Buprenorphine may provide prolonged relief of up to 8 hours in some NHP. Both oxymorphone, and hydromorphone have also been used post operatively with good results. Butorphanol is not utilized in combination with other anesthetics due to commonly seen respiratory depression.

Diseases

Various forms of trauma are common problem observed in NHP. Injuries may include lacerations, bite wounds, leg and tail fractures, and occasional head and vertebral fractures. Treatment generally involves soft tissue surgery or orthopedics. Vertebral and skull fractures generally require euthanasia.

As NHP live longer in zoo collections, geriatric conditions, including osteoarthritis, diabetes, neoplasia (...) are becoming more common. Renal disease is a common cause of mortality in older prosimians. Species commonly affected include *Eulemur*, *Varecia*, *Hapalemur*, and *Loris tardigradis*. Common neoplasms include cancer of the GI, integumentary, reproductive, and hematopoietic systems. Common age-related health issues in geriatric apes include renal disease, reproductive disorders, abdominal abscesses, cardiovascular disease, dental issues, vision degeneration, and osteoarthritis.

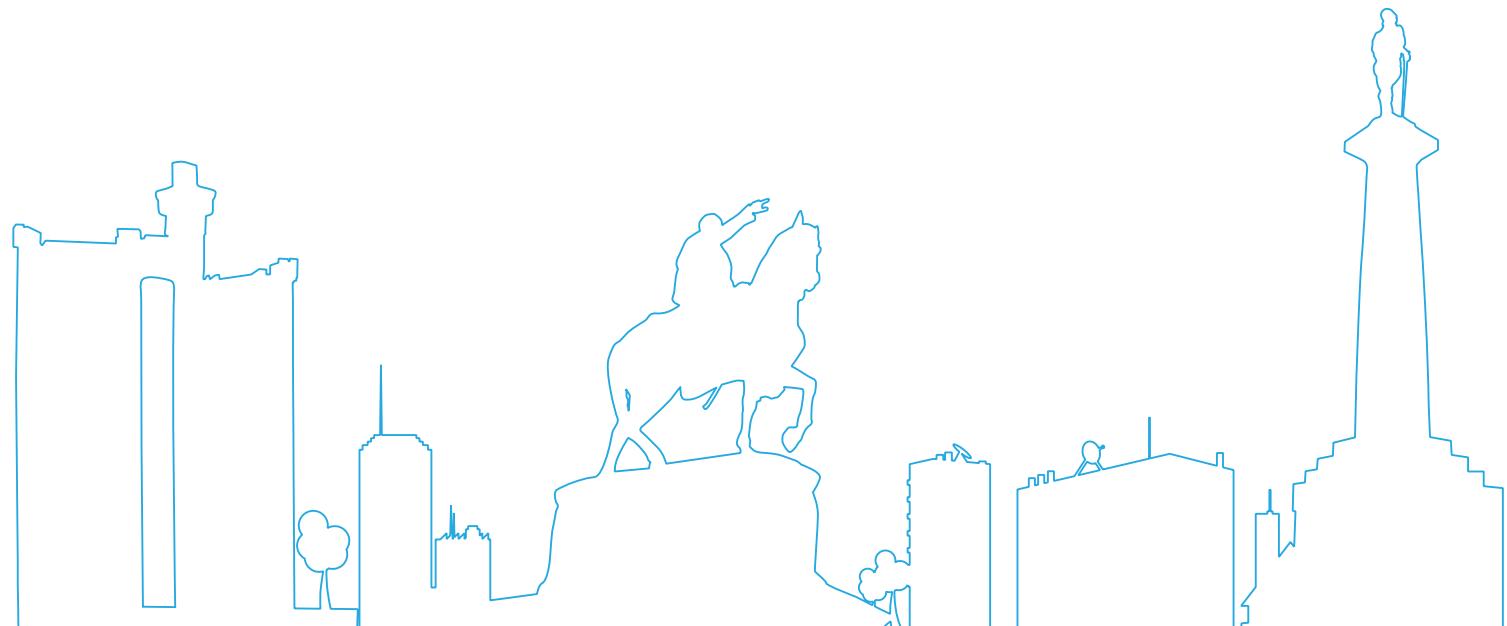
Enteric and respiratory diseases are particularly common in NW and OW monkeys and are caused by a range of pathogens, including parasites, bacteria, and viruses.

Marmoset wasting syndrome, a multi-factorial disease of callitrichids, causes chronic weight loss and diarrhea. Contributing factors are suboptimal temperatures that place metabolic demands on the animal; chronic or recurrent GI disease caused by bacterial, viral, or parasitic enteropathogens; *Trichosporirura leptostoma* infection of the pancreatic ducts resulting in pancreatic dysfunction; inappropriate diets; and gluten intolerance. The outcome of any or a combination of these disease processes is chronic lymphoplasmacytic enteropathy, a final common pathway that results in a malabsorption syndrome with chronic diarrhea and weight loss.

All NHP have a simplex uterus and hemochorionic placentation and may develop reproductive tract problems similar to those of humans (endometriosis, adenomyosis, leiomyomas, placental abruption, placenta previa, ectopic pregnancy, retained placenta, etc.). Callitrichids exhibit chorionic placental fusion of fraternal twins, with blood chimerism. Contraception may involve management actions (single-sex groups or for seasonally breeding species separation of the sexes during the mating season). Frequently used reversible technique is subcutaneous implant of long-acting gonadotropinreleasing hormone (GnRH) agonist such as deslorelin.

Surgical procedures on NHP may occur after trauma (fracture repair, extensive soft tissue injury repair), dental extraction, laparotomy (abscess drainage and removal, hernia repair, reproductive tract surgeries, GI issues, neoplasia), eye, ear, nose, and throat surgeries (cataract removals, otitis interna, sinusitis, etc.)...

INTERNAL MEDICINE



**Adeline Betting (France)**

DVM, Dipl ACVIM-SAIM

EERVC 2024 Lectures

1. Hypercalcemia through clinical cases: diagnosis and treatment -
 2. Anemia and chronic kidney disease: physiopathology, old and new treatments
 3. Urolithiasis in dogs and cats
 4. Regenerative anemias
 5. Non-regenerative anemias
- Initial training at the veterinary school of VetAgro Sup, Lyon (France) ; graduated as DVM in 2015
– rotational internship at the veterinary school of Maisons-Alfort (France)
– specialized internship in internal medicine for companion animals at the veterinary school of Toulouse (France)
– residency training at the VetSuisse Faculty, Bern (Switzerland) between 2018 and 2021
– diplomate of the American College of Veterinary Internal Medicine (Small Animals) in 2022
– I'm working as a senior clinician in internal medicine in the Small Animal Hospital Onlyvet, Saint-Priest (France) since September 2021
– Publications:
- * Betting, A., Schweighauser, A., & Francey, T. (2022). Diagnostic value of reticulocyte indices for the assessment of the iron status of cats with chronic kidney disease. *Journal of veterinary internal medicine*, 36(2), 619-628.
- * Lutz, B., Betting, A., Kovacevic, A., Durand, A., Gurtner, C., Kaiponen, TS, Kooistra, H., Campos, M., & Cui, Y. (2022). Dilated cardiomyopathy in a cat with congenital hyposomatotropism. *JFMS open reports*, 8(1), 20551169221086437.
- * Vincenti, S., Betting, A., Durand, A., Campos, M., Scanziani, E., & Martin, SS (2021). Total laryngectomy in a cat with a laryngeal peripheral nerve sheath tumor. *Veterinary surgery: VS*, 50(7), 1533–1541.
- * Lavabre, T., Betting, A., Bourgès-Abella, N., Layssol-Lamour, C., & Trumel, C. (2019). Abnormal Sysmex XT-2000iV DIFF scattergram in a cat with a prominent mastocytemia. *Veterinary clinical pathology*, 48(4), 624–629.

HYPERCALCEMIA THROUGH CLINICAL CASES: DIAGNOSIS AND TREATMENT

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I. When should hypercalcemia be suspected?

In dogs, approximately 50% of cases of hypercalcemia present with **polyuria-polydipsia (PUPD), signs of urinary tract issues related to urolithiasis, weakness, and exercise intolerance**. Urinary incontinence (likely a result of PUPD) and inappetence are somewhat less common, occurring in about 40% of cases. Weight loss and muscle wasting are observed in approximately 15-20% of cases. Less commonly, vomiting (13%), shivering (10%), constipation (6%), and a stiff gait (5%) are reported.

In cats with idiopathic hypercalcemia, the reported clinical signs are significantly different and less frequently observed than in dogs. **Approximately half of the affected cats show no clinical signs**, while 20% experience weight loss without other symptoms, and 15% exhibit signs of urolithiasis. A very small percentage (5%) present with chronic gastrointestinal signs, such as vomiting, constipation, or anorexia.

As noted above, clinical signs may be minimal or absent, which can result in delayed diagnosis. Therefore, measuring calcium levels may be warranted in a wide range of situations, either due to the presence of specific symptoms (such as PUPD) or as a secondary step when common causes have been excluded and no definitive diagnosis has yet been made (such as in cases of isolated weight loss). Additionally, hypercalcemia is associated with several specific conditions, including neoplastic diseases (such as T-cell lymphoma/leukemia, multiple myeloma, and anal sac adenocarcinoma) and calcium oxalate urolithiasis. The presence or suspicion of these diseases should also prompt the measurement of calcium levels.

II. Physiopathological principles recap

Calcium plays a critical role in numerous physiological functions, including enzymatic reactions, regulation of vascular smooth muscle tone, muscle contractility, neuromuscular transmission, cell membrane stability, coagulation, and skeletal support.

a. Calcium stores evaluation

In the body, calcium is primarily stored in the bones, accounting for 99% of total body calcium; however, this calcium is mostly unavailable for regulatory purposes. Calcium is also found in both extracellular and intracellular spaces. Intracellular calcium exists mainly in its ionized form, while extracellular calcium is divided into three fractions:

- Ionized calcium (55%): This is the free fraction of calcium and **the only active form** capable of participating in physiological processes. Because of its critical role, ionized calcium is **tightly regulated within a narrow range** to prevent dysfunction of major organs.
- Protein-bound calcium (35%): This fraction serves as a storage pool and is inactive in physiological reactions.
- Complexed calcium (10%): This fraction is bound to molecules such as phosphate, bicarbonate, sulfate, citrate, and lactate, and is largely inactive.

Measurement of total calcium reflects the sum of ionized, protein-bound, and complexed calcium. However, **total calcium is not as representative or accurate as ionized calcium for evaluating significant changes in calcium metabolism**. For instance, in cases of hypoalbuminemia, total calcium levels may fall below the reference range due to a decrease in the protein-bound fraction, but ionized calcium (iCa) typically remains within the normal range. Conversely, if protein concentrations are elevated, total calcium may increase while ionized calcium remains normal; this situation is not considered abnormal, and further investigation is usually unnecessary.

Equations that estimate ionized calcium based on total calcium concentrations have proven to be highly inaccurate and cannot reliably assess ionized calcium levels.

For these reasons, direct measurement of ionized calcium is recommended to confirm hypercalcemia. Additionally, because calcium and phosphate regulation are closely interrelated, simultaneous measurement of phosphate is recommended to aid in the diagnostic process, as explained later.

Finally, it is important to note that neither total nor ionized calcium should be measured from EDTA-treated blood samples, as EDTA is a calcium chelator and will cause artificially low calcium levels. Ionized and total calcium are typically measured from heparinized plasma or serum samples.

b. Calcium homeostasis

The regulation of serum calcium concentration is a complex process that requires the coordinated actions of parathyroid hormone (PTH), vitamin D metabolites, and calcitonin. **The primary regulators of calcium homeostasis are PTH and vitamin D**, which also influence each other. The key target organs for these calcium-regulating hormones are the kidneys, intestines, and bones. The major interactions within the calcium regulatory system are summarized in Figure 1 and elaborated upon in the following paragraphs.

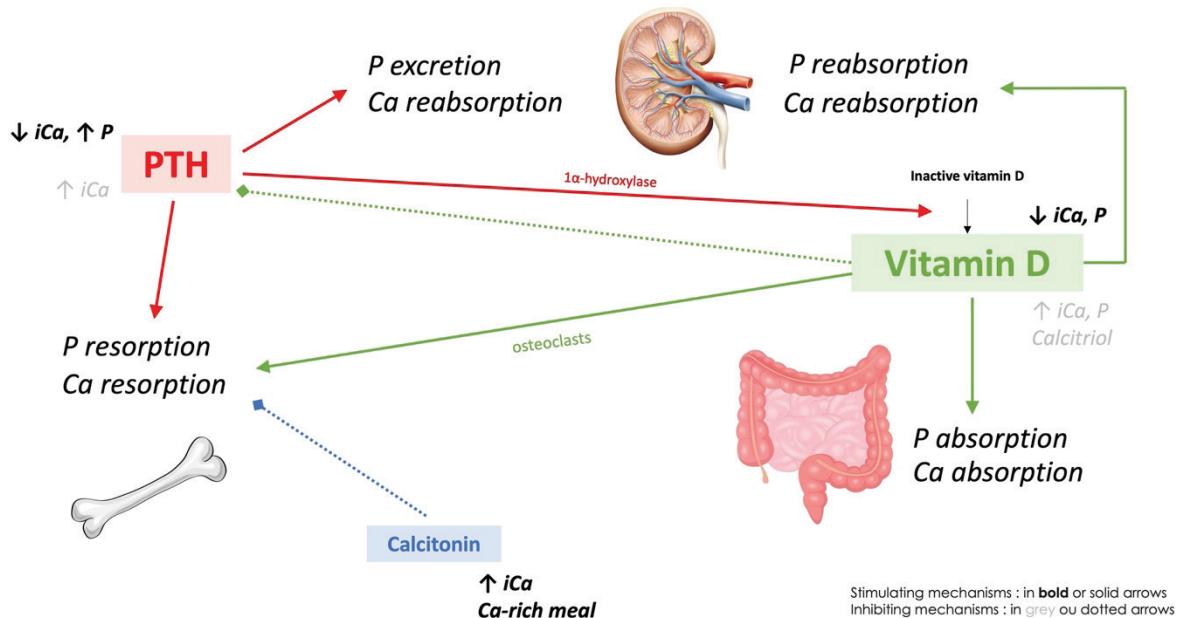


Figure 1: Calcium homeostasis

c. Actions of PTH and PTHrP

Parathyroid hormone (PTH) is synthesized by the chief cells of the parathyroid glands and is the principal hormone responsible for the minute-to-minute regulation of blood calcium concentration. **The overall effect of PTH is to increase ionized calcium levels while decreasing phosphate levels** (Figure 2).

PTH synthesis is stimulated by low iCa and high phosphate levels and is inhibited by elevated iCa and active vitamin D. PTH exerts its effects on three primary targets:

- Indirect action on the intestines: PTH increases vitamin D production by activating the enzyme 1α -hydroxylase in the renal parenchyma, which accelerates the conversion of 25-hydroxycholecalciferol to 1,25-dihydroxycholecalciferol, the active form of vitamin D (also known as calcitriol).
- Direct action on the kidneys: PTH promotes increased tubular reabsorption of calcium and enhanced tubular excretion of phosphate.
- Direct action on the bones: PTH increases bone resorption of calcium and phosphate by stimulating the proliferation of osteoclasts.

PTH-related protein (PTHrP) is a small peptide with a structure very similar to PTH, playing a central role in humoral hypercalcemia of malignancy. It is produced by certain tumor cells, particularly round cell tumors, independently of calcium concentration. PTHrP binds to and stimulates PTH receptors in bone and kidney cells with the same affinity as PTH, leading to increased iCa levels and decreased phosphate levels.

d. Actions of vitamin D

Dogs and cats have limited ability to synthesize vitamin D through their skin, making them reliant on dietary sources for sufficient vitamin D. A series of hydroxylation reactions leads to the production of calcidiol (25-hydroxyvitamin D), followed by calcitriol (1,25-dihydroxyvitamin D), the active form of vitamin D.

Vitamin D synthesis is stimulated by the activation of the enzyme 1 α -hydroxylase, which is regulated by PTH and low levels of iCa or phosphate. Calcium fluctuations can override signals from serum phosphate and PTH concentrations. Negative feedback mechanisms reduce calcitriol synthesis when its levels increase or when iCa or phosphate levels rise.

An increase in calcitriol **increases both iCa and phosphate levels** (Figure 2) by acting on several target organs:

- Intestines: Calcitriol stimulates the absorption of calcium and phosphate from the intestines.
- Bones: Vitamin D is essential for normal bone resorption by promoting the differentiation of precursor cells into osteoclasts. It also contributes to normal bone development by facilitating the production of various bone proteins by osteoblasts.
- Kidneys: Vitamin D promotes the reabsorption of calcium and phosphate from the glomerular filtrate. It also exerts a protective effect on podocytes by reducing injury and loss. Additionally, vitamin D inhibits 1 α -hydroxylase to prevent the overproduction of calcitriol.
- Parathyroid gland: Calcitriol directly inhibits PTH synthesis by binding to its receptor on parathyroid chief cells. It also indirectly inhibits PTH synthesis by increasing intestinal calcium absorption.

e. Actions of calcitonin

Calcitonin is synthesized by the C cells of the thyroid gland and plays a relatively minor role in calcium and phosphate regulation compared to PTH and vitamin D. One of its key functions is to **limit the extent of postprandial hypercalcemia** (Figure 2), and it is therefore stimulated by calcium-rich meals. The primary target organ for calcitonin is bone, where it inhibits osteoclastic bone resorption.

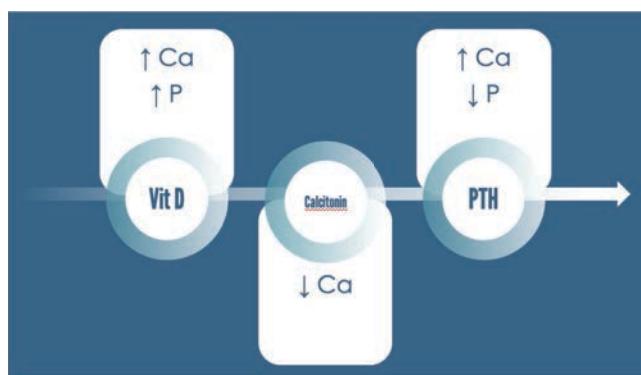


Figure 2: Summary of the main effects of the calcium regulatory hormones

III. Differential diagnosis and diagnostic approach of hypercalcemia

A commonly used mnemonic for the differential diagnosis of hypercalcemia is "HOGS IN YARD":

primary **H**yperparathyroidism
Osteolysis
Granulomatous disease
Spurious (= artifact)
Idiopathic
Neoplastic
Young
Addison
Renal
vitamin **D** toxicity

By utilizing patient history, concurrent phosphate measurements, and a comprehensive biochemistry panel, it is usually possible to narrow the differential diagnosis to three or four primary conditions:

$\uparrow\uparrow \text{Ca} \downarrow = \text{P}$	primary H yperparathyroidism
$\uparrow \text{Ca} = \uparrow \text{P}$	O steolysis
$\uparrow\uparrow \text{Ca} \uparrow\uparrow \text{P}$	G ranulomatous disease
	S purious (= artifact)
$\uparrow\uparrow \text{Ca} = \uparrow \text{P}$	I diopathic 
$\uparrow\uparrow \text{Ca} \downarrow = \text{P}$	N eoplastic
$\uparrow \text{Ca} \uparrow \text{P}$	Y oung
$\uparrow \text{Ca} = \uparrow \text{P}$	A ddison
$\uparrow\uparrow \text{Ca} \uparrow\uparrow \text{P}$	R enal
$\uparrow \text{Ca} = \uparrow \text{P}$	vitamin D toxicity

↑ Increased
 ↑ Mildly increased
 = Normal
 ↓ Mildly decreased

At this point, specific tests should be conducted to confirm or rule out these possibilities. Feline idiopathic hypercalcemia is a diagnosis of exclusion and may require an extended diagnostic workup. Table 1 summarizes the relevant tests for each differential diagnosis.

Suspected disease	Tests needed for diagnosis	Comments
Primary Hyperparathyroidism	<ul style="list-style-type: none"> • PTH measurement • Ultrasound of the parathyroid glands 	<ul style="list-style-type: none"> • Dogs >>> Cats • Older dogs (> 7 yo mostly) • Inherited in Keeshond dogs • Other breeds: mixed breeds, Labrador Retrievers, German Shepherd dogs, Golden Retrievers, Poodles, Shih Tzu, Springer Spaniels
Osteolysis	<ul style="list-style-type: none"> • Imaging • ± Histopathology or culture if indicated 	<ul style="list-style-type: none"> • Osteomyelitis, Bone metastases > Primary tumor • Usually, mild hypercalcemia
Granulomatous disease	<ul style="list-style-type: none"> • Infectious disease search (depending on which is suspected): culture, PCR, serology, imaging, etc 	<ul style="list-style-type: none"> • Fungal • Other causes (rarely reported): Angiostrongylus, Mycobacterium, sterile pyogranulomatous steatitis
Spurious	<ul style="list-style-type: none"> • Repeat iCa measurement if a doubt exists on the analysis 	
Idiopathic	<ul style="list-style-type: none"> • Exclusion of other possible diseases 	<ul style="list-style-type: none"> • Cats only
Neoplastic	<ul style="list-style-type: none"> • Imaging • PTHrP • ± Cytology (spleen, liver, bone marrow) • ± Histopathology • ± Specific tests 	<ul style="list-style-type: none"> • Most frequent: T-cell lymphoma/leukemia, multiple myeloma, anal sac adenocarcinoma, carcinomas
Young	<ul style="list-style-type: none"> • Signalment 	<ul style="list-style-type: none"> • Usually mild elevation of total and/or ionized calcium
Addison	<ul style="list-style-type: none"> • ACTH stim test 	<ul style="list-style-type: none"> • Usually mild elevation of total and/or ionized calcium (up to 33% of the dogs)
Renal disease	<ul style="list-style-type: none"> • Urinalysis • Biochemistry panel • Imaging 	<ul style="list-style-type: none"> • 10-30% of CKD patients • Up to 90% of AKI by grape/raisins toxicity
Vitamin D toxicity	<ul style="list-style-type: none"> • Anamnesis • Vitamin D metabolites measurement • Exclusion of granulomatous diseases 	

Table 1: Further tests recommended depending on the hypothesis for the origin of hypercalcemia

IV. Treatment*a. Emergency treatment, at the hospital*

In which cases is hospital treatment necessary?

- Weakness or significant reduced general condition
- Severe hypercalcemia
- Significant arrhythmia

Treatment of the underlying disease should always be initiated when feasible. When treatment of the underlying cause is not available or while the cause is being investigated, nonspecific treatments can help reduce hypercalcemia and alleviate associated clinical signs. These treatments primarily aim to increase calcium excretion through the kidneys and/or decrease bone resorption:

1. Fluid therapy: Typically involves administering 0.9% NaCl. However, if the animal is initially dehydrated, an alternative rehydration fluid may be required.
2. Furosemide: Administered if the animal is properly hydrated.
3. Glucocorticoid therapy: **Should be avoided if a definitive diagnosis has not been established**, particularly if a round-cell tumor has not been ruled out.
4. Additional Treatments: In certain situations, other agents such as sodium bicarbonate, calcitonin, or injectable bisphosphonates (e.g., pamidronate, zoledronate) may be used.

If intoxication is suspected, such as from ingestion of vitamin D-containing substances, interventions like vomiting induction, gastric lavage, and administration of intralipids should be promptly considered to reduce intestinal absorption.

b. Long-term treatment

In which cases can treatment be started at home?

- Absence of weakness or altered general condition
- Diagnostic approach has already been performed, or owners do not wish to pursue a specific diagnosis

Treatment of the underlying disease should always be initiated once a diagnosis is established. Examples include:

- Neoplastic disease: Treatment options may include surgery, chemotherapy, radiation therapy, etc.
- Primary hyperparathyroidism: Surgical removal of enlarged parathyroid gland(s), ethanol or heat ablation.
- Addison's disease: Glucocorticoid therapy with or without mineralocorticoid supplementation.

Nonspecific treatments such as glucocorticoids and/or furosemide can be employed as primary treatments if a diagnosis is not yet reached or if no specific treatment is available for the underlying disease. They can also support therapy for the underlying condition. Their effectiveness may vary based on the underlying cause. For example, glucocorticoids are reported to be particularly effective for hypercalcemia induced by leukemia/lymphoma, multiple myeloma, thymoma, hypoadrenocorticism, hypervitaminosis D, or feline idiopathic hypercalcemia.

In the past 20 years, there have been numerous reports on the use of bisphosphonates (intravenous: pamidronate, zoledronate; oral: alendronate) for treating various causes of hypercalcemia. Their use has been described for malignancy-related hypercalcemia, management of bony metastases, feline idiopathic hypercalcemia, primary hyperparathyroidism, and granulomatous disease. Bisphosphonates inhibit osteoclast activity, thereby reducing bone resorption. They appear to be very safe for long-term use, with only a few cases of osteonecrosis of the jaw or acute kidney injury reported.

In cases of feline idiopathic hypercalcemia, dietary modifications alone may resolve hypercalcemia in some instances. Various dietary strategies have been employed, including renal or urinary diets, low-calcium diets, and high-fiber diets; the addition of chia seeds has also been anecdotally reported as effective. One study indicated that transitioning to diets containing less than 200 mg of calcium per 100 kcal and with a Ca:P ratio of less than 1.4:1 resulted in a 90% response rate, with 60% of cats achieving normal iCa levels upon recheck and 30% showing significant improvement. These dietary strategies should be considered first, particularly if clinical signs are mild and hypercalcemia is mild to moderate. Multiple trials may be necessary.

If no improvement is observed, if hypercalcemia is severe, or if a rapid response is required, additional therapies may be considered:

- Oral bisphosphonates (e.g., alendronate) or injectable bisphosphonates (e.g., pamidronate, zoledronate)
- Glucocorticoids

V. Some examples

The following paragraphs provide summaries of classical and less common cases of hypercalcemia. The "Physical examination" encompasses the evaluation of muscle condition score (MCS) and body condition score (BCS), a cardio-respiratory assessment, abdominal palpation, rectal examination (except in cats), and evaluation of peripheral lymph nodes. If necessary, a neurological and/or orthopedic examination may also be conducted. The "Initial work-up" includes basic blood and urine analyses: complete blood count (CBC), biochemistry panel (including phosphate), electrolytes panel (including ionized calcium), and urinalysis. Any unmentioned findings from the physical examination or initial work-up were normal. Reference intervals are provided in brackets.

a. Falco, Boxer, male castrated, 7 years old

- **Major clinical signs:** Severe PUPD (120 mL/kg/day), decreased appetite, weight loss, intermittent generalized tremors.
- **Physical examination:** BCS 3/9
- **Initial work-up:** iCa 1.76 mmol/L (1.25-1.5), P 35 mg/L (25-68), creatinine 20.9 mg/L (< 18)
- **Hypotheses:** Based on the signalment, anamnesis, and low-normal phosphate levels, some causes were considered unlikely or excluded. Consequently, the remaining hypotheses were primary hyperparathyroidism and paraneoplastic hypercalcemia.
- **Further work-up:**
 - PTH was below reference interval, excluding primary hyperparathyroidism.
 - PTHrP was below reference interval; however, this cannot exclude paraneoplastic hypercalcemia as other factors might play a role in this situation
 - Abdominal ultrasound (US) and chest radiographs (XR): unremarkable
 - Fine needle aspiration (FNA) of liver and spleen: unremarkable
 - Bone marrow aspiration: stage 5 lymphoma or aleukemic acute lymphoid leukemia
- **Diagnosis:** Due to the duration of the disease, stage 5 lymphoma was considered more likely than aleukemic acute lymphoid leukemia.
- **Treatment:** Fluid therapy (NaCl 0.9%) while investigations were conducted. Maximum tolerated dose chemotherapy.
- **Follow-up:** Hypercalcemia and azotemia resolved quickly, with no recurrence of hypercalcemia noted during a 7-month follow-up period.

b. Mimi, European Longhair cat, female neutered, 10 years old

- **Major clinical signs:** Moderate weight loss over the last months. Intermittent vomiting for 2 months.
- **Physical examination:** BCS 3/9
- **Initial work-up:** iCa 1.60 mmol/L (1.15-1.35), P 40 mg/L (25-68)
- **Hypotheses:** Based on the signalment, anamnesis, and normal phosphate levels, some causes were considered unlikely or excluded. The remaining hypotheses were feline idiopathic hypercalcemia, paraneoplastic hypercalcemia, and primary hyperparathyroidism.
- **Further work-up:**
 - PTH was below reference interval, excluding primary hyperparathyroidism.
 - Abdominal US and chest XR: unremarkable
 - FNA of liver and spleen: unremarkable
- **Presumptive diagnosis:** Feline idiopathic hypercalcemia was deemed highly probable. Further examinations, such as PTHrP testing and bone marrow aspiration, could not be performed due to financial constraints.
- **Treatment:** Multiple diet changes (urinary, renal, high-fiber diet) were not sufficient. Alendronate (10 mg once weekly) led to resolution of hypercalcemia.
- **Follow-up:** No recurrence of hypercalcemia over the 3 months follow-up.

c. Jess, Irish Terrier, male castrated, 9 years old

- **Major clinical signs:** Intermittent vomiting for 2 months. Recurrent urolithiasis (calcium oxalate). Moderate PUPD.
- **Physical examination:** BCS 7/9
- **Initial work-up:** iCa 1.9 mmol/L (1.25-1.5), P 22 mg/L (25-68)
- **Hypotheses:** Based on the signalment, anamnesis, and low phosphate levels, some causes were deemed unlikely or excluded. The remaining hypotheses were paraneoplastic hypercalcemia and primary hyperparathyroidism.
- **Further work-up:**
 - Abdominal US and chest XR: unremarkable
 - PTH was severely elevated at 110 pg/mL (12-88) despite marked hypercalcemia
 - US of the parathyroid glands: solitary parathyroid mass (diameter 8 mm)
- **Diagnosis:** Primary hyperparathyroidism.
- **Treatment:** Medical treatment for stabilization before anesthesia: fluid therapy (NaCl 0.9%) and furosemide, followed by surgical resection of the parathyroid mass (adenoma)
- **Follow-up:** Quick resolution of hypercalcemia (6h after surgery). Mild hypercalcemia post-op (1.08 mmol/L); no need for calcium or vitamin D supplementation. No recurrence of hypercalcemia over the 7 months follow-up.

d. Cookie, Continental Toy Spaniel, female, 2 years old: an unusual cause for hypercalcemia

- **Major clinical signs:** Severe PUPD, coughing and exercise intolerance for 1 month.
- **Physical examination:** Tachypnea, increased breath sounds.
- **Initial work-up:** iCa 1.65 mmol/L (1.25-1.5), P 70 mg/L (25-68). Mild hyperglobulinemia 50 g/L (< 45).
- **Hypotheses:** Based on the signalment, anamnesis, and mildly elevated phosphate levels, some causes were deemed unlikely or excluded. The remaining hypotheses included granulomatous disease, osteolysis, vitamin D toxicity, and hypoadrenocorticism. Although renal disease was not excluded, it appeared less likely due to a creatinine concentration of 5 mg/L, which is within the lower end of the reference interval (< 18 mg/L).
- **Further work-up:**
 - Basal cortisol: 242 nmol/L; hypoadrenocorticism was excluded
 - Abdominal US: unremarkable
 - Chest XR: patchy alveolar opacity, primarily located at the periphery of the lung lobes.
 - Quick Angiostrongylus test: positive
 - Baermann fecal examination: positive for Angiostrongylus vasorum
- **Diagnosis:** Angiostrongylosis with secondary hypercalcemia (granulomatous disease)
- **Treatment:** Fenbendazole and low dose of prednisolone, as recommended by literature.
- **Follow-up:** Quick resolution of hypercalcemia.

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ANEMIA OF CHRONIC KIDNEY DISEASE: PHYSIOPATHOLOGY, OLD AND NEW TREATMENTS

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Chronic kidney disease (CKD) is a prevalent condition affecting middle-aged to older dogs and cats. A significant complication associated with CKD is the development of anemia. The pathogenesis of anemia in these patients is multifactorial, typically resulting in a normocytic, normochromic, non-regenerative anemia of varying severity, ranging from mild to severe. This condition can manifest as lethargy, weakness, and a reduced quality of life, while also exacerbating the progression of CKD. To mitigate these adverse effects, therapeutic intervention is advised when the anemia reaches moderate to severe levels.

Physiopathology of anemia in CKD

The pathogenesis of anemia in CKD is multifactorial. While erythropoietin deficiency is the primary contributing factor, the development of anemia is also influenced by chronic inflammation, bone marrow suppression and/or fibrosis, as well as persistent blood loss.

1. Erythropoietin deficiency

Erythropoietin (EPO) facilitates the maturation of erythroid precursors into mature erythrocytes within the bone marrow. The production of EPO by renal peritubular interstitial cells is primarily regulated by hypoxia-inducible factors (HIF). Although EPO is also synthesized in other tissues, such as the liver, brain, and muscles, the kidneys are the principal site of production.

HIF is a heterodimer composed of an α-subunit and a β-subunit. Under normoxic conditions, HIF-α is continuously produced but is simultaneously deactivated by prolyl hydroxylase domain (PHD) enzymes. In hypoxic conditions, PHD enzymes are unable to operate at their normal rate, allowing HIF-α to accumulate. The accumulated HIF-α binds to HIF-β, forming the HIF transcription factor, which not only promotes EPO synthesis but also upregulates the synthesis of proteins involved in iron uptake and transport.

In patients with CKD, the decline in glomerular filtration rate reduces renal metabolic activity and creates localized relative hyperoxia. This increased oxygen level triggers the degradation of HIF-α, thereby impeding EPO synthesis. Nevertheless, erythropoietin receptors can still be activated through the administration of erythropoiesis-stimulating agents (ESAs).

2. Hepcidin and iron metabolism dysregulation

Most of the total body iron is contained within heme molecules, such as hemoglobin and myoglobin, or stored intracellularly in hepatocytes and macrophages, bound to ferritin. The transport protein ferroportin facilitates the export of iron from erythrocytes, hepatocytes, or macrophages into the serum compartment. Only a small fraction of iron circulates in the bloodstream, where it is bound to transferrin. Intestinal iron absorption occurs via the DMT-1 (divalent metal transporter 1) protein, but most iron is recycled from phagocytized erythrocytes, allowing it to re-enter circulation or be stored in cells.

Physiological iron loss is minimal and occurs primarily through the shedding of epithelial cells (enterocytes, uroepithelial cells, skin cells) and blood loss, as there is no active excretory pathway for iron.

Regulation of iron stores is primarily mediated by the protein hepcidin, which functions as a negative regulator of iron homeostasis. Hepcidin synthesis is upregulated in response to chronic inflammation (as a positive acute phase protein) and elevated iron stores. This leads to the following effects:

- Degradation of the DMT-1 transport protein, resulting in reduced intestinal iron absorption.
- Degradation and downregulation of ferroportin, leading to the sequestration of iron within hepatocytes and macrophages. Consequently, the availability of iron for transport to the bone marrow is diminished.

CKD is characterized by a persistent inflammatory state, with elevated synthesis of pro-inflammatory cytokines such as interleukin (IL)-1 and IL-6. This inflammation upregulates hepcidin expression, thereby reducing the availability of iron for erythropoiesis. Additionally, impaired renal clearance in CKD contributes to increased hepcidin levels. This condition, known as functional iron deficiency, occurs when total iron stores are normal, but the iron is sequestered and unavailable for physiological use.

The inflammatory state in CKD is also associated with a reduction in transferrin concentration, as transferrin is a negative acute phase protein. This reduction is reflected in a decrease in total iron-binding capacity (TIBC), a parameter that is significantly lowered in cats with CKD.

Aluminum toxicity may further disrupt iron metabolism, potentially leading to microcytic anemia that does not respond to iron supplementation. This can occur with the use of aluminum-based phosphate binders, which are often employed to manage hyperphosphatemia in CKD patients.

Moreover, malnutrition may exacerbate iron deficiency by reducing dietary iron intake.

3. Shortened red blood cells (RBC) lifespan

Uremia has been associated to a shortened lifespan of RBC, with the underlying pathogenesis likely being multifactorial. The chronic inflammatory state in uremia contributes to macrophage activation and enhanced erythrophagocytosis. Additionally, circulating uremic toxins are believed to induce low-grade chronic hemolysis and lipid peroxidation of RBC membranes, ultimately leading to premature clearance of RBCs by the reticuloendothelial system.

4. Bone marrow suppression

Several uremic inhibitors of erythropoiesis have been identified, affecting different stages of RBC production within the bone marrow. As CKD progresses, secondary renal hyperparathyroidism often develops, resulting in elevated parathormone levels. Parathormone exerts a direct toxic effect on erythroid precursors and may also promote bone marrow fibrosis, leading to myelophthisis and reduced erythropoiesis.

Inflammatory cytokines, such as IL-1 and tumor necrosis factor- α (TNF- α), further inhibit erythropoiesis by promoting apoptosis in erythroid precursors, downregulating EPO receptors, reducing the number of erythroid colony-forming units, and diminishing the availability of other pro-hematopoietic factors.

Deficiencies in vitamins B can also impair erythropoiesis, as these vitamins are essential for erythrocyte differentiation.

Additionally, EPO release and recruitment of pluripotent bone marrow stem cells may be stimulated by angiotensin II. Consequently, the use of angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) could result in reduced erythropoiesis.

5. Chronic blood loss

Chronic blood loss contributes to absolute iron deficiency, which can exacerbate anemia in CKD. In some cases, chronic gastrointestinal bleeding may partly contribute to CKD-related anemia, though gastric or

intestinal ulcerations appear to be less common in dogs and cats with CKD compared to humans. Chronic urinary blood loss may also occur, particularly in the presence of uroliths.

Additionally, uremic thrombocytopathy, which impairs platelet function, may promote chronic microscopic blood loss.

Repeated blood sampling, especially in hospitalized animals, further worsens CKD-associated anemia.

Why treat anemia of CKD?

Anemia occurs in 30 to 65% of cats with CKD, with its severity and frequency increasing in proportion to the stage of CKD. Severe anemia is more prevalent in advanced stages of the disease. It has been identified as a potential risk factor for mortality in affected animals, with a median survival time (MST) of 84 to 189 days for cats with a PCV < 29%, compared to a MST of 586 to 794 days for a PCV ≥ 34%.

One study has demonstrated that a relatively small reduction in PCV (within the lower end of the reference range) is a factor associated with an increased risk of progression; this does not mean that anemia is responsible for progression of CKD, but other physiopathological factors support this hypothesis. For example, anemia compromises oxygen delivery to tissues, which may contribute to tubular atrophy and interstitial inflammation and fibrosis, further accelerating the progression of CKD.

Another study showed that cats that responded to ESAs treatment (sustained Ht > 25% over multiple weeks) had a significantly longer MST (238 days) compared to non-responders (83 days).

Additionally, chronic anemia negatively impacts the quality of life in both animals and humans, often resulting in poor nutrition, which can exacerbate the progression of CKD.

"Classical" treatments

1. Erythropoiesis-stimulating agents (ESAs)

The most commonly used ESAs are recombinant human epoetin alfa and darbepoetin alfa.

Darbepoetin alfa is generally preferred over epoetin alfa due to a lower incidence of pure red cell aplasia resulting from the production of anti-EPO antibodies (in less than 10% of cats treated with darbepoetin alfa, compared to 25-40% of cats treated with epoetin alfa). In these cases, the cat becomes transfusion-dependent to maintain a normal RBC mass

Other side effects of ESAs treatment include systemic hypertension, observed in up to 50% of cats, or seizures reported in approximately 15% of cats.

Darbepoetin alfa has a longer half-life, allowing for once-weekly injections, whereas epoetin alfa requires administration three times per week.

ESAs therapy is recommended for animals with chronic moderate to severe anemia (hematocrit < 20% in cats) or for those with persistent, symptomatic CKD-associated anemia.

The initial treatment protocol involves weekly subcutaneous injections of darbepoetin alfa (1 µg/kg) until the target hematocrit level is achieved (≥ 25% in cats). Maintenance therapy focuses on stabilizing the hematocrit within the normal reference range or just slightly below it, with injections administered every 2-3 weeks. The goal is to find the lowest effective dose.

ESA treatment should always be accompanied by iron supplementation (see below).

Reported response rate to darbepoetin alfa varies between 55 and 85%.

2. Iron supplementation

Iron supplementation alone is insufficient to effectively treat anemia associated with CKD due to the sequestration of iron in hepatocytes and macrophages, a consequence of the chronic inflammatory state, as well as potentially inadequate EPO levels to stimulate sufficient erythropoiesis. Therefore, iron supplementation should always be administered in conjunction with ESA therapy.

It is recommended to administer iron dextran once monthly at a dosage of 50 mg/cat IM or 10 mg/kg IM for dogs, for the duration of ESA treatment.

Oral iron supplements are generally not recommended due to potential for causing chronic gastrointestinal issues and/or blood loss, and suboptimal absorption.

3. Blood transfusion

Blood transfusions offer limited benefit in the treatment of chronic anemia associated with CKD. They may be necessary in severe cases where acute blood loss occurs in an already anemic animal, particularly when the regenerative response is expected to be slow and inefficient. In such situations, blood transfusions can provide immediate relief and stabilize the animal, but they do not address the underlying chronic anemia or CKD.

Insights into “future” treatments

In some patients, ESAs may fail to elicit a sufficient response or may be unavailable on the market. Additionally, earlier intervention for CKD-related anemia could be advantageous, as studies on anemic cats have linked early treatment to improved survival and slower CKD progression. However, prolonged ESA therapy in these early stages may heighten the risk of severe adverse effects, such as pure red cell aplasia. Alternative therapeutic options are under investigation and may offer benefit.

One of the most promising agents is molidustat, a member of the hypoxia-inducible factor prolyl hydroxylase inhibitor (HIF-PHI) class. Molidustat prevents the degradation of the HIF- α subunit, thereby enhancing EPO production. Two studies conducted in cats have demonstrated that molidustat significantly increases packed cell volume (PCV) and hematocrit in both healthy and CKD-affected cats, with a sustained response. The most commonly reported side effect is vomiting, observed in up to 40% of treated cats.

Another advantage of HIF-PHI agents is their ability to simultaneously promote iron absorption in the intestine and uptake by erythropoietic cells, thereby partly overcoming the inhibitory effects of hepcidin.

In a study conducted by Charles et al. (2024) in cats with CKD and anemia, molidustat (5 mg/kg orally once daily; 16 cats) was compared to placebo (6 cats) over a 4- to 12-week period. Treatment success was defined as a greater than 25% increase in hematocrit and/or a 4-point increase in hematocrit. By day 21, the treated group exhibited significantly higher PCV and hematocrit levels compared to both baseline and the placebo group. A 50% success rate was observed at 28 days. In the continuation phase (up to 12 weeks, 8 cats), a sustained response was reported, with a success rate of 75%.

Contrary to findings in human medicine, where HIF-PHIs may reduce blood pressure, cats treated with molidustat exhibited stable blood pressure throughout the treatment period.

Conclusion

Anemia associated with chronic kidney disease is the subject of extensive research, with emerging therapeutic strategies designed to enhance the quality of life in affected animals, slow disease progression, and minimize the risk of treatment-related adverse events.

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UROLITHIASIS

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Part I: General considerations

a. Definitions

- **Crystals** are **microscopic** compounds. Their presence suggests that the urine is in a supersaturated state (due to factors like pH, specific gravity, or chemical concentration), which can lead to the formation of crystals and stones. However, the presence of crystals does not necessarily indicate disease. Many animals exhibit crystalluria without any clinical symptoms, and the crystals found in urine do not always correspond to the type of stones present; also, stones can be present with no crystals visible on urine sediment analysis. It's important to note that struvite crystals can form in urine if it is refrigerated or left untested for too long (more than 30 minutes to 2 hours), which is why urine samples should be analyzed promptly after collection.
- **Uroliths** (or stone) refer to the **macroscopic** formations that result from crystal nucleation and growth. Nucleation is when crystals begin to compound together to initiate stone formation. Supersaturation of the urine with organic materials contributing to stone formation is a driving force of this mechanism. These stones can cause clinical symptoms and are considered abnormal in all cases.

b. Urolith anatomy and why it can help us preventing urolith formation

Exact composition of a stone can be given by a quantitative method; several quantitative techniques exist, but infrared spectroscopy and optical crystallography are the most used. A urolith is schematized in Figure 2. Stone growth begins with the formation of the nidus layer. Its composition should be the **primary focus of preventative measures** because it represents the initiation point of urolith formation. The nidus can consist of crystallized minerals or foreign materials (e.g., suture material).

The body layer constitutes the main bulk of the urolith and may have a composition that is either **similar to or different from the nidus layer**. If a nidus is not present, the composition of the body layer can guide preventative measures.

The shell (if complete) or surface (if incomplete) is the outermost concentric layer of the urolith and reflects the most recent stone formation activity. If this layer differs from the nidus or body, it indicates changes in the conditions that contribute to stone formation, such as dietary modifications.

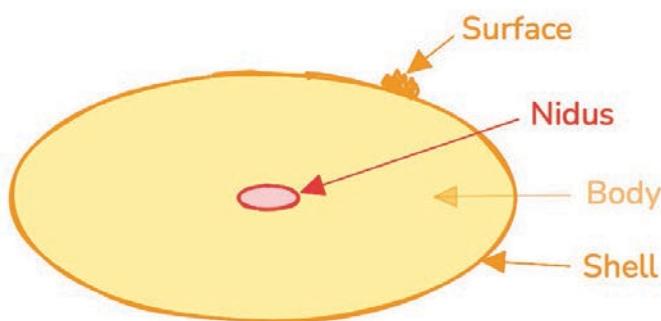


Figure 2: Schematic anatomy of a urolith

c. Why do stones develop in the urinary tract?

Urine is usually in a metastable state, where stone precursors are present in moderate to high concentrations, but the conditions (such as pH, specific gravity, and chemical concentration) do not favor crystal nucleation and growth. In supersaturated urine, spontaneous crystal nucleation occurs, followed by stone growth. Supersaturation is influenced by several factors:

- Urine pH, which affects mineral solubility
- Urine specific gravity (USG)
- Increased concentration of stone precursors, which may result from increased excretion, reduced reabsorption, or decreased urine volume

- The presence of crystallization inhibitors or enhancers

Other factors that can contribute to crystal formation include:

- The presence of foreign material in the urinary tract (such as suture material)
- The ability to expel crystal aggregates = normal voiding capacity

Each type of stone forms under specific conditions, which will be discussed further in the presentation.

d. Epidemiology

Struvite and calcium oxalate stones are the most common types found in dogs and cats, accounting for 70 to 90% of all urolith submissions. However, variations exist based on geography and other epidemiological factors, with age, sex, breed, and the presence of underlying disease or medication significantly influencing stone formation.

i. Age

Young dogs and cats develop slightly different types of stones compared to adults. In a study conducted by the Minnesota Urolith Center on nearly 500 puppies, **struvite** stones were the most common (51%) in those under 6 months old, followed by ammonium urate stones (13%). In older puppies (6 to 12 months old), **urate** stones were most prevalent (35%), with struvite (26%) and cystine (21%) also frequently observed. A similar study in kittens showed that before 6 months of age, **struvite** stones were by far the most common (68%), with calcium oxalate accounting for only a small percentage (11%). Between 6 and 12 months old, **struvite** stones still predominated (56%), but ammonium urate (17%) and calcium oxalate (14%) stones were also well represented.

These findings indicate that in young animals with stones, investigating for urinary tract infections, hepatic vascular anomalies, or genetic abnormalities can be important for effective treatment and prevention of recurrent stones.

ii. Sex

In female dogs, 74% of uroliths are composed of struvite compared to only 13% in male dogs, explained by the female predisposition for UTI.

xii. Breed

Certain breeds are genetically predisposed to abnormalities that reduce tubular reabsorption of some substances, leading to increased excretion and elevated urinary concentrations of crystallogenic precursors. Stones typically form in young adults and may recur throughout life if preventive measures are not implemented. Genetic testing is available for some of these breeds, and ideally, affected animals should not be bred to prevent the transmission of the genetic defect. Table 1 outlines the most common breeds associated with genetic defects that contribute to stone formation.

Type of stones (disease)	Predisposed breeds	Known mutation(s)
Ammonium urate (hyperuricosuria)	Dalmatian English Bulldog	SLC2A9, autosomal recessive
Cystine (cystinuria)	Type I: Newfoundland, Landseer, Labrador	SLC3A1, autosomal recessive
	Type II: Australian Cattle Dog, Miniature Pinscher	SLC3A1 or SLC7A9, autosomal dominant
	Type III: Mastiff and related breeds, Scottish Deerhound, Irish Terrier, French and English Bulldog, Basset Hound	Undetermined, sex-limited (only intact males)
	Cats: Domestic short-haired, Domestic medium-haired, Domestic long-haired, Korat, Maine Coon, Siamese, Sphynx	SLC3A1 or SLC7A9
Xanthine (xanthinuria)	Toy Manchester Terrier, Cavalier King Charles Spaniel, English Cocker Spaniel, Dachshund, mixed breed dogs	

Table 1: Examples of genetic predispositions to stone formation (non-exhaustive list)

iii. Concomitant diseases

Concomitant diseases can significantly contribute to stone formation. Hepatic vascular anomalies often result in the formation of ammonium urate crystals or stones (more commonly seen in dogs than in cats). The discovery of ammonium urate crystals or stones (or unidentified stones in a young animal) should prompt further evaluation of liver function. Additionally, hypercalcemia can increase urinary calcium excretion, leading to calcium oxalate stone formation. Therefore, if calcium oxalate stones are diagnosed, assessing ionized calcium levels is recommended.

v. Medications

Certain medications can contribute to stone formation either through direct metabolic effects or due to the poor solubility of their metabolites in urine. A common example is dogs and cats **treated with allopurinol, not fed with a low-purine diet, which are predisposed to xanthine stone formation**. Allopurinol inhibits xanthine oxidase, the enzyme that converts hypoxanthine to xanthine and then to uric acid (Figures 1a and 1b). As a result, hypoxanthine and xanthine accumulate in the urine, and since xanthine is poorly soluble, stones can form. Using a specific low-purine diet reduces the risk of xanthine stone formation in this context.



Figure 1a: Purine metabolic pathway

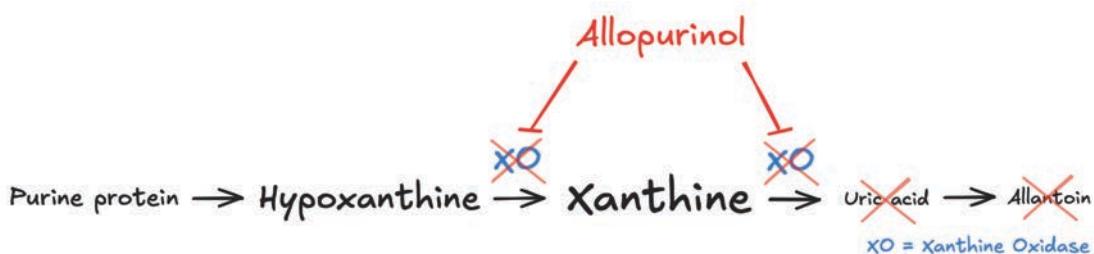


Figure 1b: Allopurinol effects on the metabolism of purine proteins

A newer concern involves cats treated for feline infectious peritonitis with **remdesivir or its metabolite GS-441524**. Since GS-441524 is poorly water-soluble, its use has been linked to stone formation in 2 case reports.

In human medicine, certain drugs (such as sulfonamides, acetazolamide, ciprofloxacin, and zonisamide) have been linked to stone formation due to metabolic changes leading to changes in the urine composition or their role as stone precursors. However, these have not yet been reported in veterinary medicine.

e. Diagnosis

Clinical signs vary depending on the location of the uroliths and whether they are obstructive.

Upper urinary tract uroliths (kidneys, ureters) are usually asymptomatic unless they cause obstruction. In some cases, large non-obstructive renal uroliths may cause pain, discomfort, or chronic hematuria. When ureteral obstruction occurs, signs such as abdominal pain, discomfort, vomiting, or anorexia may be observed. If the obstruction is unilateral and renal function remains largely intact, the animal may show no clinical signs.

Lower urinary tract uroliths (urinary bladder, urethra) typically present with symptoms of cystitis, such as pollakiuria, hematuria, and/or stranguria, often accompanied by a small bladder. Urethral obstruction leads to severe stranguria and an enlarged urinary bladder.

Blood work may reveal findings that range from no abnormalities to severe azotemia, depending on the presence of urethral or ureteral obstruction and the degree of pre-existing renal impairment. Anemia may also be present, either as a consequence of chronic kidney disease (normocytic, normochromic, non-regenerative) or chronic blood loss (microcytic, hypochromic, hyporegenerative to regenerative).

Urinalysis may detect crystals, which could reflect the underlying stone composition, though mismatches between crystal type and stone composition are common. Therefore, the presence of crystals should not be used as the sole diagnostic criterion. Urine pH provides clues about the type of uroliths, with certain stones forming preferentially in acidic conditions (e.g., calcium oxalate, urate, cystine) and others in more alkaline environments (e.g., struvite, calcium phosphate carbonate).

Urine specific gravity is useful for monitoring the effectiveness of preventative measures against stone formation.

Urine sediment may show signs of an active infection (leukocyturia, bacteriuria), particularly in cases involving canine struvite stones. Urine culture is essential in these cases and should always be performed.

Imaging studies, such as radiographs and ultrasound, are valuable for identifying the location of stones and determining whether they are obstructive. In some cases, a combination of radiographs, contrast studies, and ultrasound may be required to assess the entire urinary tract and accurately pinpoint the site of an obstruction.

Radiolucency varies based on the composition of the stone and can offer clues about its nature. Calcium-based stones (oxalate, phosphate carbonate, phosphate apatite), struvite, and silica are radio-opaque. Urate and cystine stones are mostly radiolucent, although larger stones may still be faintly visible on radiographs. Xanthine stones are fully radiolucent.

Finally, stone analysis through quantitative methods is essential for determining its composition and structure. This information is crucial for making tailored recommendations to prevent the formation of new stones in the future.

f. General considerations about treatment

i. Kidney stones

Treatment is required only for problematic nephroliths that cause obstruction, renal parenchyma compression, pain, or recurrent infections. Ideally, minimally invasive procedures should be used, such as dissolution (when appropriate), endoscopic nephrolithotomy (for cats or stones larger than 1-1.5 cm), or extracorporeal shockwave lithotripsy (dogs only). If these techniques are not available or indicated, surgical removal or nephrectomy may be considered.

ii. Ureteral stones

Ureteral obstruction requires urgent intervention, as glomerular filtration rate (GFR) declines progressively and irreversibly within weeks after obstruction begins (with a 35% permanent decrease in GFR after 7 days, 54% after 14 days, and complete loss after 40 days). No imaging-based prognostic factors exist; residual renal function can only be assessed after the obstruction is relieved, followed by several days or weeks of medical management.

Dissolution therapy alone should not be attempted in animals with ureteral obstruction. In dogs, dissolution may be considered if a ureteral stent is placed to relieve the obstruction quickly, especially when struvite stones are strongly suspected (due to a concurrent urinary tract infection or a history of recurrent struvite stones).

Medical management of obstructive ureterolithiasis is generally ineffective, with success rates of only 8-13%. Treatment options described in the literature include fluid therapy, mannitol, alpha-blockers, or tricyclic antidepressants. Antibiotics should be administered when indicated, particularly in cases of pyelonephritis or lower urinary tract infections.

Surgical options for correcting ureteral obstruction include subcutaneous ureteral bypass (SUB) placement (commonly used in cats and small dogs), ureterotomy, or ureteral stenting.

iii. Bladder stones

Urocystoliths that are unlikely to cause urinary tract obstruction due to their size or irregularity do not require removal. Instead, periodic monitoring and preventive measures are recommended. Dissolution therapy can be considered if appropriate.

Urocystoliths that are either likely to cause obstruction or are associated with clinical signs should be removed through minimally invasive procedures or cystotomy.

Minimally invasive options include medical dissolution, voiding urohydropulsion, retrograde urohydropulsion, basket retrieval, endoscopic lithotripsy, intra- or extracorporeal shockwave lithotripsy, and percutaneous cystolithotomy (PCCL).

iv. Urethral stones

When possible, urethroliths should be treated with intracorporeal lithotripsy and basket retrieval. If these options are unavailable or unsuitable due to the animal's size, retrograde urohydropulsion combined with PCCL is recommended.

Urethral surgery for urolithiasis is generally discouraged. However, if retrograde urohydropulsion is not feasible, perineal urethrostomy may be necessary to remove obstructive stones. This procedure also helps prevent recurrent urethral obstruction, particularly in cats with repeated lower urinary tract issues.

Prevention strategies will be evaluated individually for each stone category.

Part II: Common lithiasis – Calcium oxalate and Struvite

a. *Calcium oxalate uroliths*

Calcium oxalate stones account for 30-45% of all uroliths submitted for analysis in dogs and 35-70% in cats. They typically affect middle-aged to older dogs and cats, with a higher prevalence in male dogs. Certain breeds, such as the Miniature Schnauzer, Bichon Frisé, Yorkshire Terrier, and Lhasa Apso, show a strong predisposition, as do Burmese, Himalayan, and Persian cats compared to other feline breeds.

The etiology of calcium oxalate stone formation remains poorly understood. Idiopathic hypercalciuria and, less commonly, idiopathic hyperoxaluria are potential risk factors. A genetic predisposition is strongly suspected as a cause of idiopathic hypercalciuria. Hypercalcemia accounts for less than 10% of cases in dogs with calcium oxalate stones, while in cats, around 30% of those forming calcium oxalate stones have idiopathic hypercalcemia. Urine pH plays a crucial role, with acidic urine ($\text{pH} < 7$) increasing the risk of stone formation by promoting hypercalciuria and reducing concentrations of crystal inhibitors like citrate.

Calcium oxalate stones cannot be dissolved. If removal is indicated (due to urinary tract obstruction, risk of future obstruction, or clinical signs), minimally invasive procedures, SUB system placement, or surgery should be considered.

Prevention strategies for calcium oxalate stones:

- Increase water consumption and provide a high-moisture diet to decrease USG: **≤1.020 for dogs and <1.030 for cats.**
- **Urine pH > 6.5-7**
- Dietary considerations: **avoid excessive dietary protein**, as excessive protein diet increases calciuria and decreases urinary citrate concentration
- **Manage hypercalcemia** if present
- Supplements containing cranberry extract might increase urinary oxalate excretion, and should therefore be avoided.

Those aims can be met by feeding a high-moisture diet (>75%) with alkalinizing properties and moderate protein content. Increasing water intake can be encouraged through methods such as water fountains, adding water to wet food, or flavoring drinking water. High-sodium dry foods (>375 mg/100 kcal) should not be recommended as a substitute for high-moisture diets due to their short-lived effect on water con-

sumption (3-6 months). If diet alone is insufficient to elevate pH, potassium citrate can be used to alkalinize the urine.

For animals with recurrent calcium oxalate stones, thiazide diuretics may be considered as they promote calcium reabsorption in the kidneys and may reduce intestinal calcium absorption.

Routine follow-ups should include checks on urine pH, USG, and regular imaging (ultrasound being more sensitive than radiographs) to detect early recurrence of stones.

b. Struvite uroliths

Struvite stones, also known as magnesium ammonium phosphate (MAP) due to their composition, account for 35-45% of all uroliths analyzed in dogs and 25-50% in cats. They typically affect middle-aged to older animals, with a higher prevalence in females.

A key difference between species is the frequent association of struvite stones with urinary tract infections (UTIs) in dogs (infection-induced struvite), whereas struvite stones occur most often in a sterile environment in cats (sterile struvite). In dogs, these UTIs are commonly caused by urease-producing bacteria such as *Staphylococcus* and *Proteus*. These bacteria promote the production of ammonium in the urine, raising the urine pH and promoting struvite stone formation.

Struvite stones should be medically dissolved with a therapeutic diet designed to acidify the urine ($\text{pH} < 7$), reduce urinary concentrations of phosphate and magnesium, and lower the USG. Dissolution typically occurs within 1-4 weeks. If a UTI is present, targeted antibiotic therapy should be administered, ideally based on culture and sensitivity results. Follow-up urine cultures are recommended after antibiotics are discontinued, as per international guidelines. Radiographs should also be rechecked every 2-4 weeks after starting the therapeutic diet and antibiotics.

If no significant dissolution (less than 50% reduction in stone size on radiographs) is observed after 4-6 weeks of proper treatment, surgical removal should be considered. This may indicate that the stone's composition is not struvite or that it consists of mixed minerals. Additionally, if the stones are too large to be fully immersed in urine, dissolution is unlikely to be effective, and surgical removal is advised.

Preventing struvite stones depends on their cause. It is important to distinguish between sterile and infection-induced stones to provide appropriate prevention strategies.

Prevention strategies for struvite stones:

- Sterile struvite stones (cats >> dogs): Aim for a **urine pH < 6.5** with a **diet low in phosphorus and magnesium**.
- Infection-induced struvite stones (dogs >> cats): Focus on **eliminating UTIs and addressing any underlying risk factors for recurrent infections**. While foods designed for treating struvite urolithiasis do not prevent recurrence in this situation, they may help reduce or slow stone formation when an undiagnosed UTI is present.

Part III: Uncommon lithiasis – Cystine, Urate, Xanthine, GS-441524 and others

a. Cystine uroliths

Cystine stones are relatively uncommon in dogs (accounting for 0.6% to 14% of all submitted uroliths) and exceedingly rare in cats (less than 1% of submitted uroliths). Cystinuria is a genetic disorder caused by mutations in a tubular transport protein responsible for reabsorbing cystine, ornithine, lysine, and arginine. When cystine accumulates in the urine, it can precipitate into crystals and stones, especially in conditions of elevated urine pH. The other amino acids (ornithine, lysine, and arginine) do not typically cause stones or clinical symptoms. However, cats with cystinuria may exhibit neurological symptoms (e.g., hypersalivation, lethargy, behavioral changes, or seizures) possibly related to arginine deficiency.

The prevalence of cystine stones varies depending on time and geographic region. In European studies, cystine stones represent up to 25% of all urolith submissions, while in other regions (e.g., North America, Asia, Oceania), the prevalence is lower (up to 2.7%). Males are significantly more affected than females (98.8% vs. 1.2%), possibly due to androgen-dependent type III cystinuria. Several dog and cat breeds exhibit a genetic predisposition to cystinuria (Table 1); however, specific mutations have not been identified for many breeds.

Diagnosing cystine stones can be difficult, as they are only faintly radiopaque. Ultrasound and contrast studies are often necessary for localization. The urinary cystine-to-creatinine ratio can help confirm cystin-

uria, but only a few laboratories offer this test routinely. In suspected type III cystinuria, this ratio should be checked 3 to 6 months post-castration. If the ratio normalizes, the dog is no longer at risk for stone formation; if not, additional preventive measures will be needed.

Medical dissolution should be considered as a first-line treatment, provided there are no contraindications, before resorting to surgical or minimally invasive procedures. Dissolution involves a reduced level in animal proteins, alkalinizing, high-moisture diet, combined with tiopronin. Potassium citrate may be necessary to further alkalinize the urine, with the goal of maintaining an alkaline pH (≥ 7.5) and a low USG (<1.020 in dogs). The impact of castration on dissolution is unknown.

If medical dissolution is ineffective or if emergency treatment is required (e.g., for urethral obstruction), minimally invasive techniques or surgery should be pursued.

Prevention of cystine stones primarily involves a therapeutic diet that is **high in moisture (>75%), alkalinizing, low in sodium, and reduced in animal protein**. However, there is debate over the degree of protein reduction, as excessive depletion could lead to deficiencies in carnitine and taurine, potentially resulting in dilated cardiomyopathy.

If urine pH remains insufficiently alkaline, **potassium citrate** can be added. **Castration** is always advised for dogs with cystine stones: in type III cystinuria, neutering can prevent further stone formation; for other types, it helps prevent the genetic disorder from being passed on to offspring.

Tiopronin is recommended for long-term management in dogs with recurrent cystine stones because it enhances cystine solubility in the urine. D-penicillamine can be used as an alternative but carries a higher risk of side effects; D-penicillamine is a chelator of cystine.

Prevention strategies for cystine stones:

- Increase water consumption and provide a high-moisture diet to decrease USG: **≤ 1.020 for dogs and <1.030 for cats**.
- Alkalinize the urine: maintain a **pH ≥ 7.5** using an alkalinizing diet \pm potassium citrate.
- **Enhance cystine solubility or chelate cystine**: administer tiopronin or D-penicillamine.
- **Castration**: if the urinary cystine-to-creatinine ratio returns to normal ranges post-neutering (type III cystinuria), it may be the only necessary intervention. If the ratio remains elevated after six months, additional preventive measures are required to avoid recurrence.

b. Urate uroliths

Ammonium urate stones are the 4th most common type of stone in dogs (accounting for 3-5% of all submissions) and the 3rd most common in cats (2% of all submissions).

The formation of urate stones is attributed to two primary mechanisms:

- Inherited alteration of the urate transporter encoded by the SLC2A9 gene, resulting in increased urinary excretion of uric acid (hyperuricosuria)
- Hepatic portosystemic vascular anomalies

Nearly all Dalmatian dogs carry the SLC2A9 mutation, which predisposes them to a natural risk of urate stone formation. Other breeds, such as English Bulldogs and Black Russian Terriers, may also harbor this mutation. In cats, Siamese, Birman, and Egyptian Mau breeds appear to be overrepresented among urate stone formers, although no specific mutation has been identified to date.

A diagnosis of urate stones should prompt genetic testing for the SLC2A9 mutation in susceptible breeds and/or an evaluation for hepatic vascular anomalies through bile acid testing and other relevant assessments.

In dogs with hyperuricosuria, urate stone dissolution may be attempted using a combination of allopurinol and a low-purine, alkalinizing diet. Dissolution is successful in approximately 40% of cases, with most failures attributed to the formation of xanthine stones. Dissolution is not recommended in dogs with hepatic vascular anomalies due to poor outcomes; full correction of the anomaly may lead to stone dissolution, but incomplete correction yields unsatisfactory results. No data currently exists on urate stone dissolution in cats.

Prevention strategies for urate stones are determined by the underlying cause. In homozygous dogs with the SLC2A9 mutation, if dietary management alone proves inadequate, allopurinol, an inhibitor of xanthine oxidase (Figure 1b), may be used to reduce urinary uric acid excretion. However, this therapy increases the risk of xanthine stone formation.

Prevention strategies for urate stones:

- Increase water consumption and provide a high-moisture diet to decrease USG: **≤1.020 for dogs and <1.030 for cats.**
- Alkalinize the urine: **pH ≥7** through an alkalinizing diet, with or without potassium citrate supplementation.
- Dietary considerations: a **low-purine diet** is recommended to minimize uric acid accumulation in the urine.
- **Allopurinol** may be considered for dogs homozygous for the relevant mutation when dietary therapy alone proves insufficient.
- In cases involving hepatic vascular anomalies: **surgical correction** of the anomaly is strongly advised.

c. Xanthine uroliths

Xanthine stones are very rare, accounting for 0.05-0.5% of all submissions in dogs and 0.1% in cats. Xanthine is a byproduct of purine metabolism (Figure 1a) and is poorly water-soluble at any pH. Its accumulation typically occurs when xanthine oxidase is inhibited by allopurinol or due to genetic defects. Approximately 75% of canine xanthine stones result from allopurinol treatment.

In cases where xanthine stones are secondary to allopurinol, discontinuing the medication (if feasible) and transitioning to a low-purine alkalinizing diet may promote stone dissolution. However, most xanthine stones do not dissolve and require removal via minimally invasive techniques or surgical procedures.

Prevention strategies for xanthine stones:

- Dietary considerations: A **low-purine diet** is advised to minimize xanthine accumulation, especially in animals on long-term allopurinol therapy.
- **Stop allopurinol, if possible.**
- Hydration: Though studies suggest that xanthine stone formation is not directly linked to USG, increasing water intake and providing a high-moisture diet to lower USG may still be beneficial.
- Urine pH: Since xanthine is only moderately soluble at any urine pH, there are no specific recommendations for altering urine pH as a preventive measure.

d. Compound uroliths

Uroliths with varying compositions in their nucleus and envelope are referred to as compound uroliths. These can form when changes in urine conditions—due to dietary adjustments or treatment modifications—occur, or when a primary urolith (e.g., calcium oxalate or urate) facilitates urinary tract infection, leading to the crystallization of struvite over the pre-existing stone.

Management strategies should initially focus on addressing the urinary tract infection that promotes struvite formation, if applicable. Subsequent preventive measures should be tailored to the composition of the stone's nucleus.

e. Other types of uncommon uroliths

Calcium phosphate uroliths (apatite, brushite, whitlockite, octacalcium phosphate) are rare in dogs but are more frequently observed in those with hypercalcemia. Preventive strategies for these stones are similar to those used for calcium oxalate stones.

Similar to struvite, calcium phosphate carbonate uroliths typically forms as a result of urinary tract infections caused by bacteria that produce the enzyme urease. It is also frequently observed in breeds predisposed to calcium oxalate urolithiasis. Preventive strategies for calcium oxalate uroliths are relevant, and effective management of urinary tract infections and their contributing factors is crucial.

Silica stones are rarely reported but predominantly occur in male dogs (90%). The formation of these stones appears to be strongly associated with the consumption of specific dietary ingredients (such as corn gluten feed, soybean hulls, or intact grains) or water from volcanic regions (e.g., Northwest America, Central America, Japan). Additionally, German Shepherds might be considered a predisposed breed. Silica stones typically form in acidic urine with a pH below 6.5.

Preventive measures include avoiding diets or water sources high in silica, administering potassium citrate to alkalinize the urine (maintaining a pH between 7 and 8), and providing a wet diet to reduce urine specific gravity (USG) to below 1.020.

Dried solidified blood stones are primarily reported in middle-aged male cats. The pathogenesis of these stones is not well understood, but hematuria may be a predisposing factor.

Stones composed of GS-441524 have recently been reported in cats treated for feline infectious peritonitis with remdesivir or its metabolite GS-441524. This occurrence is attributed to the poor solubility of the compound in urine.

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REGENERATIVE ANEMIA

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Definition

Anemia is characterized by a reduction in hematocrit (Ht), packed cell volume (PCV), red blood cell (RBC) count, or hemoglobin concentration (Hb). The interpretation of these parameters is influenced by factors such as hydration status, age, and breed. Anemia can be classified based on the degree of erythropoietic response as either regenerative or non-regenerative (Figure 1). Additionally, anemia may be categorized according to RBC size and hemoglobin content into normocytic normochromic, macrocytic hypochromic, or microcytic hypochromic subtypes.

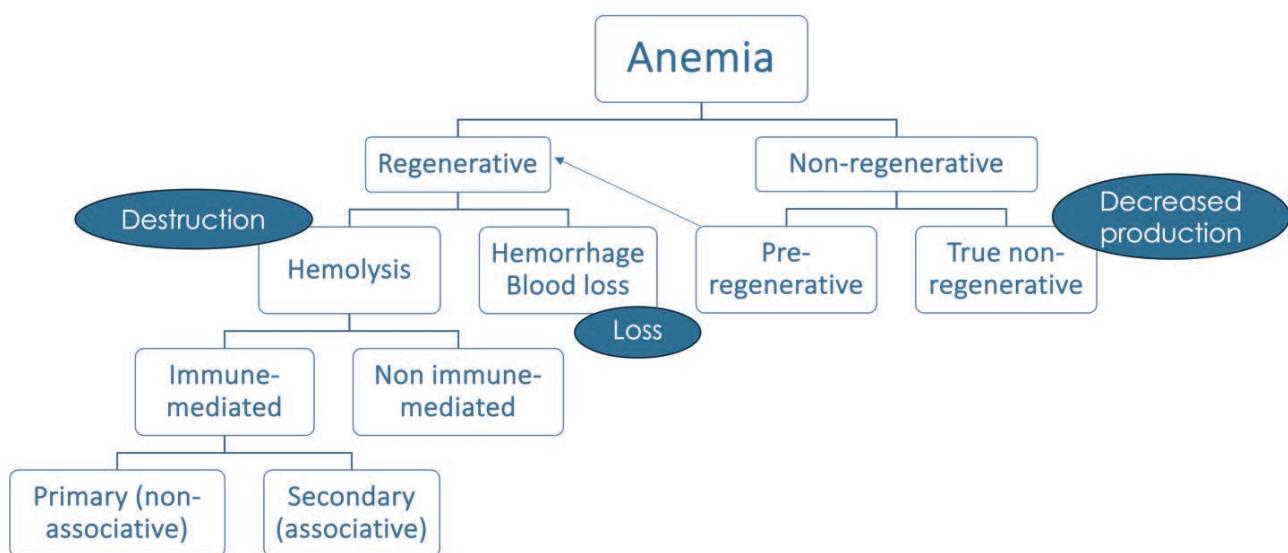


Figure 1: Classification of anemia based on the degree of erythropoietic response

Is the anemia regenerative?

The evaluation of blood smears and reticulocyte indices is essential to assess the quality of erythroid regeneration. Indicators such as polychromasia, anisocytosis*, macrocytosis*, Howell-Jolly bodies*, or the presence of nucleated RBCs* suggest regenerative activity (*these features may also result from other conditions and are not exclusively indicative of regenerative anemia). Objective measures, such as the absolute reticulocyte count (Table 1) and the corrected reticulocyte percentage ($CRP = \% \text{ reticulocytes} \times Ht_{\text{patient}} / Ht_{\text{normal}}$), are valuable for quantifying regeneration. Anemia is classified as regenerative when CRP exceeds 1% in dogs or 0.4% in cats.

Regeneration	Canine	Feline
Weak	90-200	60-100
Moderate	200-300	100-200
Strong	> 300	> 200

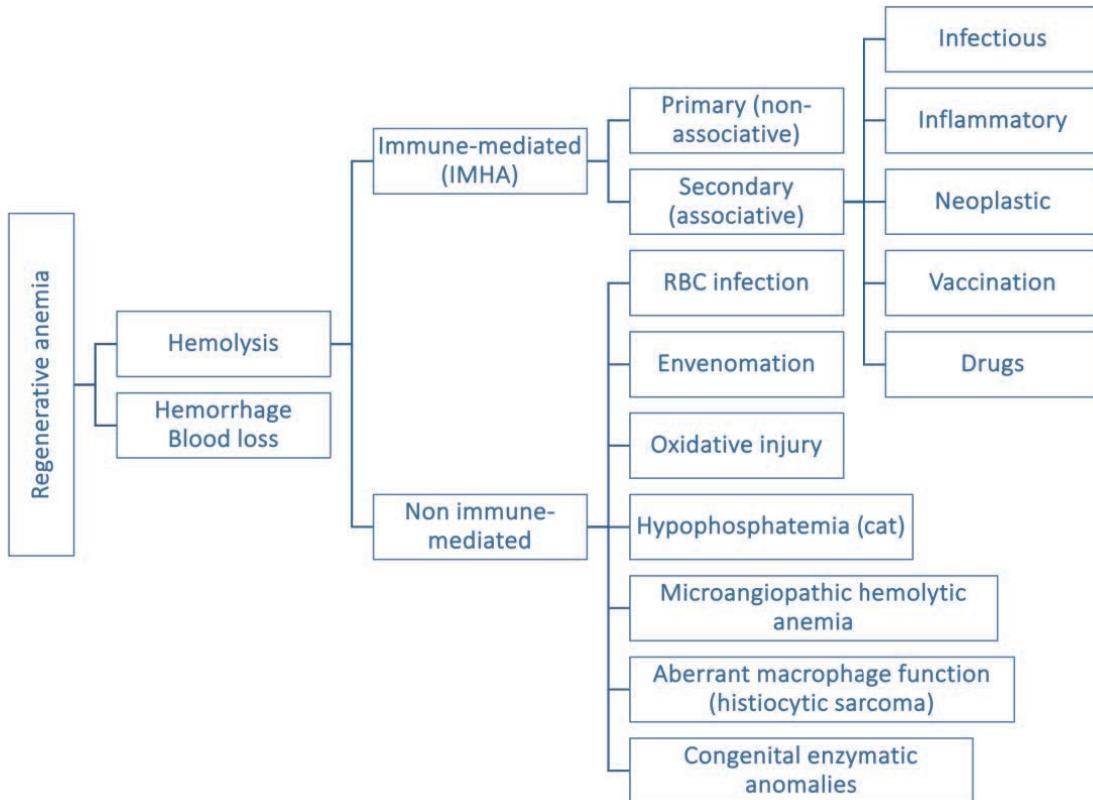
Table 1: Regeneration strength according to reticulocyte count ($\times 10^9/\text{L}$ or $\times 10^3/\mu\text{L}$)

How acute is the anemia?

The determination of whether anemia is acute or chronic can often be inferred from the patient's history (e.g., prior blood tests, known trauma) and clinical signs such as tachypnea, tachycardia, bounding pulse, and weakness. This distinction is crucial in guiding diagnostic evaluation and treatment. Acute and severe anemia may appear non-regenerative during the first 3-5 days but can require a work-up for regenerative anemia. Conversely, a severe non-regenerative anemia without signs of decompensation is unlikely to be acute and in a pre-regenerative phase. This differentiation influences treatment strategies: for instance, a

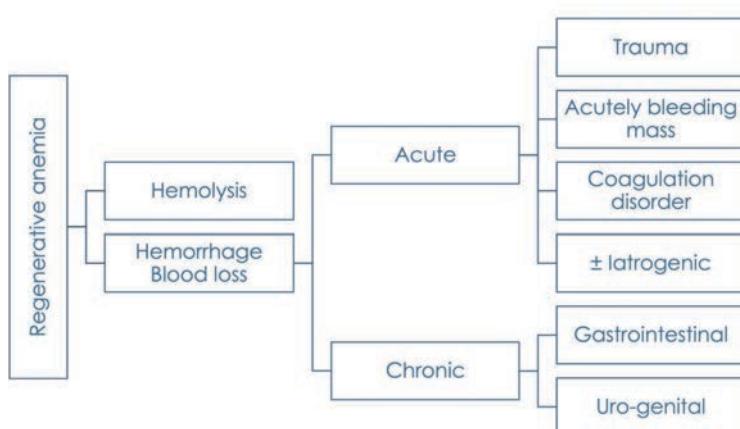
patient with moderate but very acute anemia may necessitate a blood transfusion, whereas another with chronic anemia of the same severity might not.

Differential diagnosis of regenerative anemia - Hemolysis



- Secondary IMHA: level of evidence is intermediate to high for Babesia sp and Mycoplasma sp infections (Garden et al, 2019); for all other causes, level of evidence is low or non-existent (Garden et al, 2019), but any possible cause should be excluded carefully.
- IMHA secondary to inflammatory processes: severe pancreatitis, extended necrosis
- RBC infection: Babesia sp, Mycoplasma sp, Cytauxzoon felis
- Oxidative injury to RBC: acetaminophen, aspirin, methylene blue, Allium sp, repeated propofol administration (cats), heavy metals, Zn, Cu, iron
- Congenital enzymatic anomalies: PK deficiency (dogs and cats), PFK deficiency (dogs)
- Hypophosphatemia (cats): described in refeeding syndrome or ketoacidotic diabetes mellitus
- Microangiopathic hemolytic anemia: DIC, hemangiosarcoma, etc

Differential diagnosis of regenerative anemia – Hemorrhage/Blood loss



Work-up for regenerative anemia

A baseline work-up is essential for identifying signs of hemolysis or hemorrhage and should encompass a comprehensive history and physical examination, complete blood count, biochemistry panel, urinalysis, and blood smear evaluation. Once the pathophysiological mechanism of the anemia is established, further diagnostic tests can be employed to refine the differential diagnosis.

Below is a list of guiding questions to assist in the work-up process:

1. History of envenomation, intoxication?
2. Severe hypophosphatemia?
3. Normocytic, microcytic or macrocytic? Normochromic or hypochromic?
4. Signs of bleeding: History/Signs of bleeding? Hypoalbuminemia/globulinemia?
5. Signs of hemolysis: Hyperbilirubinemia? Hemoglobinemia/uria? Erythrocyte ghosts?
6. Signs of immune-mediated process: Spherocytes? Positive saline agglutination test? Positive direct Coombs test or flow cytometry?
7. Is the cause of the anemia visible on the blood smear?
8. Erythrocyte morphology anomalies?
9. History? (vaccination, drugs, tick/flies protection)

Depending on the clinical hypotheses generated from the initial evaluation, further diagnostic tests may be necessary. These may include blood smear evaluation by a board-certified clinical pathologist, saline agglutination testing, Coombs test, PCR/serology for vector-borne diseases, and imaging studies such as chest radiographs or abdominal ultrasound. Additional procedures may involve rectal examination, fecal analysis, coagulation panel, iron studies, and cytological evaluation of organs such as the liver, spleen, and lymph nodes.

Treatment and prognosis

The primary principles of treatment include the elimination of any toxic substances, addressing the underlying condition, and ensuring that all necessary diagnostic samples are collected before initiating immunosuppressive therapy, provided that this does not excessively delay treatment. In cases of acute and/or severe anemia, blood products are frequently required. Either packed red blood cells or whole blood should be administered, with careful consideration of the potential effects of autoagglutination on cross-matching or blood-typing tests.

The decision to administer blood products should be based on the animal's clinical status, including respiratory rate, cardiac rate, pulse quality, and overall condition. For example, an animal with moderate but acute anemia (e.g., a dog with hemoabdomen and a hematocrit of 22%) may require a blood transfusion, while another animal with moderate to severe chronic anemia (e.g., a cat with pure red cell aplasia and a hematocrit of 12%) may not show signs of decompensation and therefore may not require a transfusion. For immune-mediated hemolytic anemia (IMHA) in dogs, treatment guidelines are outlined in the ACVIM consensus (2019) and should be adhered to for managing both primary and secondary IMHA. Similar principles may be applied to cats, although no large-scale studies have provided specific recommendations for feline cases.

The prognosis depends on the underlying cause of the anemia and the animal's response to treatment.

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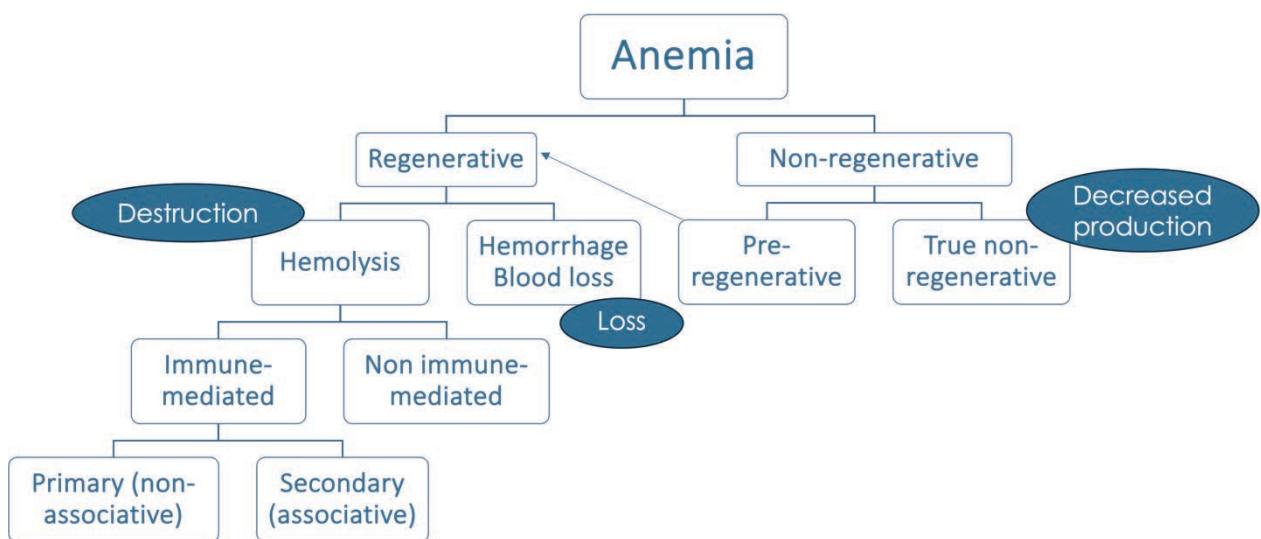
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NON-REGENERATIVE ANEMIA

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Definition

Anemia is characterized by a reduction in hematocrit (Ht), packed cell volume (PCV), red blood cell (RBC) count, or hemoglobin concentration (Hb). The interpretation of these parameters is influenced by factors such as hydration status, age, and breed. Anemia can be classified based on the degree of erythropoietic response as either regenerative or non-regenerative (Figure 1). Additionally, anemia may be categorized according to RBC size and hemoglobin content into normocytic normochromic, macrocytic hypochromic,



or microcytic hypochromic subtypes.

Figure 1: Classification of anemia based on the degree of erythropoietic response

Is the anemia regenerative?

The evaluation of blood smears and reticulocyte indices is essential to assess the quality of erythroid regeneration. Indicators such as polychromasia, anisocytosis*, macrocytosis*, Howell-Jolly bodies*, or the presence of nucleated RBCs* suggest regenerative activity (*these features may also result from other conditions and are not exclusively indicative of regenerative anemia). Objective measures, such as the absolute reticulocyte count (Table 1) and the corrected reticulocyte percentage ($CRP = \% \text{ reticulocytes} \times Ht_{\text{patient}} / Ht_{\text{normal}}$), are valuable for quantifying regeneration. Anemia is classified as regenerative when CRP exceeds 1% in dogs or 0.4% in cats.

Regeneration	Canine	Feline
Weak	90-200	60-100
Moderate	200-300	100-200
Strong	> 300	> 200

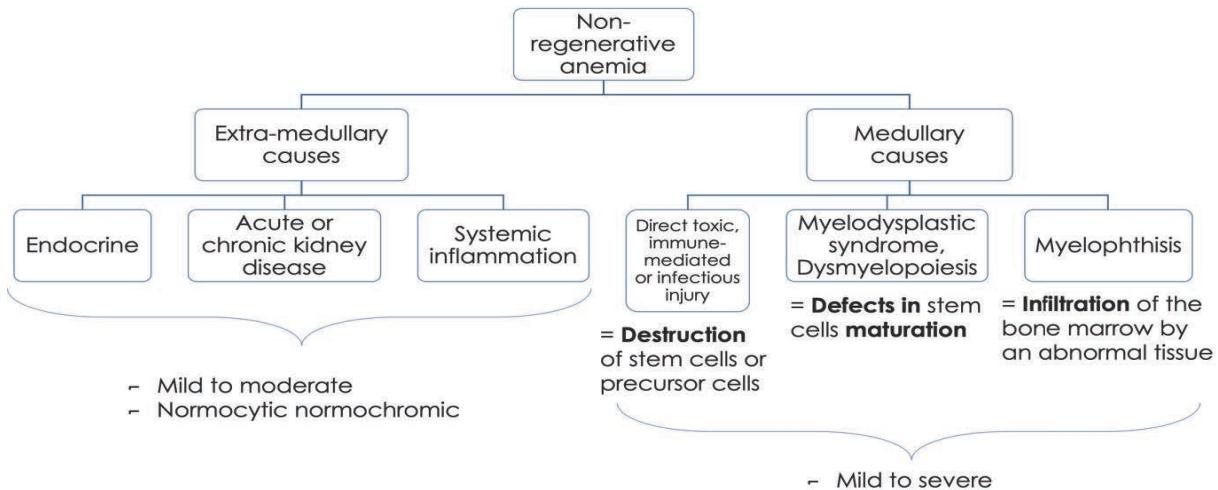
Table 1: Regeneration strength according to reticulocyte count ($\times 10^9/\text{L}$ or $\times 10^3/\mu\text{L}$)

How acute is the anemia?

The determination of whether anemia is acute or chronic can often be inferred from the patient's history (e.g., prior blood tests, known trauma) and clinical signs such as tachypnea, tachycardia, bounding pulse, and weakness. This distinction is crucial in guiding diagnostic evaluation and treatment. Acute and severe anemia may appear non-regenerative during the first 3-5 days but can require a work-up for regenerative anemia. Conversely, a severe non-regenerative anemia without signs of decompensation is unlikely to be acute and in a pre-regenerative phase. This differentiation influences treatment strategies: for instance, a

patient with moderate but very acute anemia may necessitate a blood transfusion, whereas another with chronic anemia of the same severity might not.

Differential diagnosis of non-regenerative anemia



Extra-medullary causes: These are usually normocytic, normochromic anemias.

- Endocrine
 - Hypothyroidism (30%): mild
 - Hypoadrenocorticism (~25%): mild to moderate
 - Acute or chronic kidney disease
 - Acute or chronic inflammation

Medullary causes

- Destruction of stem/precursor cells = Direct injury to stem cells or precursor cells
 - Toxic: many substances are known for their medullary toxicity; most commonly used and reported are chemotherapeutic agents, phenobarbital and derivatives, azathioprine, estrogens, carprofen, some antibiotics (sulfonamides, cephalosporins), heavy metals, griseofulvin
 - Immune-mediated: pure red cell aplasia (PRCA), precursor-targeted immune-mediated anemia (PIMA)
 - Infectious: Parvovirus, Ehrlichia canis, Leishmania infantum, FeLV, FIV, endotoxins, mycotoxins, Histoplasma capsulatum
 - Defects in stem cells maturation = Myelodysplastic syndromes/Dysmyelopoiesis
 - Invasion of bone marrow compartment by an abnormal tissue = Myelophthisis
 - Neoplastic
 - Myelofibrosis
 - Granulomatous diseases
 - Lipid-storage disorders

Work-up for non-regenerative anemia

- Thorough history
 - Thorough physical examination
 - Initial evaluation: CBC and blood smear examination, biochemistry panel, urinalysis
 - Endocrine testing if indicated
 - CRP, SAA
 - Bone marrow aspiration
 - PCR on bone marrow (FeLV, parvovirus, etc) if indicated
 - ± Bone marrow biopsy
 - ± Imaging

Treatment of non-regenerative anemia

The treatment of non-regenerative anemia typically involves addressing the underlying cause or eliminating the causative toxin, which often resolves both the anemia and any associated biochemical abnormalities. In cases of immune-mediated disorders or myelodysplastic syndromes, immunosuppressive therapy is required. One or more blood transfusions may be necessary at the onset of treatment or during the diagnostic process if the animal exhibits signs of decompensation.

The prognosis is largely dependent on the underlying cause of the anemia and can range from good to poor.

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**Alexandra Gabriel (Switzerland)**

DECVIM-CA Internal Medicine

EERVC 2024 Lectures

1. Chronic enteropathies: diagnostic challenges
2. Canine chronic bronchitis: the tough side
3. Feline bronchitis: management problematics
4. Upper respiratory tract disease: chronic rhinitis and other demanding diseases

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CHRONIC ENTEROPATHIES PART I AND II: DIAGNOSTIC AND TREATMENT CHALLENGES

Chronic enteropathies (CE) in dogs and cats encompass a spectrum of gastrointestinal inflammatory disorders characterized by chronic (3 weeks or longer) persistent or recurrent gastrointestinal signs such as vomiting, diarrhea, weight loss, nausea, borborygmus, flatulence, eructation, abdominal pain and altered appetite. The cause is thought to be a complex interaction of genetics, the microbiome and the immune system. These conditions can be classified into food-responsive enteropathies (FREs), microbiota-related modulation-responsive enteropathies (MrMREs, also named antibiotic-responsive enteropathies (AREs)), immunosuppressant-responsive enteropathies (IREs), and non-responsive enteropathies (NREs). The diagnostic work-up of CE is challenging and typically involves a combination of a thorough patient history, physical examination, clinicopathologic evaluation and diagnostic imaging to exclude other gastrointestinal or extra-gastrointestinal disease. Histopathology (endoscopic or laparoscopic biopsies) and adequately designed treatment trials aid in disease classification and in determining the optimal individual treatment plan. Clinical scoring systems can help to assess the initial clinical disease severity and monitor patient evolution during treatment.

The treatment approach for CE is largely dependent on the category and the severity of the disease. Nutritional management, particularly with hypoallergenic or hydrolyzed diets, plays a central role in treating CE. As alterations in the gut microbiota composition and function are involved in the CE pathogenesis and the use of several antibiotics has been shown to increase antimicrobial resistance and to result in deleterious effects on the gut microbiota, the treatment approach of CE has evolved. The use of antibiotics diminishes and is replaced by other treatments (probiotics, fecal microbiota transplantation, etc) aiming to restore diversity and function of the gut microbiota. IREs often require immunosuppressive therapy, with corticosteroids being the most commonly used agents. In cases of NREs, the prognosis is guarded. Individualized work-up and therapeutic plans with practitioner-client communication are crucial for the diagnosis and the treatment of CEs in dogs and cats.

These references provide a comprehensive overview of chronic enteropathies in dogs and cats, their complex nature and the various approaches to diagnosis and management.

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CANINE CHRONIC BRONCHITIS: THE TOUGH SIDE

Canine chronic bronchitis (CCB) is a persistent inflammatory condition of the lower airways, commonly affecting middle-aged to older dogs. It is characterized by a chronic cough lasting more than two months, without an identifiable underlying cause such as infection, neoplasia, or cardiac disease. The condition may be linked to exposure to environmental irritants, such as cigarette smoke or dust amongst others, which lead to the chronic airway inflammation, but usually the exact cause of spontaneously occurring CCB remains unknown. Clinical manifestations include a dry, hacking cough that may worsen with exercise, excitement, or exposure to irritants, and respiratory distress in advanced cases. Diagnosis is typically based on history, clinical signs and physical examination findings, exclusion of other respiratory conditions by thoracic radiographs and bronchoscopy with analysis of bronchoalveolar lavage fluid. Treatment focuses on managing clinical signs, slowing disease progression and improving the quality of life, often involving a combination of different treatments: corticosteroids, bronchodilators, and antitussives, along with lifestyle and environmental modifications. Prognosis varies depending on the severity of the disease and the response to treatment, but many dogs, despite the absence of a definitive cure, can lead a good quality of life with proper management. Individualized long-term management and practitioner-client communication is critical to improving the quality of life for affected dogs.

These references provide a comprehensive overview of canine chronic bronchitis, its diagnosis and management.

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FELINE BRONCHITIS: MANAGEMENT PROBLEMATICS

The term feline inflammatory lower airway disease regroups what was called feline bronchitis: feline asthma (FA) and feline chronic bronchitis (FCB) are considered the most common. These conditions are characterized by chronic inflammation of the lower respiratory tract. Pathophysiology involves an interplay of genetic predisposition, environmental factors and hypersensitivity reactions. Clinically, affected cats may present with symptoms such as coughing, wheezing and dyspnea. Diagnostic work-up typically involves a combination of a thorough history, physical examination, clinicopathologic evaluation, thoracic radiography, bronchoscopy and bronchoalveolar lavage to rule out other potential causes of chronic cough such as neoplasia or infections. Management of these diseases often requires lifelong therapy that focuses on controlling inflammation and relieving bronchoconstriction through the use of corticosteroids, bronchodilators and environmental management to reduce exposure to potential allergens. While acute episodes might be life-threatening, with appropriate treatment and monitoring, many cats can lead relatively normal lives. Effective practitioner-client communication and empathy is essential in the disease management. Ongoing research aims to better understand the disease's etiology, improve diagnostic accuracy, and develop more effective treatments.

These references provide a comprehensive overview of feline inflammatory lower airway disease, its diagnosis and management.

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UPPER RESPIRATORY TRACT DISEASE PART I AND II: CHRONIC RHINITIS AND OTHER DEMANDING DISEASES

Upper respiratory tract diseases in cats and dogs are commonly encountered in small animal practice. Clinical signs include nasal discharge, sneezing, dyspnea amongst others. They are often similar regardless of the specific underlying disease process, but some signs may be more often identified with a specific cause, such as facial deformity with nasal neoplasia rather than other causes of nasal disease. Nasal neoplasia, dental disease, infectious (viral, bacterial or fungal) or inflammatory rhinitis, foreign bodies, malformations and nasal polyps (cats) belong to the differential diagnoses of upper respiratory tract disease. The diagnostic work-up typically involves a combination of a thorough patient history, physical examination, clinicopathologic evaluation (hematology, biochemistry, PCR, culture, etc) and diagnostic imaging techniques (dental radiographs, thoracic radiographs, computed tomography of the skull, rhinoscopy, etc). Rhinoscopy allows direct visualization and interventions such as foreign body extraction, collection of biopsies for histopathology, treatment for nasal aspergillosis and others. Treatment, medical and/or surgical, depends on the underlying cause. Individualized work-up and therapeutic plans with practitioner-client communication are crucial for the diagnosis and the treatment, especially in chronic disease.

These references provide a comprehensive overview of upper respiratory tract disease in cats and dogs, covering aspects of diagnosis, treatment, and management.

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EERVC 2024 Lectures

1. Protein-losing enteropathy
2. Promoting healthy weight in senior cats
3. Feline IBD vs lymphoma
4. Caring for dogs and cats with obesity
5. Prevention of obesity

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Clinical and Research Interests

- Internal medicine
- Gastroenterology
- Obesity biology
- Healthy aging science
- Evidence-based medicine

PROTEIN-LOSING ENTEROPATHY

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Introduction

Protein-losing enteropathy (PLE) is a syndrome caused by protein leakage into the gut lumen at a level that exceeds plasma protein synthesis. The syndrome manifests as chronic diarrhoea associated with hypoproteinaemia, and usually requires intestinal biopsy to define the cause. For many cases, the prognosis is guarded and aggressive therapy is often required, and correct choice of drugs is vital to success. This lecture will describe how cases of PLE manifest, discuss the approach to diagnosis, and also describe the latest information regarding therapy.

Disease associated with protein-losing enteropathy

Although a range of diseases can cause chronic diarrhoea in dogs, only a handful are associated with concurrent gastrointestinal protein loss. The three most important conditions include chronic enteropathy (CE, also known as inflammatory bowel disease, IBD), lymphangiectasia and alimentary lymphoma. Although gastrointestinal blood loss can also lead to hypoalbuminemia, usually the clinical signs relate to reduce red cell mass rather than to consequences of PLE. Further, there have also been descriptions of dogs with small intestinal crypt lesions presenting with PLE, although it is not clear whether this represents a distinct disorder, or whether they are instead a form of CE. Indeed, WSAVA histopathology standards have included crypt lesions in the grading scheme for cases of IBD. Finally, in some parts of the world, some gastrointestinal infections can also cause GI protein loss (e.g. histoplasmosis, pythiosis).

Chronic enteropathy (inflammatory bowel disease)

Inflammatory bowel disease (IBD) is a collective term describing a group of disorders characterised by persistent or recurrent GI signs, with histological evidence of intestinal inflammation. Variations in the histological appearance of the inflammation suggest that idiopathic IBD is not a single disease entity, and nomenclature reflects the predominant cell type present. It is a controversial, enigmatic, condition and much remains to be understood of its aetiopathogenesis, diagnosis and optimal treatment.

IBD is most common in middle-age animals, and there is no apparent gender predisposition. Although IBD can potentially occur in any dog or cat breed, some breeds are predisposed e.g. GSDs, soft coated wheaten terriers, Shar peis and Siamese cats. Vomiting and diarrhoea are the most common clinical signs. Severe disease is associated with weight loss and PLE, with consequent hypoproteinaemia and ascites.

Treatment usually involves using a combination of dietary modification, antibacterials and immunosuppressive therapy, with immunosuppressive therapy being most important when PLE is present (see below). Additional benefit can also be seen with dietary modification, with hydrolysed diets being most effective. Some veterinarians use antibacterials in the treatment of IBD, although this is becoming more controversial. The two drugs most commonly used are metronidazole and tylosin. The author now uses these drugs less frequently but, when he does, his preference is to use tylosin. Finally, supplementation with either injectable or oral cobalamin is indicated if serum concentrations are subnormal.

Lymphangiectasia

Lymphangiectasia is defined as abnormal dilatation of intestinal lymphatics. Its aetiology is unknown, and is seen most frequently in Yorkshire terriers, Maltese terriers, and Rottweilers. Pathophysiology involves leakage of lipoproteins and lymphocytes from lacteals ± associated lymphangitis. In some cases, lymphangiectasia results from obstructed lymphatics (e.g. from an inflammatory cell infiltrate); in other cases, the reason for lymphatic obstruction is unknown. Clinical signs include severe weight loss, diarrhoea, and altered fluid homeostasis due to hypoproteinaemia leading to ascites, and subcutaneous oedema.

With regard to diagnosis, a number of abnormalities can be noted on *routine laboratory analysis*:

- hypoproteinaemia
- hypocholesterolaemia
- lymphopenia
- normal TLI, folate and B12

Intestinal biopsy demonstrates the typical histopathological characteristics. However, a full-thickness biop-

sy may be required to make the diagnosis since superficial mucosal biopsies may miss the lesions. Treatment may include the following:

- A low fat diet
- Medium chain triglyceride (MCT) oil was previously recommended on the basis that they were absorbed directly into the circulation rather than into the lymphatics. However, this has subsequently been disproved and MCTs are now rarely used.
- Anti-inflammatory drugs (prednisolone) are indicated for some cases (if the underlying aetiology is an inflammatory disease). However, they may not benefit all cases.
- Cases of lymphangiectasia have a very guarded prognosis

Lymphoma

In dogs, alimentary lymphoma usually presents with severe clinical signs (vomiting, diarrhoea, weight loss) and has a rapid progression. The disease may either be diffuse or nodular in distribution. In diffuse cases, there may be signs consistent with PLE. Occasionally, cases will present with ulceration leading to haemorrhage and then anaemia. As mentioned above, PLE is only an occasional manifestation in alimentary lymphoma.

Clinical signs include anorexia, depression, weight loss, vomiting & diarrhoea ± ascites. With regard to diagnosis, there may be findings consistent with PLE on routine laboratory analyses. However, intestinal biopsy is required to make a definitive diagnosis. Full-thickness biopsies are again preferred as endoscopic biopsies can be misleading. This is because lymphoma changes can be deep, or be interspersed with IBD. It has also been postulated that progression from IBD to lymphoma can occur in some cases.

Prognosis is usually poor. There may be temporary palliation with prednisolone. However, there is usually a poor response to chemotherapy. In addition, if treatment with chemotherapy is effective, rapid lysis of neoplastic lymphocytes may lead to intestinal perforation.

Clinical presentation

As described above for the individual conditions, PLE can present in dogs of either sex, with a wide range, though most typically in adult dogs (>2y old). Dog breeds predisposed to PLE include Basenjis, Norwegian Lundehunds, Soft-coated Wheaten terriers, Yorkshire terriers, and Shar peis. Associated clinical signs include weight loss, diarrhoea, vomiting, melaena, subcutaneous oedema, ascites, and pleural effusion. Thromboembolism secondary to hypoproteinaemia is a feature of some cases of PLE.

Diagnosis

Diagnosis requires a combination of laboratory testing, diagnostic imaging and GI biopsy. Serum concentrations of both albumin and globulin are decreased in most patients with PLE. Exceptions are hyperglobulinaemia found in histoplasmosis and basenji enteropathy. Renal and hepatic causes of hypoalbuminemia can be eliminated by measuring serum bile acid concentrations and urinary protein loss (e.g. by calculating protein:creatinine ratio), respectively. Hypocholesterolaemia and lymphopenia are common in PLE, whilst hypocalcaemia and hypomagnesaemia are also reported. Measurement of faecal loss of alpha1-protease inhibitor may be a sensitive test for PLE, although the assay is difficult to perform and is not widely available.

Survey abdominal radiographs often are unhelpful in patients with PLE because of the loss of contrast, but ultrasonography may reveal intestinal thickening, and/or mesenteric lymphadenopathy, and/or abdominal effusion. Recent studies have suggested that transverse linear striations correlate with the presence of protein-losing enteropathy; although these striations are commonly assumed to be the result of lacteal dilation, this has **not yet been confirmed**, and the finding has not been related to a particular histopathological pattern. Intestinal function tests (e.g. measurement of folate and cobalamin) may confirm the presence of malabsorption, but they rarely provide a definitive diagnosis, and intestinal biopsy is more appropriate. Because many intestinal causes of PLE are diffuse, endoscopy is the safer way to obtain biopsies, but surgical biopsy may be required for definitive diagnosis for transmural lymphoma and lymphangiectasia.

Thus, histopathological assessment of biopsy material remains the gold standard, but interpretation is subjective, and agreement between pathologists is often poor. Further, it can be difficult to differentiate severe IBD changes from those of alimentary lymphoma. Thus, results should always be interpreted in light of the clinical presentation, and other findings should be used in conjunction, most notably response to treatment.

Treatment and prognosis

Whatever the diagnosis, hypoalbuminemia is a poor prognostic indicator. As a result, it is essential that cas-

es are treated aggressively and rapidly. In the initial stages, treatment involves provide intravenous colloid support or plasma transfusion, during the perioperative period when collecting biopsy specimens, and diuretics may reduce ascites. Spironolactone may be more effective than furosemide for treating ascites.

Specific treatments for the most common conditions have been discussed above. Alimentary lymphoma carries a poor prognosis in dogs and, whilst chemotherapy can be attempted, the chance of success is low. All remaining cases are treated with combination therapy, which involves dietary management (a hydrolysed diet), parenteral cobalamin (if hypocobalaminhaemia is present), and immunosuppressive therapy. In the past, prednisone or prednisolone and azathioprine was the treatment of choice, with ciclosporin reserved for those cases that fail to respond. However, response to therapy was poor with the majority (~75%) not improving. However, a recent study (Dandrieux et al., 2013) demonstrated good response to a prednisolone-chlorambucil combination as follows:

- Prednisolone at 1-2 mg/kg q24h PO. The drug is tapered gradually if response is favourable.
- Chlorambucil at 4-6 mg/m² q24h PO. The drugs is also tapered gradually, usually once the prednisolone has been reduced to an ever-other-day dose.

Haematological parameters and serum proteins are monitored regularly, to confirm that there are no side effects (bone marrow suppression) and determine response to therapy. Therapy is gradually tapered if a response is seen, usually by decreasing the prednisolone dose first (by 25-33% every 2-3 weeks), and chlorambucil thereafter. In the author's experience, cases often relapse after withdrawal of therapy. In such cases long-term low-dose therapy should be continued.

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PROMOTING HEALTHY WEIGHT IN SENIOR CATS

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Introduction

It has long been known that the body condition of cats is associated with overall health and wellbeing. Indeed, there are risks to cats both when they are above and significantly below their ideal condition. Maintaining ideal weight is key to ensuring the health and wellbeing of cats. This can become especially difficult in senior cats. Obesity is a problem of increasing importance and almost half of all cats are overweight, including many senior cats. This can affect quality of life in the older cat mainly through the comorbidities that may arise. Other senior cats may be prone to weight loss, often as a result of chronic diseases that are prevalent in this age group. These cats tend to lose bodyweight. This lecture will focus on maintaining healthy weight in senior cats, both in terms of maintaining weight in cats prone to weight loss, and modest weight loss in those that are overweight. In both instances, preserving lean body mass (LBM) is key to ensuring health and wellbeing.

Underweight senior cats

As mentioned above, many cats are prone to weight loss during their senior phase and this has been associated both with increased morbidity¹ and shortened lifespan.² Broadly speaking, two syndromes of weight loss are recognized, **cachexia** and **sarcopenia**.³ Sadly, there is little research on these two conditions in cats, and much of what we know has been extrapolated from human studies.

Cachexia

Cachexia, a loss of LBM, is common in cats that suffer from with chronic diseases, such as congestive heart failure, chronic kidney disease and cancer.³ The weight loss that occurs in cachexia is different from that seen in healthy animals that lose weight; when a healthy animal that is receiving insufficient dietary energy to meet requirements, metabolic adaptations allow fat to be used as the primary fuel source, thus preserving LBM. Conversely, acute and chronic diseases alter concentrations of a variety of mediators (e.g., inflammatory cytokines, catecholamines, cortisol, insulin, glucagon), which then decrease the ability to

make metabolic adaptations required to switch to fat utilization, and amino acids continue to be used as a primary source of energy. Therefore, LBM is rapidly catabolized and, although fat and bone mass are also lost, this tends to be less pronounced relative to LBM. In humans, cachexia has been associated with increased energy requirements, decreased nutrient absorption, and decreased dietary energy intake due to partial or complete anorexia.³ Unfortunately, research into the pathogenesis of feline cachexia is limited and further work is required.

Sarcopenia

The term sarcopenia comes from the Greek words 'sark' (meaning flesh) and "penia (meaning loss) was originally meant to represent age-related loss of muscle mass with aging.⁴ The condition also arises during ageing and is similar to cachexia in that it is characterized by loss of LBM. However, unlike cachexia there is no apparent associated chronic illness. Further, in sarcopenia, the loss of LBM often is accompanied by an increase in fat mass so the total weight may not change (or may even increase), thus masking the sarcopenia muscle loss.⁴ Most human definitions of sarcopenia rare related to loss of muscle mass, for example a percentage LBM more than one standard deviation below the reference values from young, healthy individuals measured with bioelectrical impedance.⁵ Given the lack of research into the condition in cats, such defines are not yet well defined. Like cachexia, sarcopenia is associated with increased mortality and also has important effects on quality of life and strength leading to increased frailty. Indeed, many scientists believe that loss of muscle strength (which does not necessarily correlate with the amount of LBM) is more important in disease morbidity than loss of LBM *per se*.⁴ The mechanism of sarcopenia is poorly understand and but is likely multifactorial involving decreased physical activity, increased cytokine production, decreased concentrations of growth hormone and testosterone, changes in type II muscle fibers, insulin resistance, and decreased protein synthesis.³

Sarcopenic obesity

A further confusion in definitions comes from is the recognition of so-called 'sarcopenic obesity', whereby here is a combination of low muscle mass and increased fat mass.⁴ There terminology is arguably problematic since sarcopenia is said to occur in the absence of other chronic illnesses, but obesity is now known to be a chronic disease. Although the syndrome has not been formally defined in cats, and there is little available research, but many clinicians have observed cases with an ideal BCS (see below), but with significant muscle wasting.

The condition can be a challenge to recognize in humans when definition of obesity is based on measures such as body mass index (BMI). Given the loss of muscle mass in sarcopenic obesity, some individuals might have a normal BMI but excessive body fat, so-called "TOFIs" (Thin on the outside, fat on the inside).⁴ Such individuals can be diagnosed if other measures of adiposity are considered including abdominal circumference, waist:height ratio, dual-energy X-ray absorptiometry and computed tomography. Arguably, similar problems can arise in cats if veterinarians rely solely on body weight and body condition when diagnosing obesity. Incorporation of muscle condition scoring (MCS; see below) in conjunction into routine physical examinations can help to identify those whose BCS and MCS may have diverged.

Diagnosis

The major challenges of diagnosing cachexia and sarcopenia in cats are the lack of an accurate definition and a clinically relevant way to diagnose this syndrome.³ Concerns often arising in senior cats when a loss of bodyweight is identified. The main challenge arises with the fact that total weight loss is an insensitive measure of muscle loss and, therefore, cases might not be spotted until they are advanced. Whilst both CT and DEXA are very sensitive measures of LBM, these techniques are not widely available. To address this, veterinarians should be pro-active in identifying and acting on any changes in bodyweight and condition. This increases the chance of identifying cachexia and sarcopenia during the early stages, thereby improve the options for management. As part of practice policy, all cats should have their body weight, BCS and also MCS throughout life and, especially, in seniors.⁴ Use of the 9-point BCS is strongly recommended since this has been most extensively validated and used.⁶ Like BCS, MCS is a quick and simple semi-quantitative method that has been validated against DEXA.⁷ However, MCS complements BCS because it specifically evaluates muscle mass. In this respect, BCS and MCS are often not directly related because an animal can be obese but still have substantial muscle loss (equivalent to sarcopenic obesity in humans). The method involves visual examination and palpation of the head, scapulae, epaxial muscles over the thoracic and lumbar vertebrae, and pelvic bones. Palpation is required in order to measure both BCS and MCS accurately in cats with medium or long hair coats.³

Epidemiological studies suggest that, in adult cats, a BCS of 4-5/9 is associated with the lowest risk of associated diseases,^{1,8} whilst a BCS of 4-7/9 is associated with the longest lifespan.² In order to maximize longevity, whilst minimizing the risk of disease, the author would suggest that most adult cats maintain

a BCS of 4-5/9. However, during the senior stage onwards, and in light of the impact of chronic diseases on LBM, a slightly higher BCS may be desirable (ie, a BCS of 6-7/9). It is important to emphasize that the evidence in support of such an approach is not yet available and, even in animals with these diseases, a BCS >7/9) should be avoided. The author recommends routinely recording these measures at least every 6 months during adult life, and every 3 months from 10 years onwards. Veterinarians should consider further investigations in the event that unexpected changes in body weight are seen, for example a 2% reduction over 3 months and, especially, when there is an unexplained change in muscle mass (decline in MCS). Cachexia should be anticipated in animals with chronic diseases such as CHF, CKD, cancer, and diseases that might alter metabolism.³ Consistently evaluating MCS in all cats will help identify muscle loss at an early stage, rather than waiting until muscle loss is moderate or severe, and consequently more challenging to manage.³

If concerns over bodyweight or muscle mass are identified further investigations should be considered, but full details are beyond the scope of this article. Briefly, initial investigations should include general history, nutritional assessment, physical examination. Further investigations can then be considered as appropriate including CBC, serum biochemistry, T4 measurement, urinalysis, blood pressure analysis, and diagnostic imaging. Others tests can then be considered as the need arises to ensure that the cat's current health is ascertained, and any chronic diseases characterized and appropriately staged.

Management

For cats with chronic diseases and concurrent weight or muscle mass loss, therapy for the underlying disease should be optimized, but a complete discussion is beyond the scope of this lecture. Whatever the associated condition, nutrition and food intake should also be optimized. In many cases, dietary modification, assisted feeding, or other feeding strategies might be needed. Concurrent dental disease can also affect appetite as part of an overall management strategy. Chronic pain is commonly a feature of many chronic diseases in older cats, and may further affect willingness to eat, whilst the need for an owner to medicate the cat can also have an impact.

Ideally, the food that is offered to cats with cachexia and sarcopenia should be complete and balanced, and be palatable to ensure adequate intake.³ However, occasionally, compromises are needed in order to maximize intake. Care should be taken to ensure that the cat consumes sufficient to meet its maintenance energy needs but, since the energy density of senior foods can be highly variable and, as a result it is advisable measure portions accurately using electronic scales. Asking owners to maintain an accurate diary record of food offered and consume can help to monitor efficacy of your feeding strategy. It is recommended that at least the AAFCO minimum for protein (65g/1000 Kcal for cats)^{9,124} is important, although many recommend higher dietary protein levels if it is safe to feed them. Again, veterinarians should be careful when selecting foods because commercially-available senior diets can vary markedly in terms of protein content (48-131g/1000 Kcal for commercial senior dog foods in one study).⁹ Other nutrients can also vary in different diets, and this can have significance when concurrent chronic diseases are present such as CHF and CKD. Most notably, dietary sodium content can vary markedly amongst diets, and additional dietary sodium can arise from feeding extra food to increase palatability. In such cases, veterinarians should consider feeding an appropriate therapeutic diet.

Anorexia is common in cats with chronic disease and, as discussed above, has been implicated in the pathogenesis of cachexia.³ Anorexia can sometimes remain a problem even when the underlying disease is being well managed from a medical perspective and, in such cases, partial rather than complete anorexia is more likely, or there might a change in food preference. Decreased food intake in a cat that previously had a normal appetite might be an early indicator of worsening of the underlying disease. Some cats with chronic disease, for example CKD are reported to have a cyclical appetite, whereby they eat one food for several days or weeks and then refuse it, only to eat it again at a later date. To address decreased food intake, client communication is important. For example, clients can be advised to try different foods that are nutritionally appropriate for their cat (for example different flavors of the same therapeutic diet or therapeutic diets from different manufacturers) in order to maximize daily food intake. A nutritionally balanced, home-prepared diet formulated by a veterinary nutritionist also is an alternative option in clients who are open to it.³ Advising the owner to feed the daily food ration over a series of small frequent meals and warming food can also help to improve overall food intake whilst, in some cases flavor enhancers can help (as long as there are no medical contraindications (e.g. high-sodium and high phosphorus flavor enhancers in CHF and CKD, respectively).

Appetite stimulants (e.g. mirtazapine) can sometimes help, although these should be reserved for those with partial rather than complete anorexia and short-term use is preferable. If such drugs are used, the exact intake should be observed closely (using an owner diary), and bodyweight, BCS and MCS should also

be monitored. This is because it can be easy to over-estimate the efficacy of such a strategy and assume that a cat is meeting their daily need if observed to eat, when in fact they are not. Further, if owners have used other foods to tempt the cat, the cat might prioritize such food to the expense of eating their main meal. A final strategy to consider where food intake remains a concern is nutritional support for example using an esophagostomy tube. Whilst the strategy might not be acceptable to all owners, it can ensure adequate intake of the most appropriate diet. In such cases, early tube placement should be considered because outcome is likely to be better than waiting until the cat is already debilitated from severe weight and LBM.

Finally, exercise is effective in maintaining muscle mass in humans with cachexia and sarcopenia,³ and a balanced regime of aerobic and resistance exercise is usually recommended. Although, similar benefits might be expected, such strategies are likely to be difficult to implement in cats.

Overweight senior cats

The veterinary profession has recently been formally recognized as a disease in pets,¹⁰ with published studies suggesting associations with overweight cats being at risk of developing other diseases such as diabetes mellitus, osteoarthritis, and urinary tract disease and neoplasia.⁸ Given such adverse affects on health and quality of life, feline obesity presents a major welfare challenge for veterinary surgeons. Recent estimates have suggested that almost half of all pet cats are above their ideal weight but, more concerning is the fact that the disease prevalence is rapidly increasing.¹¹ The prevalence of obesity increases steadily throughout early adulthood, reaching a peak at during middle age⁸ and, whilst the prevalence decreases somewhat during the senior phase, a significant proportion of cats in their later years are overweight and the consequences can be particularly problematic, not least because of the challenges of managing body-weight in the face of concurrent disease.

Controlled weight loss and its benefits

Successful weight management has two main phases, weight loss and subsequent weight maintenance. Most commonly, a purpose-formulated diet is used, usually in combination with increasing activity, and feeding a high protein diet results in greater loss of fat mass and greater retention of lean body tissue than feeding a diet containing 30% crude protein. The most effective diets minimize signs of hunger, in order to reduce food-seeking behavior and thereby improve compliance, and this is usually achieved with a diet that has a modestly increased amount of dietary fiber (e.g. 23% total dietary fiber as fed) and protein (e.g. 34% as fed).¹²

Obesity can have a number of adverse effects on health, including shortening lifespan, predisposing to disease, causing metabolic and functional derangements, and worsening the severity of pre-existing disease. Whilst it is commonly suggested that successful weight loss may have a positive impact on all of these adverse effects, there is limited direct evidence for this. For example, the author is not aware of any studies that convincingly demonstrate that weight loss leads to either increases in lifespan or decreased disease risk. However, there is emerging evidence of benefits to function and alleviation of concurrent disease severity. For instance, there is evidence of improved insulin sensitivity and other metabolic derangement when successful weight loss occurs.¹³ Other functional benefits include improved mobility and respiratory function, although, to date, these have only be documented in dogs rather than cats.^{14,15}

Overall success of weight loss in cats

Many veterinarians mistakenly believe that weight loss is straightforward and that most cats will succeed in time. However, studies of human obesity have demonstrated that rate of weight loss tends to plateau after about 6 months on diet-based weight loss program, meaning that most people never reach their target weight, and often then regain the weight they originally lost. Studies in cats reveal a similar pattern: rate of weight loss is relatively fast in the early stages but steadily declines as weight loss progresses. Further, when success of weight loss is studied, only about half of those starting a weight loss plan actually reach their target weight.¹⁶ The degree of obesity is the strongest predictor of failure with the fattest cats being most likely to fail.¹⁶ Moreover, about half of the cats that reach target weight then regain weight. Whilst these studies suggest that current weight management strategies are poorly effective, it should be emphasized that 80% of pet cats lose more than 6% weight, which is often which, as stated above, would be sufficient to produce measurable health benefits.¹⁴ One other issue is that, although the main aim of controlled weight loss is to reduce body fat mass, some loss of LBM is inevitable, and increases as the overall percentage of body weight loss increases.¹⁶ If weight loss is modest <10-15%, loss of LBM is minimal, but significant LBM can occur once >15% of body weight has been lost. Therefore, in the author's opinion, aiming to return as severely obese >40% above ideal weight) cat to its ideal weight may be counterproductive.

In the author's opinion, many owners make the mistake of focusing too heavily on 'the numbers' when judging success of weight management, for example achieving a rate or weight loss of 1-2% per week or reaching the cat's ideal weight. In so doing, the may pay insufficient attention to the overall aim, which is to improve the pet's quality of life, permanently. Thus, rather than worrying if weight loss is too slow, or whether the cat will ever reach its 'perfect weight', the clinician could focus on whether the cat is happier, more active, and less affected by any concurrent disease it may have (e.g. lameness, diabetes mellitus, respiratory diseases etc). This particularly important in senior cats given the fact that concurrent diseases are common (see below). Further, success should be viewed less in terms of reaching a nominal 'ideal weight', than in permanently maintaining any weight lost, so that the health benefits of weight management are maintained. Unless bad owner habits are permanently changed (i.e. avoiding excessive treating, insufficiently exercising their pet), weight management will fail in the long run.

Promoting healthy weight by tailoring weight loss to the individual

As described above, clinical research about weight management in obese pet cats has highlighted the fact that failure in weight management is common revealed the challenges it brings, and the realities in terms of long-term outcomes. Clinicians must accept that current strategies are not perfect, and that many cats will fail. The following clinically proven facts should be considered¹⁶:

- There are potential health and welfare benefits when an obese cat loses weight, most notably through improved function and quality of life.
- Weight management is challenging for owner and cat and becomes increasingly challenging as weight loss progresses.
- Weight management can lead to loss of LBM, most notably for cats losing significant amounts of weight (>15%), and this might have adverse effects on wellbeing, not least given the importance of preserving lean body mass in senior cats.
- Many cats fail to reach their target weight and, even if they do, many cats then rebound.
- However, most cats lose some weight, even if they do not reach target, and even modest amounts of weight loss can have significant health benefits.

Most importantly, the weight loss process in obese cats is a clear example of diminishing returns, namely that the more weight that must be lost, the more difficult it is. The payoff of success in these circumstances is dramatic energy restriction, and the likelihood of some lean tissue loss. In the author's opinion the process of **tailoring weight management** to the individual is key to maximizing success. This concept involves understanding the priorities for weight management for each case, setting case-specific targets, and establishing a realistic plan that will maximize the chance of benefitting the patient long-term. Broadly speaking, two strategies could be considered, **complete weight loss** and **partial weight loss**. As the name suggests, the purpose of a complete weight loss program would be to return the cat to its ideal weight. This has the benefit of 'normalizing' adipose tissue mass, with the aim of producing the maximal benefit to health in terms of improving metabolic status and organ function. Although not proven, returning a cat to ideal condition, and keeping it there might also increase the likelihood of a longer lifespan. Such an approach would be most applicable where obesity develops early in life i.e. young adulthood, where rapid return to normal weight would maximize any potential for extending lifespan. A complete weight loss strategy might also be beneficial for obese cats that do not yet have an obesity-associated disease, since many such diseases, for instance osteoarthritis, are chronic in nature developing insidiously over many years. Once again, return to ideal weight would reduce the impact of obesity on the chronic disease process, thereby delaying its onset or preventing signs from manifesting at all. The disadvantage of a complete weight loss program is the fact that the chance of failure is greater (remember the likelihood of failure is greater when duration is longer), and there is a risk that any weight initially lost might be regained again. Further, as mentioned above, there is no evidence yet available that potential benefits such as increase lifespan and disease prevention actually occur. Moreover, such benefits are less likely to be pertinent for older animals and for those with pre-existing disease.

Instead, devising a shorter-term 'partial' weight loss plan might be preferable. Here, a target weight is deliberately set which is **above the ideal weight**, and this strategy is particularly applicable to weight loss in senior cats not least if concurrent disease is present. Here the priority is to achieve sufficient weight loss to improve function and quality of life, but to minimize the overall duration (to maximize owner compliance) and amount of weight loss (e.g. <15%, to reduce the risk of significant LBM loss). In this respect, evidence is available that modest amounts of weight loss (e.g. ≥ 6%) can lead to alleviation of signs of concurrent disease such as osteoarthritis.¹⁴ Published evidence in cats suggest that over 80% would succeed with such

a plan, and would be likely to lose 8-12% of their starting bodyweight.¹⁶ A partial weight loss plan is particularly suitable for senior cats, not least given the concurrence of other chronic diseases and the importance of maintaining LBM.¹⁷

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FELINE IBD VS LYMPHOMA

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Chronic enteropathies (CE) are common in cats, and can present a challenge to the clinician in terms of diagnosis and treatment. Lymphoplasmacytic enteritis (LPE) and Alimentary Lymphoma (AL) are the two most common such conditions; since both conditions present with similar clinical signs, it can be difficult to differentiate the two. This lecture will provide current information about each of these conditions, in terms of diagnostic and treatment approaches, and the latest information on their classification and differentiation.

Overview of LPE and AL

Lymphoplasmacytic Enteritis (LPE) is a subtype of Inflammatory Bowel Disease (IBD) characterised by chronic inflammation of the gastrointestinal tract, primarily involving lymphocytes and plasma cells. Although the aetiopathogenesis is poorly understood, most hypotheses suggest that it results from an inappropriate immune response to luminal antigens, be they dietary antigens, commensal bacteria or other environmental factors. Although not yet proven, genetic predisposition, immune system dysfunction, and environmental influences might play significant roles.

Alimentary Lymphoma (AL) is a form of cancer arising from the lymphoid tissues within the gastrointestinal tract, and occurring in two main forms: small-cell and large-cell lymphoma. Small-cell lymphoma (now most commonly referred to as low-grade intestinal T-cell lymphoma, LGAL) is generally indolent, with a slower progression, whereas large-cell lymphoma is more aggressive and rapidly progressive. Overall, many more cats suffer from LGAL rather than large cell lymphoma. It has been suggested that chronic inflammation, such as that seen in LPE, might be a potential risk factor for the development of AL, particularly LGAL.

Clinical Presentation

Both LPE and LGAL can present with nonspecific gastrointestinal signs, making clinical differentiation challenging. Clinical signs may include chronic vomiting, diarrhoea, weight loss and altered appetite, all of which can vary in both severity and duration. Cats with both LPE and LGAL can experience intermittent signs that wax and wane over time, whilst cats with large-cell lymphoma can often exhibit a more rapid and severe signs.

The diarrhoea in both LPE and LGAL is typically of small bowel origin, although blood can be more present, and this is more likely in cases of large cell lymphoma. Weight loss is a hallmark of LPE and AL, thought to be the result of malabsorption and/or decreased caloric intake. When weight loss is seen in cases of LPE and LGAL, it is most often gradual and can be mild; in contrast, weight loss in cases of large-cell lymphoma can be severe, sometimes occurring rapidly. Appetite changes are also common, occasionally involving polyphagia but, more often, presenting as partial or complete anorexia. Besides the specific gastrointestinal signs, there may also be systemic signs such as lethargy and poor coat condition.

Diagnostic Approach

Given the overlapping clinical signs, a thorough diagnostic workup is essential, which typically includes a combination of history taking, physical examination, laboratory tests, imaging studies and taking tissue samples for histopathological evaluation.

- **History and Physical Examination.** A detailed history can provide clues about the chronicity and nature of the signs, whilst physical examination findings, such as abdominal pain, palpable masses, or thickened intestines, may raise suspicion of AL, especially if the mass effect is noted.

• Laboratory Tests.

- Recommended *blood tests* include haematology and serum biochemistry, which enables the clinician to assess overall health status and rule out other systemic diseases. Findings can be similar in cases of LPE and LGAL, where there might be mild anaemia or hypoproteinaemia, the latter of which is more less common than in dogs. Increased liver enzyme activity can sometimes be present, not least in cases with concurrent hepatic, biliary system or pancreatic involvement. In cases of large-cell lymphoma, more significant changes might be observed including marked anaemia, hypercalcaemia.
- *Faecal Analysis* is usually recommended as this enables parasitic infections to be ruled out.

• Diagnostic imaging.

- *Survey radiography* is often recommended as this is a better means of assessing overall organ size and shape, as well as identifying masses and obstructive lesions. Thus, it may complement ultrasonography, although many clinicians do not perform it for cost reasons.
- *Abdominal ultrasonography* can help to assess the structure of the gastrointestinal tract and identify abnormalities such as intestinal thickening, masses, or lymphadenopathy. In LPE, the ultrasound may reveal diffuse or segmental thickening of the intestinal walls with preserved wall layering. In AL, especially large-cell lymphoma, there may be more pronounced focal thickening, loss of normal layering, and the presence of discrete masses or enlarged lymph nodes. Where abnormalities are identified, ultrasound can help to direct cytological sampling.

• **Cytology.** Occasionally, a diagnosis can be made through cytological examination of fine-needle aspirate (FNA) samples taken either from lesions in the alimentary tract, liver or mediastinal lymph nodes. Malignant changes are usually seen with high-grade lymphoma, but definitive changes are not always evident in cases of LPE or LGAL.

• **Histopathology.** Tissue samples obtained through *endoscopy* or *exploratory coeliotomy*. Endoscopic biopsies allow for a minimally invasive approach, but may only obtain superficial mucosal samples, which can be insufficient for distinguishing between severe LPE and small-cell lymphoma. However, in many cases, the most noticeable changes may be beyond the reach of the endoscopy (e.g., mid to lower small intestine). Further, the full extent of abnormalities will be missed in cats whose LPE is part of the triaditis complex since other organs cannot be sampled. Full-thickness biopsies obtained at exploratory coeliotomy provide are larger and therefore superior, although this approach is more invasive and expensive. The typical histology pattern expected in LPE, would be infiltration of the intestinal mucosa with lymphocytes and plasma cells, along with varying degrees of villous atrophy, crypt distortion, and fibrosis; for AL, it is suggested that neoplastic lymphocytes will be present, which may infiltrate the mucosa, submucosa, and deeper layers of the intestinal wall. However, there is considerable overlap, not least between LPE and LGAL, meaning that these two conditions can often not be differentiated (Marsilio et al., 2023).

• **Advanced differentiation techniques.** Given the challenges with differentiating LPE from LGAL, more advanced diagnostic techniques have been suggested including immunohistochemistry (IHC) and polymerase chain reaction (PCR) for antigen receptor rearrangements (PARR). The aim of both such techniques is to distinguish between the presence of reactive lymphocytes in LPE and clonal, neoplastic lymphocytes in LGAL. Unfortunately, it may still not be possible to provide an accurate diagnosis in all cases since findings overlap between the two conditions (Marsilio et al., 2023).

Treatment Strategies

LPE is primarily managed through immunosuppression and dietary modifications, while AL typically requires chemotherapy. However, there is a reasonable degree of overlap in the treatment regimens used for LPE and AL, although some differences do exist, most notably for high-grade alimentary lymphoma cases.

Treatment of LPE

• **Dietary Management.** Diet plays a crucial role in the management of LPE, with hydrolysed protein diets most commonly recommended. The primary aim is to minimise the antigenic load on the gut, thereby reducing the inflammatory response. Initial dietary therapy is usually for a period of 2-4 weeks before other treatments are considered.

• Pharmacological Treatment:

- **Corticosteroids.** Prednisolone is the main pharmacological treatment for LPE, owing to its potent anti-inflammatory and immunosuppressive effects. It is typically administered at immunosuppressive doses initially (~2-4 mg/kg/day), with the goal of inducing remission. Once clinical improvement is achieved, the dose is gradually tapered to the lowest effective maintenance dose.
- **Immunosuppressive Drugs.** In cases where corticosteroids alone are ineffective additional immunosuppressive agents, with chlorambucil being the author's favourite choice. Owners should be instructed about the careful handling required for cytotoxic drugs, and haematology should be performed regularly during treatment to ensure that there are no problems with bone marrow suppression.
- **Probiotics and Antibiotics.** Both probiotics and antibiotics have been suggested for the treatment of LPE but there is little evidence of any meaningful benefit for either. Consequently, the author no longer recommends them.
- **Other therapy.** In some cases, other pharmacological treatments might be required depending on clinical presentation. These include antiemetics, appetite stimulants, and cobalamin supplementation (usually via the oral route). The latter is particularly important since hypocobalaminemia is a negative prognostic indicator in cases of feline chronic enteropathy.

Treatment of AL

- **Chemotherapy.** The primary treatment for AL is chemotherapy, with the choice of protocol depending on the type and severity of the lymphoma.
 - **LGAL.** Treatment typically involves oral chemotherapy using a combination of chlorambucil and prednisolone. Such a regimen is usually well-tolerated, with many cats achieving long-term remission. Given its indolent nature of small-cell lymphoma, some cats can live for several years with appropriate treatment, and outcome not dissimilar to that of therapy for LPE.
 - **Large-Cell Lymphoma.** Given its more aggressive nature, a more intensive chemotherapy protocol is usually required, often involving multiple drugs (e.g., CHOP protocol: Cyclophosphamide, Doxorubicin, Vincristine, and Prednisolone). While this treatment can induce remission in many cases, the prognosis is poorer than for small-cell lymphoma, with median survival times ranging from a few months to a year, depending on the response to therapy.
- **Other therapy.** Dietary management, cobalamin supplementation, anti-emetics and appetite stimulants are occasionally used as with LPE.

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CARING FOR DOGS AND CATS WITH OBESITY

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Introduction

Obesity is defined as an accumulation of excessive amounts of adipose tissue in the body; the disease predisposes to a variety of diseases including diabetes mellitus, osteoarthritis, and cardiorespiratory diseases. In most animals, obesity is the result of a simple imbalance between energy intake and energy expenditure. This lecture will discuss current thoughts on management of obesity in dogs and cats.

Causes of obesity

Obesity can arise secondary to a number of diseases including endocrinopathies (e.g. hypothyroidism and hyperadrenocorticism in dogs), drugs (e.g. polyphagia caused by glucocorticoids and anti-convulsant drugs), and rare genetic disorders (in humans), although the main reason for development of obesity is an

imbalance in the 'energy balance equation'. In this respect, either excessive dietary intake or inadequate energy utilisation can lead to a state of positive energy balance, leading to increased white adipose tissue deposition. Numerous factors may influence the relative ease with which weight is gained, and these include genetics, age, neuter status, amount of physical activity, calorific content of the diet.

Overview of treatment of obesity in dogs and cats

In humans, current therapeutic options for obesity include dietary management, exercise, psychological and behavioural modification, and surgery. Temporary weight loss, by liposuction, does not have an equivalent effect and does not affect metabolic risk. Liposuction removes only subcutaneous fat, which carries little metabolic risk, and energy intake is unaffected; therefore, body weight will rise again to achieve energy balance.

Bariatric surgery is the most successful method of weight loss in humans, and average weight loss is ~23%. Various approaches are described including gastric banding and the roux-en-y procedure. This success comes at a cost, since the complication rate is high (including peri-operative mortality, short-term and long-term consequences). For companion animals, it is not considered ethically justifiable to manage obesity through surgical means. For a short while, pharmaceuticals were available for weight management in dogs. However, these drugs have now been withdrawn from the market. For long-term success, it is ESSENTIAL to modify owner and animal behaviour. Unless steps are taken to change feeding habits and exercise patterns, weight regain will occur. This rebound effect is a well-known phenomenon of any weight loss program. Therefore, to achieve long-term success weight loss is only the start rather than the end of therapy. Conventional options for weight management include dietary therapy and behavioural modifications; such strategies are likely to remain for dogs and, given that no pharmaceutical agents have yet been approved for cats, this approach will remain the mainstay of therapy in this species.

Dietary management

The weight reduction protocol should always be tailored for the individual patient. Although complete starvation leads to rapid (~7%/week) weight loss, it has the disadvantages of causing excessive protein (and thus lean body mass) loss and requiring hospitalisation to monitor. Further, adverse effects on body system function have been reported (e.g. cardiovascular function is compromised). Therefore, it is preferable to use purpose-formulated diets, and most formulated rations are restricted in fat and calories, whilst being supplemented in protein and micronutrients. Protein supplementation is important since, although weight loss is not more rapid, the amount of lean tissue lost is minimised. Supplementation of micro-nutrients ensures that deficiency states do not arise. Other diet components that have been employed in weight management include L-carnitine supplementation (to maintain lean mass), conjugated linoleic acid, and use of high-fibre diets (to provide satiety; see below).

L-carnitine is an amino acid which is synthesised *de novo*, from lysine and methionine, in the presence of ascorbate. In one double-blind placebo-controlled study, orally-administered L-carnitine in a moist weight loss diet for cats lead to more rapid weight loss than placebo. Dietary supplementation of L-carnitine improves nitrogen retention and body composition in favour of increased lean mass and decreased fat mass. Incorporation of L-carnitine, at a level of 50-300 parts per million, in weight reduction diets has been shown to maintain lean tissue during weight loss. Possible mechanisms for this protective effect on lean tissue include enhancing fatty acid oxidation and energy availability for protein synthesis during times of need.

Conjugated linoleic acid (CLA) is a family of fatty acid isomers derived from linoleic acid. Studies in experimental animals have suggested an anti-adipogenic effect; the mechanism of action is not known, but possibilities include inhibition of stearoyl-CoA desaturase activity which limits synthesis of monounsaturated fatty acids for triglyceride synthesis, and suppression of elongation and desaturation of fatty acids into long-chain fatty acids. Currently, date are conflicting data on the benefit of CLA as an anti-obesity agent in humans and cats, with the most recent data suggesting lack of a significant effect. Therefore, more information is required before its use can be recommended, although a recent meta-analysis of human data suggested a modest beneficial effect in favour of weight loss.

A major hurdle to conventional weight loss programs is the fact that energy restriction causes hunger, leading to increased begging and scavenging activity. This puts increased strain on the owner-animal bond, causing owner non-compliance or complete withdrawal from the program. Therefore, developing strategies to improve satiety would greatly assist in case management. The results of many human studies have shown that absorption of macronutrients is lower following consumption of high-protein foods than after consumption of foods with a high carbohydrate or fat content. The amino acids from the digestion

of proteins are absorbed slowly and the main path of their metabolism is gluconeogenesis. Therefore, proteins are sources of glucose that induce little insulin secretion and delay the appearance of hypoglycaemia (which contributes to the feeling of hunger). The satiety effect of proteins is variable, because speed of digestion varies amongst different proteins, and different amino acids induce the secretion of insulin to varying degrees. Dietary fibre may also increase satiety, due to gastric distension which causes cholecystokinin release and a subsequent slowing of gastric emptying. Under certain conditions in humans, dietary fibre has been shown to exert a satiety effect, although some studies have failed to detect significant reduction in appetite. There are similar discrepancies in canine studies with some, but not all studies suggesting effects on satiety. Apparent inconsistencies are likely the result of differing investigative methods and of the dose and type of fibre used. In recent studies in colony dogs, three different diets (HPHF, high protein [103g/1000Kcal] high fibre [60g/1000Kcal]; HP, high protein [104g/1000Kcal] moderate fibre [35g/1000Kcal]; HF, moderate protein [86g/1000Kcal] high fibre [87g/1000Kcal],) designed for weight loss were assessed for their satiety effect. Voluntary food intake was measured in five sequential crossover studies, and palatability was assessed with taste tests. Short- (food offered for 15min every hour for 4h) and medium-term (food offered 3h after the first meal) satiety was best for the HPHF diet. Voluntary food intake at the second meal (fed 3h after a restricted meal of 25% of daily metabolic energy requirements) was significantly lower than the first meal for the HPHF diet, but not the HP or HF diets. The HPHF and HP diets had equivalent palatability, and both were more palatable than the HF diet. These studies suggest that diets supplemented in both protein and fibre have the greatest satiating effect, and may improve compliance with conventional weight loss programs.

Lifestyle management

Increasing physical activity is a useful adjunct to dietary therapy during weight management; studies in humans suggest that increasing activity promotes fat loss, whilst preserving lean tissue during weight loss. The exact program must be tailored to the individual, and take account of any concurrent medical concerns. Suitable exercise strategies in dogs include lead walking, swimming, hydrotherapy, and treadmills. It is possible to encourage exercise in cats by increasing play activity, using cat toys (e.g. fishing rod toys), motorised units, and feeding toys. Cats can also be encouraged to 'work' for their food by moving the food bowl between rooms prior to feeding, or by the use of feeding toys. Activity monitors (accelerometers, pedometers) have recently been validated for dogs, and may help to provide a more objective assessment of activity during weight loss programs in the future.

Monitoring weight loss

In addition to the above strategies, it is essential that the whole weight reduction regime be closely supervised. This is labour-intensive, requires some degree of expertise and training in owner counselling, and often requires a dedicated member of staff. Nevertheless, in the author's opinion, correct monitoring is the single most important component to the weight loss strategy. A recent study has demonstrated that weight loss is more successful if an organised strategy is followed with regular weigh-in sessions. It is essential to continue to monitor body weight after ideal weight has been achieved to ensure that weight that was lost is not regained; as with humans, a rebound effect has been demonstrated after weight loss in dogs. This has been seen in ~50% of dogs that successfully lose weight.

Managing consequences of obesity

There are many comorbidities and other health consequences that can develop in cases of obesity and these should be managed alongside the obesity (e.g., anti-inflammatories and physiotherapy in dogs with obesity). A complete discussion of all recommendations is beyond the scope of this talk.

Review of conventional weight loss programmes

Conventional weight loss regimes, involving dietary caloric energy restriction, are highly successful in obese colony dogs. Rates of weight loss of 1.3-2.6%/week have been achieved with caloric allocations of 50-87% maintenance energy requirement (MER). However, weight loss in client owned dogs is slower (average 0.85% body weight/week), and requires a greater degree of energy restriction i.e. mean 52% of MER at target weight. Mean energy intake during a weight loss regime is 32 Kcal/kg TW has been reported in pet cats with naturally occurring obesity. With this degree of restriction, the average rate of weight loss is 0.8% body weight/week.¹⁰

The most important factors that influence response include breed, gender and neuter status. Previous work with colony dogs has demonstrated breed differences in the level of energy restriction required to achieve the same rate of weight loss, with Labrador retrievers requiring a greater level of restriction than beagles. Age, sex, neuter status and activity level have also been shown to be of importance. The main factor that affects the rate of weight loss is the level of caloric energy restriction. Nevertheless, whilst the level of protein does not appear to affect rate of weight loss, the proportion of lean tissue loss in dogs is lower on a high-protein diet, compared with a diet of moderate protein content. As mentioned above, the use of formulated weight loss diets that are supplemented in both protein and fibre improve outcomes of weight loss in dogs.

Preventing weight regain

In humans, long-term success of weight management strategies is disappointing, with some studies suggesting that some participants on diet-based weight loss strategies regaining more weight than they had originally lost. Whilst the reasons for this 'regain' are still unclear, the most likely explanation is that, when obese humans are returned to a lean state, their resting metabolic rate is lower. Studies in dogs have demonstrated a similar tendency for weight regain, with maintenance energy requirements decreasing significantly after weight loss. A recent study has examined long-term follow-up in obese pet dogs that had successfully reached target weight: 42% of dogs maintained weight, 9% lost further weight, and 48% regained weight. Dogs fed a purpose-formulated weight management diet, during the weight maintenance phase, regained less weight than those switched onto a standard maintenance diet. In a similar study in cats, age was the principle factor associated with weight regain, with younger cats (<9y age) more likely to rebound.

Summary

Successful weight loss in dogs and cats requires dedication and commitment. Conventional weight loss strategies involving diet and exercise can be highly successful in both dogs and cats, whilst pharmaceutical agents provide another means to achieve target weight. Successful weight loss involves not only achieving an ideal body weight, but maintaining it subsequently. In the author's opinion, preventing the known and predictable rebound effect seen with any weight loss strategy, requires changes in both owner and pet behaviour, and is the key factor in true success.

PREVENTION OF OBESITY

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Introduction

Although weight loss using a therapeutic diet can be very successful, it can be very challenging for owners and rebound can often occur. Therefore, it makes more sense to prevent inappropriate weight gain from developing in the first place. This can be more challenging than it sounds, not least because the problem develops insidiously, such that it is often missed until it is too late. Prevention of obesity is a lifelong problem, requiring interventions right from the early growth phase through to the senior adult years. It requires the veterinary professional to pay constant attention to maintaining a neutral energy balance (in terms of dietary energy intake relative to energy expenditure) whilst, at the same time, confirming this through regular monitoring of body weight and condition. Whilst the main tools for obesity prevention are similar, the strategy adopted is different at different stages, which are considered separately below.

Tools for monitoring weight and body condition

Body weight. Arguably, taking regular bodyweight measurements are the most important monitoring strategy for preventing inappropriate weight gain and, provided that the same set of calibrated electronic scales is used, it is much more precise and objective than other approaches (e.g. body condition scoring, using a tape measure). Even small deviations from optimal weight can be spotted quickly enabling early intervention. Items such as clothing or harnesses should be removed, if possible, and the animal should be positioned so that they are standing with all four feet on the scales. The animal should also be as still as possible during the weighing process, and the result should

be immediately recorded in the animal's case notes (to two decimal places for all cats and small breed dogs, and one decimal place for medium to giant breed dogs). If not doing so already, veterinary clinics should instigate a policy of weighing all animals at every single visit since, over time, an individualized historical record of body weight will be available for all registered patients.

Body condition. Body condition scoring (BCS) is the most widely accepted clinical method of assessing body composition because it is very quick and easy to perform, whilst at the same time being reliable. It is important that the veterinary professional conduct the assessment, because owners under-estimate the condition of their pet. Various BCS systems have been described, but the WSAVA Global Nutrition Committee recently recommended that the 9-point system be universally adopted. As well as determining current weight status (underweight, optimal weight, overweight), knowledge of the current BCS can be used to estimate optimal weight, should it deviate from normal.

Dietary strategies for obesity prevention

To best frame discussions regarding diet and food intake, a nutritional assessment is recommended, ideally in accordance with the recent recommendations of the WSAVA Global Nutrition Panel. Such an assessment will enable the individual needs of the animal to be considered when setting the prevention strategy to adopt. Dietary strategies that can help in preventing inappropriate weight gain include determining the most appropriate main meal to feed, adopting a responsible plan for providing treats and extra food, accurate measurement of food portions, managing feeding activity within and outside the home, and adapting food intake as nutritional requirements change.

Periodically, it is advisable to reappraise the animal's nutrition (again using the WSAVA Nutritional assessment), since actual feeding regimes can 'drift' gradually over time. Such a review helps to refocus priorities, adjust the strategy if there have been changes in circumstance, and ensure continued commitment from the owner in preventing weight gain.

Main meal feeding. All dogs and cats should be fed a diet that is nutritionally complete and balanced and, preferably, tailored to the correct life stage, be it for growth, the early adult period, or for the senior years. If the current diet is appropriate, and both owner and pet are happy with it, then there is no need to change. Instead, attention should be focused on ensuring that the correct amount is being fed for requirements, and adjusted as needs change. Care should be focused when changing to a new diet since energy content may be different and portion sizes may differ.

For dogs and cats that show marked food seeking behaviour or excessive begging, a change of food type can be considered. The same characteristics as those used in a purpose-formulated weight loss food will also help for weight maintenance. For example, food can be supplemented with protein and fibre, are known to minimise signs of hunger and reduce voluntary food intake in both dogs and cats. Increasing food volume can also reduce voluntary food intake, for instance by adding water (or using a wet food) or expanding a kibbled food with air. Finally, the shape of a kibbled diet can also be altered, which can force a dog or cat to chew food more, thereby slowing intake.

Accurate portion size measurement. It is critical that there is both precision and accuracy when measuring food portions. This is most important for dry food because it is so energy dense, and small errors can lead to large differences in actual energy intake. Whilst measuring cups may be the simplest method of measuring dry food, they are unreliable and can lead to overfeeding especially for small portion sizes (such as those fed to small dogs and cats). For those animals known to be at risk of inappropriate weight gain or over-eating, more accurate measurements are critical, and a different method is strongly recommended, if possible, using digital gram scales. In practice, it takes very little additional time in measurement and gives owner and veterinary professional reassurance that correct portions are being delivered day in and day out. Other methods that are currently in development include 'smart bowls' and computer-controlled food hoppers, which include gram scales within them, and automatically measure out the correct portion with the minimum of effort.

Responsible feeding of treats and extra food. It is critical to control the feeding of extra food, such as tit-bits, table scraps, treats, and food scavenging. Most owners frequently are not aware of the contribution that such food sources can make to the daily ration. During the initial nutrition review, time should be taken to obtain a detailed understanding of the extra food the animal receives, and, to make this as accurate as possible, it is advisable to question multiple family members. In theory, it is preferable to avoid feeding any additional food, since there is a danger that it may make an otherwise balanced main meal unbalanced. Most owners will not accept this because rewarding their pet is such an instinctive behaviour. The solution is to develop a formal program of treating, which either makes use of existing food or, instead, permits the owner to use a controlled number of approved treats, to a maximum of 10% of MER, so as to ensure that the diet remains in balance overall. The energy content of such treats should be calculated and the amount of main meal fed should be reduced accordingly.

Ideally, human food and table scraps should still not be fed. To minimize temptation, it is best to ensure that pets are not in the kitchen area where food is being prepared, and not allowed access to the dining area during mealtimes. Food preparation areas and dinner tables should be cleared before the pets are allowed back in, whilst trash bins and food stores should be properly secured. Finally, care should be taken when dogs are taken for walks. If scavenging at this time is a problem, it may be necessary for the dog to wear a muzzle or not be let off the lead.

Methods of feeding. Again, the initial nutritional assessment should include a discussion of how the owner feeds their pet(s). Most commonly, dogs will be given 1-2 main meals each day, and meal feeding is also common for cats, though some owners will leave food out all day. The latter should be discouraged, not least in multi-animal environments and with cats that are unable to regulate their daily food intake (see below).

It is worth considering the use of puzzle feeders as part of the overall feeding strategy. These are an excellent method of slowing food intake, thereby extending the feeding period. Not only does it help to minimize over-eating (since there is more time for gastrointestinal hormones that lead to satiation to be released and affect the hunger centre in the brain), but it also is more enjoyable for the pet.

In multi-pet households, it is critical to ensure that each has their own tailored feeding plan and only receives its own food. Various strategies can be used. It can be a particular challenge when managing multi-cat households with one grazer cat (that is in ideal weight) and an overweight cat that does not regulate intake. Food should never just be left out and, instead, pets in the same household must be fed separately. For example, a 'grazer', that can self-regulate, can be allowed long periods to be fed whilst the cat that tends to over-eat is given food by puzzle feeder. Alternatively, food could be left out for the grazer in a location that the overweight cat cannot access (on a high surface, within a small 'creep' area (e.g. box or cupboard with small hole through which the overweight cannot pass), using a smart bowl to allow free access for the grazer, whilst meal feeding or using a puzzle feeder in the overweight cat.

If desired, food can be given in two, or more, meals per day, preferably providing more food at times when the owner is with the pet (since this is when begging is most likely to occur). However, use of an interactive feeding device is preferred (e.g. puzzle feeding toy, or modified feeding bowl). These devices have the effect of slowing food intake, thereby improving satiety.

Physical activity as a means of obesity prevention

In addition to controlling food intake, promoting energy expenditure is a valuable means of helping to prevent inappropriate weight gain. The main approach in both cats and dogs is to increase physical activity, which has a modest but significant effect on energy expenditure in most animals. Indeed, in a recent canine study, each 1000 steps of walking increased energy expenditure by 1kcal per kg^{0.75} of body weight. In addition to burning calories, physical activity can improve and maintain cardiovascular and musculoskeletal fitness, and improve the owner-pet bond. Regular daily sessions are recommended for both species, although the approach varies (Table 2). For dogs, at least one daily walk of 30 minutes is recommended. When play sessions are used in cats, short periods are sufficient activity, typically 1-2 minutes at a time 2 or more times per day.

The recommended exercise should take account of any concurrent medical concerns, and also be tailored to the capabilities of the pet. For example, if a dog has an orthopaedic disease (such as osteoarthritis), controlled forms or exercise, such as leash walking are preferable to vigorous activity (off-leash running and playing with a ball). Alternatively, hydrotherapy could be used, dependent on cost and availability in the local region. Finally, the agreed plan should also take account on the preferences and capabilities of the owner, in terms of the time available, timing, and type of exercise.

A final method that can help to promote movement in both cats and dogs is the use of puzzle feeders, hollow toys in which you place a small amount of kibbled food. The cat or dog must then play with the toy to remove the food. Most animals rapidly learn how to use these, and will play for extended periods, often well after the toy is empty.

Recommended monitoring strategy according to life stage

Early life prevention

The importance of ensuring that dogs and cats grow at an appropriate rate during their early years cannot be over-emphasised since this is the foundation to a healthy weight for the whole of the adult years. Epidemiological studies in humans have demonstrated that inappropriate growth is a predictor of obesity later in life. Indeed, a rapid rate of growth, catch-up growth (where an underweight for age child grows faster than average), and high early-life body mass index are all independently associated with the risk of obesity at 7 years of age. Recent studies have also demonstrated rapid growth to be a risk factor for later-life obesity in both cats and dogs.

Given such an importance to the growth period, regular monitoring of body weight throughout the growth phase is essential. Indeed, growth standards are now widely used in human paediatrics, with the most adopted being those endorsed by the World Health Organization (WHO). Such growth standards enable appropriately-trained health workers to monitor individual growth in children and verify that it is appropriate compared with a healthy reference population. Regular weight monitoring can then be performed, and guidance can be given should the child's growth deviate from optimal. Recently, evidence-based growth charts have been developed for puppies and kittens, which can be accessed online (<https://www.waltham.com/resources/puppy-growth-charts>), with detailed instructions on how to use them. These enable veterinarians to monitor the growth of individual cats and dogs precisely and, where rapid growth is identified, adjustments to food intake can be implemented. Such charts also enable an optimal adult weight to be determined ("healthy adult weight"), which can be used as a guide for the weight the dog or cat should maintain during their adult life (see below).

Monitoring strategy for adult animals

Inappropriate weight gain is an insidious phenomenon, and the early adult years are a particular period of risk. Indeed, the prevalence of overweight animals steadily increases in this period to a peak in mid-adult life. Therefore, regular and proactive monitoring is critical as a means of identifying at risk animals, and making early corrections to prevent animals becoming overweight. The author recommends recording bodyweight 2-4 times per year and BCS 1-2 times per year. Interventions should be considered when there has been a weight gain of 5% (from the animal's healthy adult weight, set at the end of the growth phase; see above).

Monitoring strategy for the post-neutering period

Neutering is a risk factor for inappropriate weight gain in both dogs and cats, although its influence varies amongst individuals. Consequently, close monitoring of body weight and condition is essential during the post-neutering period. The author recommends recording bodyweight at 2, 4, 8 and 12 weeks after neutering, with adjustments to food intake made if unexpected weight gain is seen.

Monitoring strategy for senior animals

Weight checks should be continued into the senior life stage. Not uncommonly activity can decline at this stage, not least because of concurrent diseases such as osteoarthritis have developed. Any increases in weight should prompt a nutritional and lifestyle review, with adjustments made as required. Of course, old age is also a time when chronic diseases are common, many of which can lead to loss of body weight, and especially body mass. Thus, the clinician should be alert to this possibility, and any unexpected decline in body weight should be investigated proactively to elucidate the cause. It is also common for animals to gain adipose tissue whilst at the same time losing muscle mass. For this reason, regular assessment of body condition is also vital, and this should include a subjective assessment of muscle condition. For most senior animals, 6-monthly checks will be sufficient. However, more regular monitoring should be considered for animals known to have a chronic disease that causes loss of body weight e.g. chronic kidney disease, hyperthyroidism etc.

Conclusion

The process of inappropriate weight gain is insidious, and many animals are at risk of becoming overweight or obese. Prevalence reaches its peak during the middle-aged years and, once obesity is established, it can be immensely challenging to treat. Few animals start a weight loss program with many of those that doing either failing to reach target weight or rebounding afterwards. As a result, veterinary professionals should focus on prevention of obesity, rather than attempting to manage it once it has developed. Veterinary practices should consider establishing a formal program of monitoring body weight and regularly assessing BCS, with strategies tailored to the life stage. As soon as there is evidence of inappropriate weight gain, there should be early intervention with corrective measures. Finally, if an obese animal does successfully lose weight, veterinarians should closely monitor post-weight-loss period, with regular follow-up weight checks to ensure that bodyweight remains stable. Continuing to feed the therapeutic weight loss diet during the maintenance phase can help to prevent rebound from occurring.

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BSc BVetMed DSAM DipECVIM-CA FRCVS, CVS and European Specialist in Small Animal Internal Medicine

EERVC 2024 Lectures

1. Solving the Puzzle; how problem-orientated medicine can enhance your diagnostic success
2. I Think I have a Drink Problem; getting to the bottom of patients with PUPD
3. The Blood Sugar Rollercoaster; strategies for managing challenging diabetic patients
4. "Ions in Action"; Navigating the maze of abnormal electrolytes in cats and dogs

Rob qualified from the RVC in 1996 having previously undertaken a Physiology and Pharmacology BSc at King's College London. After four years in mixed general practice, he undertook his residency training in small animal internal medicine at the University of Cambridge where he obtained his CertSAM, DSAM and DipECVIM-CA and he became a recognised Specialist in 2005.

Rob joined Dick White Referrals in 2003, where he set up and ran both the internal medicine and medical oncology services from 2004 to 2016. He then became a Shared Venture Partner and Clinical Director for five years before leaving DWR in December 2021. In addition to his clinical work, Rob has an on-going research project looking to develop gene therapy as a treatment for diabetic dogs. Rob has been actively involved in teaching at the University of Nottingham since 2008 and has been a Professor of Small Animal Medicine at this institution since 2020 where he currently delivers the urogenital and infectious disease modules of the University's certificate/AVP course. Rob also enjoys delivering CPD and has spoken regularly throughout the UK and Europe, particularly in the fields of small animal endocrinology, gastroenterology, problem-orientated medicine and medical oncology. Away from work, Rob is married and has three teenage children, two dogs and a rabbit! He relaxes by being a (very slow!) 10K and half marathon runner and by playing bass in a local band.

Solving the Puzzle; How problem- orientated medicine can enhance your diagnostic success

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1

Plan

- Welcome!
- Let's get thinking – a clinical conundrum!
- Problem-orientated medicine – how can it help?
- Looking ahead - what to expect



2

Bella, a 6 year old FN Samoyed Cross

- Presented with a six-day history of progressively worsening diarrhoea
- Initially was eating but started to vomit three days ago and hasn't wanted to eat since
- Owner doesn't think she has drunk much in the past 24 hours



Bella, a 6 year old FN Samoyed Cross

- Up to date with vaccinations and no travel history
- On examination, HR 76, RR 28, T 38.5°C, mm pale pink but tacky/dry
- Cardio-pulmonary auscultation NAD
- Abdominal palpation – appears uncomfortable but no obvious abnormality



3

4

Bella, a 6 year old FN Samoyed Cross

- What further questions would you like to ask the owners?
- What are your initial thoughts?



From her history and presenting signs, what would you do next?

- Advise 24 hours of starvation then bland food?
- Dispense symptomatic medication?
 - If so, what?
- Perform serum biochemistry and CBC?
 - What is your rationale for doing so?
- Obtain radiographs?
- Admit for IVFT?
 - If so, what fluid type?
- What would you do and why?



5

6

Actions!



- Admit for IVFT
- **Create a problem list!**
- Diarrhoea
- Vomiting
- Dehydration
- Inappetance

7

Problem list and possible differential diagnoses



- **Diarrhoea +++**
- Need to characterise further - ? SI/LI
- Consider DAMNIT-V to help consider possible differential diagnoses
- Dietary - hypersensitivity, intolerance, diet change, poisoning
- Anatomic - foreign body, intussusception
- Metabolic - hypoadrenocorticism, acute pancreatitis
- Neoplasia - SI or LI, hepatic
- Infectious - parvo, coronavirus, adenovirus, salmonella, campylobacter, E.coli, parasites
- Toxic - food, other sources
- Vascular - mesenteric thrombosis

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Problem list and possible differential diagnoses



- Vomiting
- Could be considered in a different way - Gastric/Intestinal/Metabolic/CNS
- Dietary - indiscretion
- Anatomical - intestinal volvulus, intussusception, GDV
- Metabolic - hypoadrenocorticism, hypokalaemia, hypercalcæmia, diabetic ketoacidosis, acute pancreatitis, acute hepatitis, acute urogenital tract disease, peritonitis, (pyometra)
- Neoplasia - GI, intestinal, pancreatic, hepatic, peritoneal, UT, CNS
- Infections - CPV, CDV, FPL, FELV, FIV, coronavirus, salmonella, campylobacter, clostridia, E.coli, protozoa, ascarids, hookworm, whipworm
- Toxic - heavy metals, digoxin, NSAIDS, erythromycin

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Problem list and possible differential diagnoses



- **Diarrhoea +++**
- **Vomiting**
- Inappetance
- Dehydration
- ? Consequences of cause of GI upset

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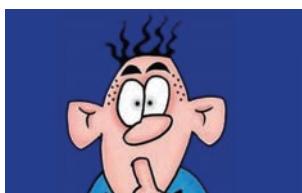
Differential diagnosis list – how would you prioritise them?



- 1.Acute gastroenteritis
- 2.Intestinal obstruction
- 3.Hypoadrenocorticism
- 4.Neoplasia
- 5.Renal Failure

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So what would you do next?



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Serum biochemistry

• Total protein	53	g/l	55.0 - 75.0
• Urea	14.2	mmol/l	2.5 - 6.7
• Creatinine	80	μmol/l	20 - 150
• ALT	46	IU/L	5.0 - 60.0
• ALP	278	IU/L	<130
• Sodium	108	mmol/l	135 - 155
• Potassium	6.0	mmol/l	3.6 - 5.6
• Chloride	77	mmol/l	100 - 116
• Phosphorus	1.4	mmol/l	0.8 - 1.6
• Calcium	2.2	mmol/l	2.40 - 2.90
• cPLI			< 200
• Na:K ratio			18:1



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Haematology

• PCV	53	%	37 - 55
• WBC	17.4	$\times 10^9/l$	6.0 - 17.0
• Neuts	10.8	$\times 10^9/l$	3.0 - 11.5
• Lymphs	5.2	$\times 10^9/l$	1.0 - 4.8
• Monos	0.7	$\times 10^9/l$	< 0.8
• Eos	0.6	$\times 10^9/l$	0.1 - 1.3



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Summary of significant findings



- Low Na^+ and Cl^-
- High K^+
- Low Na:K ratio
- Elevated urea
- Lymphocytosis in a stressed animal

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So has your differential list priority changed?



- 1.Hypoadrenocorticism
- 2.Acute gastroenteritis
- 3.Intestinal obstruction
- 4.Neoplasia
- 5.Renal Failure

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So what would you do next?



- A. Urinalysis
- B. ACTH stimulation test
- C. Abdominal ultrasound
- D. Abdominal radiography
- E. Anything else?

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Further tests



- Based on our problem-orientated medicine approach, we have a young-middle aged female dog with a history, clinical signs and clinical examination findings potentially compatible with hypoadrenocorticism, hyponatraemia, hyperkalaemia and a lymphocytosis
- Main DDx is therefore hypoadrenocorticism
- ACTH stimulation test is the most logical next step

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Further tests

- ACTH Stimulation:
 - Pre-ACTH 145 nmol/l (ref 15 - 110)
 - Post-ACTH 437 nmol/l (ref 220 - 550)



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Further tests

- ACTH Stimulation:
 - Pre-ACTH 145 nmol/l (ref 15 - 110)
 - Post-ACTH 437 nmol/l (ref 220 - 550)
- What to do now????



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Summary of significant findings



- Low Na⁺ and Cl⁻
- High K⁺
- Low Na:K ratio
- Elevated urea
- Lymphocytosis in a stressed animal
- Normal ACTH stimulation test result

Summary of significant findings



- Low Na⁺ and Cl⁻
- High K⁺
- Low Na:K ratio
- Elevated urea
- Lymphocytosis in a stressed animal
- Normal ACTH stimulation test result
- We need to re-consider our differential diagnosis list!

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Differential diagnosis list for hyponatraemia and hyperkalaemia



- Hypoadrenocorticism
- Severe enteritis (consider bacterial enteritis)
- Trichuris vulpis
- Third space fluid loss

So what would you do next?

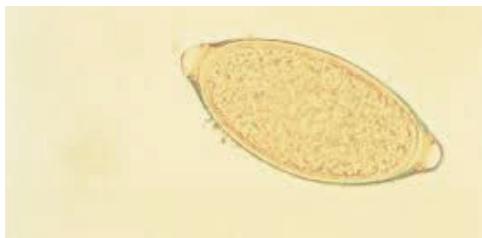
- A.Thoracic &/or abdominal radiography
- B.Abdominal ultrasound
- C.Faecal parasitology
- D.Faecal culture
- E.Commence antibiosis



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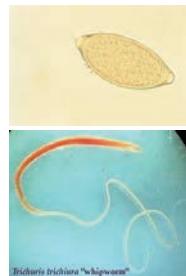
Faecal parasitology



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Pseudohypoadrenocorticism

- Severe diarrhoea
- Whipworm - *Trichuris*
- Salmonella
- Loss of Na⁺ (& Cl⁻) into gut
- Also loss of HCO₃⁻ into gut -> acidosis
- Acidosis -> hyperkalaemia



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Bella - outcome

- IVFT commenced (dehydration corrected within 24 hours) with 0.9% NaCl
- Anthelmintics administered
- Faecal culture run – commensals only identified
- Complete recovery



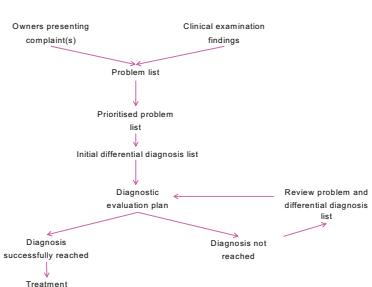
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Problem-orientated Medicine

- Advantages:
- Produces a unique, patient-centred approach
- Creates a logical diagnostic pathway based on the clinical history, clinical examination findings and clinicopathological data
- Creates a plan as to how to proceed if you obtain results you weren't expecting!
- Enables you to know what to do when you don't know what to do!



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Interpreting test results

- Sensitivity and Specificity
- Sensitivity reflects the number of patients who have a disease and who test positive
- We can therefore use this to help us rule OUT patients who don't have the disease, as a negative result is more likely to be truly negative the more sensitive the test is
- Sensitivity rule out = "snout"
- Therefore tests with high sensitivity are useful as screening tests



30

Interpreting test results

- Sensitivity and Specificity
- Specificity reflects the number of patients who do not have a disease and who test negative
- We can therefore use this to help us rule IN patients who do have the disease, as a positive result is more likely to be truly positive the more specific the test is
- Specificity rule in = "spin"
- Therefore tests with high specificity are useful as confirmatory diagnostic tests

31

Positive and negative predictive value

- Sensitivity and Specificity relate to a population in which we do not know if they have disease or not
- PPV and NPV apply to the population in which we know they do or do not have the disease
- PPV asks, if a test result is positive, what is the probability that the patient actually has the disease
- NPV asks, if a test result is negative, what is the probability that the patient does not have the disease

32

Conclusions

- By adopting a problem-orientated approach, we were able to develop a personalised investigation plan appropriate for this case
- Testing was focused and rational – we had a plan of what to do when we didn't know what to do!
- Reflection and re-consideration of problem list and consequential DDx is VITAL!
- Accurate diagnosis reached quickly, enabling accurate and rapid treatment

33

Think I have a
Drink Problem;
getting to the
bottom of patients
with PUPD

Rob Foale BSc BVetMed DSAM
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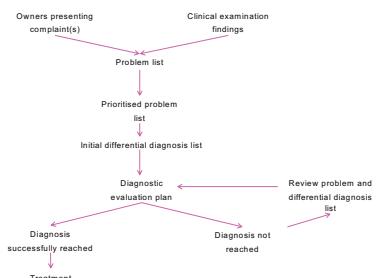
1

Plan

- Welcome!
- Let's get thinking – a clinical conundrum!
- Problem-orientated medicine – how can it help?
- Looking ahead - what to expect



2



3

Case study

- "Bobby", a 9-month old ME Shih Tzu
- BIOP since 8-weeks of age and had always drunk more than they were expecting according to the owner
- 6.7kg
- On presentation, his daily water intake had been measured at 1.9L a day
- Owners were having great difficulty managing his urination
- Vaccination and worming history up to date
- No travel history
- RVS had given a 7-day course of oral trimethoprim-sulphur prior to referral but no improvement seen in his water intake



4

Bobby clinical examination

- 6.7kg
- BAR and in good condition
- Cardiopulmonary auscultation NAD
- Abdominal palpation NAD
- MM and peripheral pulses normal
- Rectal temperature normal
- Neurological examination normal
- Drank water in the consult and emptied the bowl...



5

Bobby, a 9-month old ME Shih Tzu

- What further questions would you like to ask the owners?
- What are your initial thoughts?



6

Actions!

- Create a problem list!
- Polydipsia – 283ml/kg!
- Polyuria
- Nocturia



7

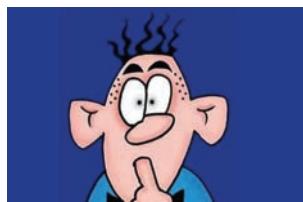
Differential diagnosis for PU/PD

- Endocrine disease
 - HAC
 - HyAC
 - HyperT4
 - HyperPTH
 - DM
 - DI
- Non-endocrine disease
 - Renal failure
 - Neoplasia
 - 1^o Hypercalcemia
 - Hyperviscosity
 - 1^o PCV
 - 2^o PCV
 - Multiple myeloma
 - Renal infections
 - Pyometra
 - Hepatic disease
 - Renal glomerular or tubular Dz
 - PLN
 - Fanconi syndrome
 - Renal tubular acidosis
 - Primary polydipsia



8

So what would you do next?



9

Differential diagnosis for Bobby

- Endocrine disease
 - HAC
 - HyAC
 - HyperT4
 - HyperPTH
 - DM
 - DI
- Non-endocrine disease
 - Renal failure
 - Neoplasia
 - 1^o Hypercalcemia
 - Hyperviscosity
 - 1^o PCV
 - 2^o PCV
 - Multiple myeloma
 - Renal infections
 - Pyometra
 - Hepatic disease
 - Renal glomerular or tubular Dz
 - PLN
 - Fanconi syndrome
 - Renal tubular acidosis
 - Primary polydipsia



10

Urinalysis

• Appearance:	Clear, very pale yellow	• Red cells	Negative
• SG	1.003	• White cells	Negative
• ph	6.5 (ref. 5.5 -7.5)	• Epithelial cells	Negative
• Protein	Negative	Occasional sperm	
• Nitrite	Negative	• Casts	Negative
• Leucocyte esterase	Negative	• Crystals	Negative
• Blood/Hb	Negative	• Bacteria	Negative
• Glucose	Negative	• Urine	Culture negative
• Ketones	Negative		
• Bilirubin	Negative		
• Urobilinogen	Negative		



11

Differential diagnosis for Bobby

- Endocrine disease
 - HAC
 - HyAC
 - HyperT4
 - HyperPTH
 - DM
 - DI
- Non-endocrine disease
 - Renal failure
 - Neoplasia
 - 1^o Hypercalcemia
 - Hyperviscosity
 - 1^o PCV
 - 2^o PCV
 - Multiple myeloma
 - Renal infections
 - Pyometra
 - Hepatic disease
 - Renal glomerular or tubular Dz
 - PLN
 - Fanconi syndrome
 - Renal tubular acidosis
 - Primary polydipsia



12

Serum biochemistry

Total protein	69g/l	(54 - 77)	Glucose	6.7mmol/l	(3.3 - 5.8)
Albumin	36g/l	(25 - 40)	ALT	65 IU/L	(13 - 88)
Globulin	33g/l	(23 - 45)	AST	37 IU/L	(13 - 60)
Urea	13.8mmol/l	(2.5 - 7.4)	ALP	50 IU/L	(14 - 105)
Creatinine	108umol/l	(40 - 145)	GGT	1 IU/L	(0 - 10)
Potassium	5.4mmol/l	(3.4 - 5.6)	Bilirubin	5 umol/l	(0 - 16)
Sodium	155mmol/l	(139 -154)	Triglyceride	1.0 mmol/l	(0.56 -1.14)
Chloride	111mmol/l	(105-122)	CK	241u/l	(0 - 190)
Calcium	3.0mmol/l	(2.1 - 2.8)	CTLI	21.3 ug/l	(5.0 - 40.0)
Magnesium	0.93mmol/l	(0.62-0.90)			
Phosphate	2.5mmol/l	(0.60 - 1.40)			

13

Extra testing

Pre-prandial bile acids	2.2 umol/l	(0 - 10)
Post-prandial bile acids	15.4 umol/l	(0 - 15)

14

Differential diagnosis for Bobby

Endocrine disease	Non-endocrine disease
HAC HyAC HyperT4 HyperPTH DM DI	Renal failure Neoplasia 1 ^o Hypercalcaemia Hyperviscosity <ul style="list-style-type: none"> • 1^o PCV • 2^o PCV • Multiple myeloma Renal infections <ul style="list-style-type: none"> • Pyometra • Hepatic disease • Renal glomerular or tubular Dz <ul style="list-style-type: none"> • PLN • Fanconi syndrome • Renal tubular acidosis • Primary polydipsia

15

Complete Blood Count

Haemoglobin	18.4 g/dl	(12.0 - 18.0)
RBC	8.82 x10 ¹² /l	(5.5 - 8.5)
HCT	0.56 l/l	(0.37 - 0.55)
MCV	73.0 fl	(60.0 - 77.0)
MCHC	34.3g/dl	(30.0 - 38.0)
MCH	25.0 pg	(19.5 - 25.5)
White cell count	12.31 x10 ⁹ /l	(6.0 - 15.0)
Neutrophils	9.7.01 x10 ⁹ /l	(3.0 - 11.5)
Lymphocytes	1.8 x10 ⁹ /l	(1.0 - 2.5)
Platelets	271 x10 ⁹ /l	(200 - 500)
Platelet morphology: Platelets are consistent with analyser count. No clumping seen		

16

Differential diagnosis for Bobby

Endocrine disease	Non-endocrine disease
HAC HyAC HyperT4 HyperPTH DM DI	Renal failure Neoplasia 1 ^o Hypercalcaemia Hyperviscosity <ul style="list-style-type: none"> • 1^o PCV • 2^o PCV • Multiple myeloma Renal infections <ul style="list-style-type: none"> • Pyometra • Hepatic disease • Renal glomerular or tubular Dz <ul style="list-style-type: none"> • PLN • Fanconi syndrome • Renal tubular acidosis • Primary polydipsia

17

Diagnostic imaging

Abdominal ultrasound unremarkable apart from very mild pyelectasia, thought consistent with his degree of polyuria

Orthogonal CXR's and lateral AXR unremarkable

(CT unavailable at the time)

18

Initial way forward



- Elected to give a three-week course of oral clavulanate-potentiated amoxycillin
- Broad spectrum (E.coli)
- Urinary concentrated

19

Initial way forward



- Elected to give a three-week course of oral clavulanate-potentiated amoxycillin
- Broad spectrum (E.coli)
- Urinary concentrated
- **No improvement in PUPD noted at all**

20

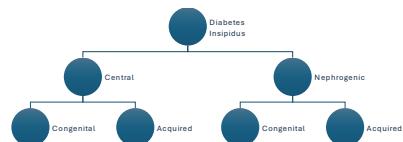
Differential diagnosis for Bobby



- Endocrine disease
 - HAC
 - HyAC
 - HyperT4
 - HyperPTH
 - DM
 - DI
- Non-endocrine disease
 - Renal failure
 - Neoplasia
 - 1st Hypercalcemia
 - Hyperviscosity
 - 1st PCV
 - 2nd PCV
 - Multiple myeloma
 - Renal infections
 - Pyometra
 - Hepatic disease
 - Renal glomerular or tubular Dz
 - FAN
 - Fanconi syndrome
 - Renal tubular acidosis
 - Primary polydipsia

21

Diabetes Insipidus



22

Water deprivation test



- Although the most likely Dx is DI, we need a WDT to confirm
- In light of pre-renal azotaemia at presentation (checked several times subsequently after free-access to water), we chose to perform a modified WDT, very carefully!
- 150ml/kg/day 72hours before admission, 120ml/kg/day 48-hours before admission and 100ml/kg/day 24-hours before admission

23

Water deprivation test



- USG 1.005
- Standard WDT protocol now
- 5% BW loss recorded after 6 hours; USG 1.005
- Therefore primary polydipsia ruled out as suspected and **diabetes insipidus confirmed**

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Water deprivaton test



- DDAVP (0.5ml of 4µg/ml injectable solution) was therefore administered intramuscularly
- Maintenance water requirements (3ml/kg/hr) provided
- The bladder was emptied hourly and USG recorded
- USG increased gradually to 1.016 eight hours post DDAVP administration
- Bobby remained bright during this period with no evidence of CNS depression

25

So have we now reached a definitive diagnosis?



- A. Yes, Bobby has central diabetes insipidus
- B. Yes, Bobby has nephrogenic diabetes insipidus
- C. I'm not sure, because his DDAVP response is sub-optimal

26

Answer!



- A. Yes, Bobby has central diabetes insipidus
- B. Yes, Bobby has nephrogenic diabetes insipidus
- C. I'm not sure, because his DDAVP response is sub-optimal

27

Bobby outcome



- Treated with oral DDAVP tablets (0.1mg) BID
- Choice to use tablets was based on the fact they are easier to give and in my experience, more consistently efficacious
- Eye drops a cheaper, but the bottles are glass so I think not as easy to use
- Bobby lost to follow up after six years but had remained well throughout this period with normal water intake

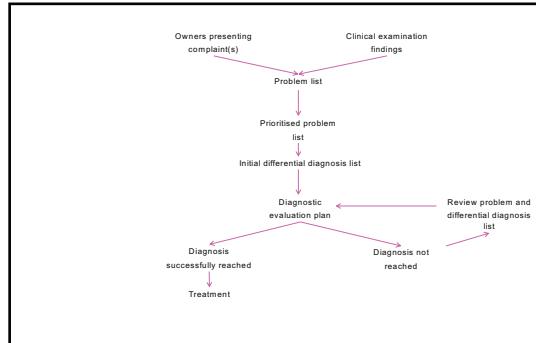
28

Conclusions – I hope this differential list is a practical help for your future cases!



- | | |
|---|---|
| <ul style="list-style-type: none"> • Endocrine disease <ul style="list-style-type: none"> • HAC • HyAC • HyperT4 • HyperPTH • DM • DI | <ul style="list-style-type: none"> • Non-endocrine disease <ul style="list-style-type: none"> • Renal failure • Neoplasia • Hypercalcaemia • Hyperviscosity <ul style="list-style-type: none"> • 1st PCV • 2nd PCV • Multiple myeloma • Renal infections • Pyometra • Hepatic disease • Renal glomerular or tubular Dz <ul style="list-style-type: none"> • PLN • Fanconi syndrome • Renal tubular acidosis • Primary polydipsia |
|---|---|

29



30

The Blood Sugar Rollercoaster;
strategies for managing
challenging diabetic
patients

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Granta Veterinary Specialists, UK



1

Plan

- Welcome!
- Let's get thinking – a clinical conundrum!
- Problem-orientated medicine – how can it help?
- Looking ahead - what to expect



2

Case history

- Arthur, an 11-y/o MN DSH cat
- Presented with a two-week history of eating more and his owners noticing him drinking indoors
- He was an indoor-outdoor cat but his owners also noticed that he was staying indoors more often and he was sleeping more than usual



3

Physical examination

- On examination, Arthur was quiet but alert and responsive
- No abnormalities on cardiopulmonary auscultation
 - HR 148bpm
 - mm pink
 - Femoral pulses normal
- Abdominal palpation non-painful and no obvious abnormalities
- LN palpation unremarkable
- T = 38.2°C
- Weight gain noted since
 - Since vaccination 10 months previously he had gone from 5.2kg to 6.8kg
 - Didn't feel particularly fat though!



4

Next steps

- What further questions would you like to ask the owners?
- What are your initial thoughts?
- What do you want to do?



5

Actions!

- Create a problem list!
- ? Polydipsia
- Polyphagia
- Weight gain
- Lethargy

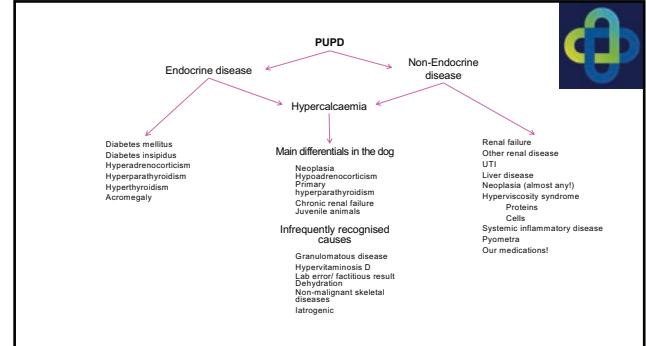


6

What are your thoughts on possible differential diagnoses?



7



8

Differential diagnosis for PU/PD



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 - HAC
 - HyAC
 - HyperT4
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 - DI
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 - Renal failure
 - Neoplasia
 - 1^o Hypercalcaemia
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 - 1^o PCV
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 - Renal glomerular or tubular Dz
 - PLN
 - Fanconi syndrome
 - Renal tubular acidosis
 - Primary polydipsia

9

Polyphagia differential diagnosis list



- Primary PP
 - Destruction of satiety centre
 - Trauma
 - Mass lesion
 - Infection
 - Psychogenic causes
 - Stress
 - More palatable diet
- Secondary PP
 - Physiologic increase in metabolic rate
 - Cold
 - Lactation
 - Pregnancy
 - Growth
 - Increased exercise
 - Pathological increase in metabolic rate
 - Hyperthyroidism
 - Acromegaly
 - Decreased energy supply
 - DM
 - Malabsorption
 - EPI
 - IBD
 - Parasitic disease
 - Lymphangiectasia
 - Reduced intake
 - Megaoesophagus
 - Low calorie diet
 - Hypoglycemia
 - Unknown
 - HAC
 - PSWHE
 - SARDs

10

Polyphagia differential diagnosis list



- Primary PP
 - Destruction of satiety centre
 - Trauma
 - Mass lesion
 - Infection
 - Psychogenic causes
 - Stress
 - More palatable diet
- Drug-induced
 - Glucocorticoids
 - Anticonvulsants
 - Antihistamines
 - Benzodiazepines
 - Cyproheptadine
- Secondary PP
 - Physiologic increase in metabolic rate
 - Cold
 - Lactation
 - Pregnancy
 - Growth
 - Increased exercise
 - Pathological increase in metabolic rate
 - Hyperthyroidism
 - Acromegaly
 - Decreased energy supply
 - DM
 - Malabsorption
 - EPI
 - IBD
 - Parasitic disease
 - Lymphangiectasia
 - Reduced intake
 - Megaoesophagus
 - Low calorie diet
 - Hypoglycemia
 - Unknown
 - HAC
 - PSWHE
 - SARDs

11

Weight gain DDx grouping



- Physiologic
 - Excessive calorie intake
 - Reduced calorie expenditure
- Pathological
 - Reduced metabolic rate
 - Hypothyroidism
 - Increased appetite
 - Endocrine disease
 - Unusual to cause weight gain if not hypothyroid
 - Acromegaly
 - Satiety centre pathology
 - Fluid accumulation
 - Ascites

12

Weight gain DDx grouping

- Physiologic
 - Excessive calorie intake
 - Reduced calorie expenditure
- Pathological
 - Reduced metabolic rate
 - Hypothyroidism
 - Increased appetite
 - Endocrine disease
 - Unusual to cause weight gain if not hypot4
 - Acromegaly
 - Satiety centre pathology
 - Fluid accumulation
 - Ascites



13

Lethargy group DDx

- Metabolic disease
 - Hepatic disease
 - Biliary disease
 - Renal disease
 - Cardiac disease
 - Endocrine disease
 - Electrolyte imbalance
- Discomfort/pain
 - IVDD
 - OA
 - UTI
- Neoplasia
- Haematological disease
 - Anaemia
 - Polycythaemia
- Respiratory disease
- Neuro-muscular disease
 - Myositis
 - Neuropathies
- Inflammatory disease
- Infectious disease
- Blood pressure abnormalities



14

What to do next?

- ? What to do next?



15

Next steps

- Main differentials appear to be grouped in:
 - Endocrine disease
 - CNS disease
- Therefore, our workup needs to focus on the possible differentials we have
- Need:
 - CBC
 - SBC
 - UA
 - Neurological examination



16

Haematology

- | | | |
|---------------|------------------------|---------------|
| • Red cells | $6.2 \times 10^{12}/l$ | (5.5 - 8.5) |
| • Haemoglobin | 14.8 g/dl | (12.0 - 18.0) |
| • PCV | 0.35 l/l | (0.27 - 0.45) |
| • MCV | 64.6 fl | (59.0 - 77.0) |
| • MCH | 23.1 pg | (20 - 26) |
| • MCHC | 33.2 g/dl | (30 - 36) |
| • White cells | $13.7 \times 10^9/l$ | (6.0 - 15.0) |
| • Neutrophils | $9.4 \times 10^9/l$ | (2.5 - 12.5) |
| • PLT count | $337 \times 10^9/l$ | (150 - 450) |
- Film comment: Red cells appear normocytic normochromic. No abnormal white cells seen. Platelet numbers appear consistent with machine count.



17

Serum biochemistry

• Total protein	73.8 g/l	(55.0 - 75.0)
• Albumin	37.1g/l	(25.0 - 40.0)
• Globulin	36.7 g/l	(20.0 - 45.0)
• Urea	9.7 mmol/l	(2.5 - 6.7)
• Creat	91.0 μ mol/l	(20 - 150)
• ALT	72 IU/L	(10.0 - 60.0)
• ALP	83 IU/L	(< 130)
• Glucose	21.4 mmol/l	(3.3 - 5.8)
• Chol	3.8 mmol/l	(2.8 - 7.8)
• Bilirubin	4.9 μ mol/l	(0.1 - 5.1)



18

Serum biochemistry

• Total protein	73.8 g/l	(55.0 - 75.0)
• Albumin	37.1g/l	(25.0 - 40.0)
• Globulin	36.7 g/l	(20.0 - 45.0)
• Urea	9.7 mmol/l	(2.5 - 6.7)
• Creat	91.0 umol/l	(20 - 150)
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• ALP	83 IU/L	(< 130)
• Glucose	21.4 mmol/l	(3.3 - 5.8)
• Chol	3.8 mmol/l	(2.8 - 7.8)
• Bilirubin	4.9 umol/l	(0.1 - 5.1)



Further serum biochemistry

• CK	215 µmol/l	(20 – 225)
• Sodium	150.0 mmol/l	(135 – 155)
• Potassium	3.36 mmol/l	(3.6 - 5.6)
• Chloride	155 mmol/l	(100 – 116)
• Phos	1.08 mmol/l	(0.8 - 1.6)
• Calcium	2.52 mmol/l	(2.40 - 2.90)



- What else should we assess?

19

20

Urinalysis

• Appearance:	Clear, pale yellow	• Red cells	2 - 4 per hpf
• SG	1.024	• White cells	10 - 20 per hpf
• ph	8.8 (ref. 5.5 - 7.5)	• Epithelial cells	Negative
• Protein	+++	• Casts	Negative
• Nitrite	Negative	• Crystals	Negative
• Leucocytes	+++	• Bacteria	+ve
• Blood/Hb	++	• Urine culture	Profuse growth of E.Coli
• Glucose	++++		
• Ketones	Negative		
• Bilirubin	Negative		
• Urobilinogen	Negative		



So what would you do next?

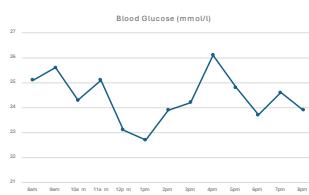


21

22

Treatment

- Started treatment with 0.5iu Caninsulin BID
- Changed food to Purina DM
- Commenced antibiosis with amoxycillin 75mg BID
- Performed BGC after seven days:



Urinalysis

• Appearance:	Clear, pale yellow	• Red cells	0 - 1 per hpf
• SG	1.020	• White cells	0 – 2 per hpf
• ph	6.8 (ref. 5.5 - 7.5)	• Epithelial cells	Negative
• Protein	Negative	• Casts	Negative
• Nitrite	Negative	• Crystals	Negative
• Leucocytes	++	• Bacteria	-ve
• Blood/Hb	Negative	• Urine culture	Negative
• Glucose	+++		
• Ketones	Negative		
• Bilirubin	Negative		
• Urobilinogen	Negative		

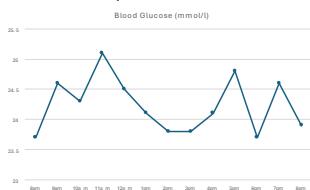


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24

Treatment

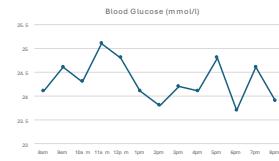
- Increased treatment with 0.6iu Caninsulin BID
- Continued feeding Purina DM
- No further antibiosis
- Performed repeat BGC after seven days:



25

Treatment

- Continued to increase Caninsulin dose by 0.1iu BID once a week for next 8 weeks with BGC
- Continued feeding Purina DM
- No further antibiosis
- Performed repeat BGC after seven days of 1.5iu/kg BID:



26

Treatment of DM

- Cats and dogs generally have different pathogenesis behind their disease
 - Dogs usually analogous to insulin-deficiency DM and cats usually analogous to insulin resistance DM
 - However, as JVIM paper showed, there are many possible causes of DM
- However, both require insulin therapy to control their disease
 - Accurate evaluation of possible underlying conditions important
- Diabetic remission possible in 15-50% of cats; generally not possible in dogs unless cause of resistance found and treated
- Diets do help improve response to Tx, especially in cats
- The better the level of glycaemic control, the better the outcome
 - Aim to get BG < 10mmol/l for the majority of the day!
- All diabetics develop diabetic cataracts unless control excellent (but cats not usually clinically affected) but cataract surgery successful – refer early!!!!



27

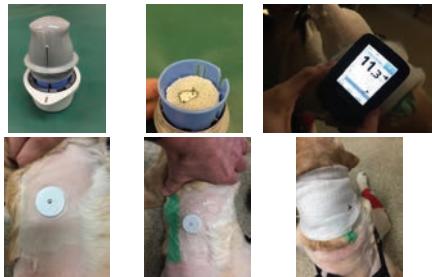
What to do next?

- Most patients require between 0.8 – 1.2 iu/kg/dose to stabilise in the author's experience
- Clinically relevant reduction in insulin efficacy should be suspected at doses > 1.5iu/kg with true insulin resistance being defined as no response at doses > 2.2iu/kg
- If this is the case, firstly rule out owner feeding, insulin care and Tx compliance
- If this is good however then there is peripheral resistance and you need to investigate the cause
- Therefore, before undertaking blood glucose curves it is important to investigate simple causes of instability
 - Check handling and storage of insulin and injection technique
 - Check owners log and feeding records
 - Clinical examination may reveal a cause of insulin antagonism
 - Consider main DDx!



28

Blood Glucose Curves



29

Blood glucose curves

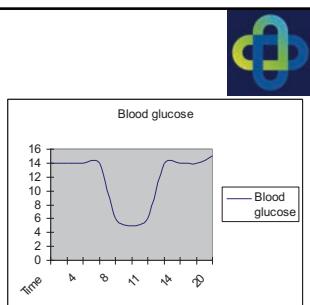
- May highlight four possible causes of instability:
 - Inadequate response – dose needs to go up
 - Rapid insulin metabolism
 - Somogyi over-swing (much less common than thought!)
 - Insulin resistance (go looking for the cause)



30

Rapid Insulin Metabolism

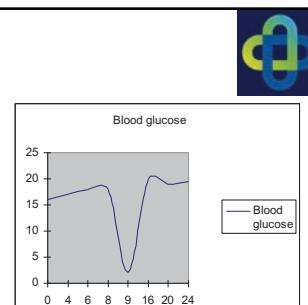
- In some animals, intermediate forms of insulin do not last for 12 hours
- Blood glucose drops after each insulin injection but returns to be excessive within ~ 6 hours
- Can be corrected by:
 - dosing twice daily with PZI if Caninsulin/Prozinc being used
 - twice daily dosing if SID PZI is being used
 - switching to Glargin
 - considering use of human lente



31

Somogyi overswing

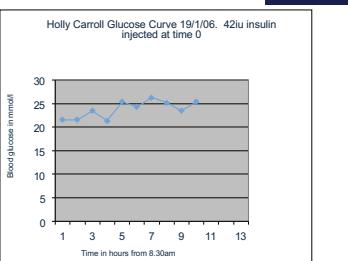
- Excessive insulin leading to paradoxical hyperglycaemia
- Large dose can result in hypoglycaemia (clinical signs may not be obvious) and release of counter-regulatory hormones (adrenaline, glucagon, cortisol, GH) which rapidly increase blood glucose levels
- Much less common than people think!
- History usually of rapid and marked increases in insulin doses
- Therefore, should be avoidable



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Potential causes of insulin resistance

- Infection (eg: UTI)
- Concurrent endocrinopathy (eg: hyperadrenocorticism, hyperthyroidism, acromegaly)
- Neoplasia
- (Stress)
- (True insulin resistance)



33

Potential causes of insulin resistance

- HAC most common co-endocrinopathy in dogs
 - ACTH stimulation best test to choose
 - Lower risk of false +ve
 - Only test in dogs with compatible clinical signs
 - If positive, treat as usual but reduce insulin dose by 25%
- Hypothyroidism can make management of DM challenging as the metabolism of insulin will be altered, but hypoT4 doesn't itself cause insulin resistance
- The prolonged progesterone phase in entire female dogs causes insulin resistance and spaying ASAP if DM develops is crucial!
- Canine acromegaly is seen in entire females as a result of progesterone-induced IGF-1 production from the mammary glandular epithelium
 - Spaying should help overcome insulin resistance, but physical features may remain
 - GH-producing pituitary tumours are rare in dogs

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Potential causes of insulin resistance

- In cats, acromegaly is now known to be more common than we used to think:
 - Niessen et.al. (2015), *PLoS One*
 - 1221 cats screened for acromegaly
 - 319 (26.1%) blood test positive
 - 60/63 (subset) were confirmed to have a causal pituitary mass
 - The study concluded that the overall prevalence of acromegaly in diabetic cats in the UK was 24.9%
 - Interestingly, only 24% of the attending clinicians suspected any of the positive cases to have acromegaly, indicating the majority of acromegalic cats do not display the classical clinical signs
 - The author now recommends screening all newly diagnosed diabetic cats for acromegaly as part of the serum biochemistry investigations
 - Almost always 9-12 y/o male cats, cf: acromegaly in dogs

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Diabetic ketoacidosis

- Anorexia, vomiting, dehydration, depression
- Lethargy, weakness, plantigrade stance
- Ketone breath
- Most have a previous history of PU/PD although some animals first present with DKA
- Azotaemia
- Hyperglycaemia
- Ketonuria and glucosuria
- ↑ hepatocellular enzymes
- Decreased / normal potassium
- Decreased / normal phosphate
- Metabolic acidosis

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DKA

- Ketones measured by urine dipstick only measure acetoacetate & acetone and not beta-hydroxybutyrate (BHB)
- BHB predominates in hypovolaemic severely ketotic patients
- As these patients are treated they produce more acetone and acetoacetate and therefore dipsticks will indicate a more positive reaction before becoming negative
- Therefore, use BG as marker of response!



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DKA Treatment

- Baseline bloods – biochemistry (incl electrolytes) and haematology
- Treatment goals:
 - a) Rehydration
 - b) Stop ketogenesis
 - c) Correct electrolyte abnormalities
 - d) Treat underlying disease
 - e) Consider antibiosis – go looking for UTI!

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Rehydration

- Most ketotic patients are >5% dehydrated
- Accurate assessment of fluids required
- Shock doses can be given initially (90ml/kg/hour)
- 50% deficit + maintenance requirements in first 6 hours then remaining deficit
- 0.9% NaCl /Hartmanns can be used
- Urine output should be monitored
- ? Should we also add in dextrose?

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DKA Insulin therapy

- CRI – add 25iu soluble insulin to 500ml fluids and infuse at 1.0ml/kg/hour
- Can use a syringe pump using 2.5iu in 50ml saline and infuse at same rate
- Can piggy-back into fluid line or separate IV access
- Alternatively, use intermittent pulse i/v administration giving 0.1iu/kg IV q 1hour until BG is between 8 – 15mmol/l
- Continue treatment until glucose falls to < 15mmol/l
- Then start s/c Caninsulin or Prozinc at 0.5iu/kg (or as required if not a newly Dx DM patient) but continue to monitor BG and re-start soluble insulin if BG not responding
- Careful monitoring of blood glucose required but remember that patients likely to remain ketone +ve for ~ three days

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Electrolyte abnormalities in DKA

- Deficits in whole body potassium are extremely common in DKA
- Serum potassium may be normal at presentation
- During treatment potassium levels fall due to:
 - rehydration (dilution effect)
 - correction of acidosis (transcellular shift)
 - insulin therapy (insulin-mediated cellular uptake)
 - Recommend checking potassium q 12 hours as a minimum in DKA patients once you start insulin treatment
- DO NOT UNDERESTIMATE THE POSSIBLE SERIOUSNESS OF HYPOKALAEMIA IN D.K.A.!

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Normal cat dental arcade cf: Arthur



42

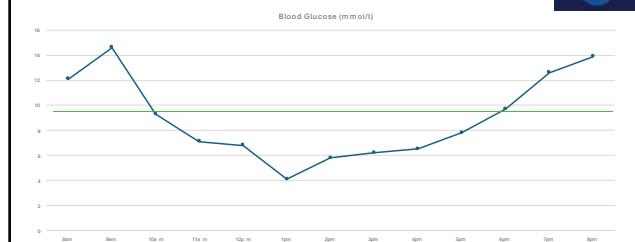
What happened to Arthur

- Measured IGF-1: Result 1220 iu/l
 - Performed MRI of brain and identified a pituitary microadenoma
 - Treated with 10-fractions of external beam RTx at The University of Cambridge
 - Insulin dose reduced back to 0.6 iu/kg BID and he stabilised perfectly!
 - Other treatment options are:
 - Hypophysectomy (few centres!)
 - Parlotide (somatostatin analogue) has also been shown to reduce IGF-1 levels and improve insulin sensitivity



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Arthur BGC three months post RTx, 0.6iu Caninsulin given at 8am with Purina DM breakfast



44

Possible future treatment in cats?

- Gilor C et.al. (2021), JVIM
 - Used a novel ultra-long acting recombinant insulin that requires injection only once a week
 - Feline insulin fused with IgFc, to protect the insulin from proteolysis
 - Only five cats in the study
 - Transitioned from BID Glargine on which they had all been stable
 - Showed maintenance of successful control with the one weekly injections
 - Huge potential!
 - All waiting for larger scale trial data...

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Possible future treatment in dogs?



Long-Term Efficacy and Safety of Insulin and Glucokinase Gene Therapy for Diabetes: 9-Year Follow-Up in Dogs

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Conclusions

- In unstable diabetics, always check the basic things first!
 - If you are happy that the insulin handling, injection technique and feeding are all good, then perform a glucose curve
 - Gradually increase the insulin dose but not changing the dose more than once every five days
 - If you get to 1.5iu/kg or greater, go looking for causes of insulin resistance:
 - Infection
 - Concurrent endocrinopathy
 - Neoplasia
 - Genuine insulin resistance
 - In which case, move to Glargin

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Ions in Action; Navigating the maze of abnormal electrolytes in cats and dogs

Rob Foale BSc BVetMed DSAM
DipECVIM-CA FRCVS
Granta Veterinary Specialists, UK



1

Case history

- Katy, a 9-year old FE Irish Setter
- Presented with a two week history of progressively worsening lethargy, reduced exercise tolerance/willingness to exercise, reducing appetite leading to anorexia for past 48 hours and weight loss
- No travel history and no abnormalities found at booster vaccination five months previously



2

Physical examination

- On examination, Katy was quiet and although initially responsive, she quickly lay down and seemed disinterested in her surroundings
- HR 132bpm, mm pale pink and tacky, femoral pulses subjectively weaker than expected
- RR 24/min with reduced pulmonary sounds on auscultation
- Abdominal palpation non-painful and no obvious abnormalities
- LN palpation unremarkable
- T = 39.4°C
- Weight loss noted since vaccination 5 months previously
 - 24.9kg to 22.4kg



3

Next steps

- What further questions would you like to ask the owners?
- What are your initial thoughts?
- What do you want to do?



4

Actions!



- What do you want to do?
- Create a problem list!

5

Actions!



- What do you want to do?
- Create a problem list!
- Lethargy
- Weight loss and reduced/no appetite
- Tachycardia
- Tachypnoea
- Mild pyrexia

6

What are your thoughts on possible differential diagnoses?



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Lethargy group DDx

- Metabolic disease
 - Hepatic disease
 - Biliary disease
 - Renal disease
 - Cardiac disease
 - Endocrine disease
 - Electrolyte imbalance
- Discomfort/pain
 - IVD
 - OA
 - UTI
- Neoplasia
- Haematological disease
 - Anaemia
 - Polycythemia
- Respiratory disease
- Neuro-muscular disease
 - Myositis
 - Neuropathies
- Inflammatory disease
- Infectious disease
- Blood pressure abnormalities



8

Weight loss DDx

- Inadequate diet
 - Starvation
 - Underfeeding
 - Poor quality food
- Adequate diet
 - Chronic inflammation
 - Any cause
 - Pain
 - Neoplasia
- Adequate diet with no evidence of chronic inflammation or neoplasia
 - Environmental/housing
 - Oral/dental disease
 - Malabsorption/malabsorption
 - Endocrine disease
 - DM, hyperT4
 - Protein-losing disease
 - Cardiac disease
 - End-stage renal disease
 - Chronic infection



9

Anorexia DDx

- Interested in food
 - Environmental stress
 - Unpalatable dietary change
 - Nausea worsened by smell of food
- Not interested in food
 - Unable to smell food
 - Medications
 - Chemotherapy
 - Oral cavity disease
 - Pain or impairment
 - Respiratory disease
 - Systemic disease
 - Inflammatory
 - Infectious
 - Immune-mediated
 - Neoplastic



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Tachycardia DDx

- Physiological
 - Exercise
 - Excitement
 - Anxiety/Fear
 - Pregnancy
- Pathological
 - Pyrexia
 - Pain
 - Cardiac disease
 - Chronic lung disease
 - Hypoxia
 - Hypovolaemia
 - Anaemia
 - Hypotension
 - Thyrotoxicosis



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Pyrexia DDx

- Non-febrile hyperthermia
 - Excessive environmental heat/heat stroke
 - Exercise
 - Seizures/Tetany
 - Hyperthyroidism
- True pyrexia
 - Infection/inflammation
 - Immune-mediated disease
 - Neoplasia



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What to do next?

- ? What to do next?



Next steps

- Main differentials are broad with cardiac or thoracic disease, inflammatory disease and metabolic disease groupings main foci
- Therefore, our workup needs to be broad and involve a combination of diagnostic modalities
- Need:
 - Urinalysis
 - CBC
 - SBC
 - Possibly ECG
 - Diagnostic imaging
 - CXR
 - Possibly an echo?



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Urinalysis

- | | | | |
|----------------|----------------------|--------------------|---------------|
| • Appearance: | Clear, pale yellow | • Red cells | 0 - 1 per hpf |
| • SG | 1.042 | • White cells | 1 - 2 per hpf |
| • pH | 6.8 (ref. 5.5 - 7.5) | • Epithelial cells | Negative |
| • Protein | + | • Casts | Negative |
| • Nitrite | Negative | • Crystals | Negative |
| • Leucocytes | Negative | • Bacteria | Negative |
| • Blood/Hb | Negative | • Urine culture | Negative |
| • Glucose | Negative | | |
| • Ketones | Negative | | |
| • Bilirubin | Negative | | |
| • Urobilinogen | Negative | | |



Haematology

- | | | |
|---------------|------------------------|---------------|
| • Red cells | $6.2 \times 10^{12}/l$ | (5.5 - 8.5) |
| • Haemoglobin | 13.8 g/dl | (12.0 - 18.0) |
| • PCV | 0.37 l/l | (0.35 - 0.55) |
| • MCV | 63.4 fl | (59.0 - 77.0) |
| • MCH | 22.8 pg | (20 - 26) |
| • MCHC | 33.2 g/dl | (30 - 36) |
| • White cells | $19.7 \times 10^9/l$ | (6.0 - 15.0) |
| • Neutrophils | $17.4 \times 10^9/l$ | (2.5 - 12.5) |
| • Lymphocytes | $0.8 \times 10^9/l$ | (1.0 - 2.5) |
| • PLT count | $441 \times 10^9/l$ | (150 - 450) |
- Film comment: Red cells appear normocytic normochromic. Increased neutrophil count but with no sign of toxic change. Platelet numbers appear consistent with machine count.



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Haematology

- | | | |
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- Film comment: Red cells appear normocytic normochromic. Increased neutrophil count but with no sign of toxic change. Platelet numbers appear consistent with machine count.



Serum biochemistry

- | | | |
|-----------------|-------------------|---------------|
| • Total protein | 72.8 g/l | (55.0 - 75.0) |
| • Albumin | 36.3g/l | (25.0 - 40.0) |
| • Globulin | 36.5 g/l | (20.0 - 45.0) |
| • Urea | 10.7 mmol/l | (2.5 - 6.7) |
| • Creat | 123.0 μ mol/l | (20 - 150) |
| • ALT | 72 IU/L | (10.0 - 60.0) |
| • ALP | 283 IU/L | (< 130) |
| • Glucose | 6.4 mmol/l | (3.3 - 5.8) |
| • Chol | 5.8 mmol/l | (2.8 - 7.8) |
| • Bilirubin | 2.7 μ mol/l | (0.1 - 5.1) |



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Further serum biochemistry



• CK	215 µmol/l	(20 – 225)
• Sodium	124.0 mmol/l	(135 – 155)
• Potassium	4.9 mmol/l	(3.6 - 5.6)
• Chloride	92.8 mmol/l	(100 – 116)
• Phos	1.08 mmol/l	(0.8 - 1.6)
• Calcium	2.52 mmol/l	(2.40 - 2.90)

- What else should we assess?

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Further serum biochemistry



• CK	215 µmol/l	(20 – 225)
• Sodium	124.0 mmol/l	(135 – 155)
• Potassium	4.9 mmol/l	(3.6 - 5.6)
• Chloride	92.8 mmol/l	(100 – 116)
• Phos	1.08 mmol/l	(0.8 - 1.6)
• Calcium	2.52 mmol/l	(2.40 - 2.90)
• Na:K	25.3	

- What else should we assess?

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So what would you do next?



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Hyponatraemia DDx



True hyponatraemia

- Iatrogenic
 - Inappropriate IVFT
- Sodium loss
 - GIT loss
 - Significant D+ with only water replacement
 - Urinary loss
 - ARF
 - Hypoadrenocorticism
 - Third-space fluid accumulation
- Excess water
 - Water toxicity
 - Reduced renal perfusion
 - Resulting in excessive water retention
- SIADH

Pseudo-hyponatraemia

- Hyperlipidaemia
- Hyperproteinæmia

Hyperglycaemia

- Causes dilutional hyponatraemia if hyperglycaemia marked

Lab error

- Unusual with current electrode measurement methods

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Taken from Heinz J. and Cook A., Today's Veterinary Practice, 2022

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Hyponatraemia DDx



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24

Next steps

- White cells $19.7 \times 10^9/l$ (6.0 - 15.0)
- Neutrophils $17.4 \times 10^9/l$ (2.5 - 12.5)
- Lymphocytes $0.8 \times 10^9/l$ (1.0 – 2.5)
- ? Hypoadrenocorticism likely?



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Next steps

- White cells $19.7 \times 10^9/l$ (6.0 - 15.0)
- Neutrophils $17.4 \times 10^9/l$ (2.5 - 12.5)
- Lymphocytes $0.8 \times 10^9/l$ (1.0 – 2.5)
- ? Hypoadrenocorticism likely?
- Basal cortisol 178 nmol/l
- Post-stim cortisol 443 nmol/l



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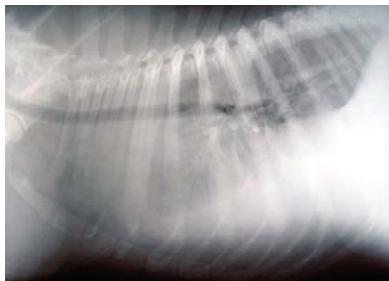
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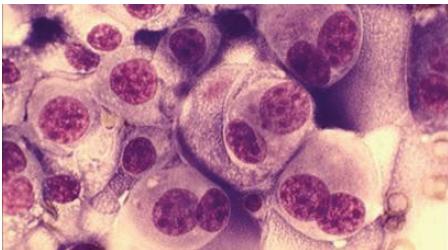
27

R. Lat CXR



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Thoracocentesis cytology



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A word on fluid therapy in hyponatraemia

- Increasing plasma $[Na^+]$ concentration too rapidly risks permanent neuronal osmotic damage, especially if the situation has been long-standing and plasma sodium < 125 mmol/l
- In situations like Katy's, we should aim to increase the plasma $[Na^+]$ no more quickly than 10mmol/l/24 hours and we can calculate the sodium deficit and how quickly to replace this by:
 - Expected change in $[Na]$ with 1 litre of fluids = $(\text{Fluid } [Na + K] - \text{Patient } [Na]) / (\text{TBW} + 1)$
 - Where:
 - TBW = Total body water = Weight in kg $\times 0.6$
 - Sodium deficit (mmol) = $(\text{Target } [Na] - \text{Patient } [Na]) \times \text{TBW}$
 - Where:
 - Target $[Na] = 150 \text{ mmol/L}$
 - Time to replace sodium deficit (hr) = $(\text{Target } [Na] - \text{Patient } [Na]) \times 2.4 \text{ hr}$



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Further steps for Katy

- Thoracic fluid a neoplastic exudate
- Cellular appearance consistent with a carcinoma
- Therefore, question is, is this a primary intra-thoracic neoplasia, or an unusual manifestation of metastatic disease?
- Therefore action:
 - Commenced careful IVFT monitoring her $[Na^+]$ every 2 – 3 hours
 - Drained pleural fluid
 - Repeated orthogonal CXR's – no pathology seen
 - CT typically down that day!
 - Undertook AUS
 - Identified large, irregular, heterogenous left ovarian mass lesion
 - Appearance potentially consistent with ovarian CA



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Further steps

- After much discussion, owner elected for ovariectomy and follow-up intra-pleural chemotherapy with carboplatin
- Corrected serum $[Na^+]$ first!
 - Took 62 hours to achieve stable $[Na^+]$ WNL
- CT confirmed no other mass lesions at all
- Histology on L ovary confirmed an ovarian carcinoma
- Instilled 300mg/m² carboplatin (50% RHS, 50% LHS) diluted 1:1 with 0.9% saline into pleural space five days post-op
- Katy made a good recovery, serum $[Na^+]$ remained normal and pleural effusion did not recur
- Three more intra-pleural chemotherapy administrations given q 4 weeks without incident
- Katy remained well for 18 months but then effusion recurred and owner opted for PTS



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But why did this/did this explain the hyponatraemia?

- Third-space fluid loss is an uncommon, but recognized, cause of hyponatraemia
 - Fluid movement into thorax reduces circulating volume, so thirst response triggered
 - If only water ingested and patient anorexic, then hyponatraemia can result
 - Reduced circulating volume also triggers ADH release, which in turn causes increased reabsorption of pure water, potentially exacerbating the dilutional effect on $[Na^+]$
 - This explanation seemed most likely, as Katy was slightly dehydrated on exam, had a high USG and had a poor appetite leading to anorexia but she had continued to drink water
- SIADH
 - Unusual/rare condition where there is excessive and inappropriate ADH release secondary to another pathology (eg: pituitary tumour)
 - Causes hyponatraemia with hypo-osmolar serum but normovolaemia; not the case with Katy



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Approach to sodium abnormalities

- Hypernatraemia
 - Excessive sodium intake
 - Food
 - Salt water
 - Play Doh
 - Inappropriate IVFT
 - Excessive sodium retention
 - Hyperaldosteronism
 - Usually accompanied by hypokalaemia
 - Excessive free water loss
 - Diabetes Insipidus
 - Acute kidney injury causing polyuria
 - Marked vomiting and/or diarrhoea
 - Heat stroke
 - Inadequate water intake
 - Primary polydipsia
 - Congenital
 - Inflammatory
 - Neoplastic
- Hyponatraemia
 - Pseudo-hyponatraemia
 - Hyperlipidaemia
 - Hyperproteinæmia
 - Hyperglycaemia
 - Iatrogenic
 - Inappropriate IVFT
 - Excessive sodium loss
 - GIT loss
 - Urinary loss
 - ARF
 - Hypoadrenocorticism
 - Diuretics
 - Third-space fluid accumulation
 - Excess water intake/retention
 - Water toxicity
 - Reduced renal perfusion
 - SIADH



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Other electrolyte abnormalities

- Chloride
 - As a general rule, chloride follows sodium to maintain electrical neutrality, so usually changes in sodium are proportional to changes in chloride and the underlying pathology will relate more often to the sodium change (which relates to free water volume) than the chloride change
 - However, chloride is also linked to a patient's acid-base status, because via Stewart's principles, chloride is a strong anion so it effectively acts as an acid such that:
 - Chloride levels can rise in metabolic acidosis
 - Chloride levels can fall in metabolic alkalosis
 - Therefore, knowing whether or not any change in [chloride] is due primarily to changes in free water volume or due to an acid-base imbalance can be helpful
 - To do this, if the blood pH is not known, calculating the corrected [chloride] will enable us to interpret the chloride levels with respect to the [sodium]:



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Corrected chloride

- Corrected Chloride = (normal $[Na^+]/\text{measured } [Na^+]$) $\times \text{measured Chloride}$
 - where, normal Na^+ is the midpoint of the sodium reference interval
- If we use this formula and then compare the result with the chloride reference range, we can establish if there is a true change in the [chloride] and then use the following table as a differential diagnosis list:



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Approach to chloride abnormalities

- Hyperchloraemia
 - Artifact
 - Most commonly caused by bromide treatment
 - Iatrogenic
 - Excess chloride-containing fluids such as hypertonic saline
- Hyperchloraemic metabolic acidosis
 - Caused by abnormal loss of HCO_3^- and retention of Cl^- in the kidney
 - Renal failure
 - Diarrhoea
 - Proximal renal tubular acidosis
 - Distal renal tubular acidosis
 - Compensatory metabolic acidosis
 - Deliberate loss of HCO_3^- and retention of Cl^- in the kidney in response to respiratory alkalosis
 - Hypocapnia or hyperventilation



Renal tubular acidoses

- Proximal RTA (type II)
 - Caused by reduced ability of PCT to reabsorb HCO_3^-
 - Results in a mild-moderate hyperchloraemic metabolic acidosis but this is self-limiting as DCT can still excrete H^+
 - Urine pH therefore usually < 6.0
 - Can be seen alone or in combination with other tubular defects (eg: Fanconi syndrome)
 - Treatment with oral NaHCO_3 often not effective and leads to bicarbonaturia with potassium wasting
- Distal RTA (type I)
 - Caused by reduced ability of DCT to excrete H^+ , minimizing ability of kidneys to maximise urinary acidification
 - Results in a moderate-severe hyperchloraemic metabolic acidosis with hypokalaemia
 - Urine pH however usually > 6.0
 - Treatment with NaHCO_3 usually effective and improves hypokalaemia
 - However, potassium or sodium citrate considered preferred treatment options



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Potassium

- Main intracellular cation, with 98% of total body K^+ being intracellular
- However, levels are influenced by many factors:
 - Extracellular pH and HCO_3^- concentrations
 - Acidosis causes potassium to move extracellularly to maintain electrical neutrality as H^+ moves intracellularly and alkalis vice versa
 - Insulin concentration
 - Causes movement of K^+ intracellularly
 - Catecholamine release
 - Cause activation of Na-K ATPase and lowers plasma $[\text{K}^+]$
 - T4 levels
 - Ta activates the Na-K ATPase and lowers plasma $[\text{K}^+]$
 - Renal function
 - Potassium is freely filtered and readily reabsorbed in the PCT (70%) and LoH (20%), but potassium balance is mainly controlled in the cortical portion of the collecting duct due to high numbers of Na-K ATPase pumps on the basolateral membrane here
 - ADH and aldosterone
 - Both promote kaliuresis



Approach to potassium abnormalities

- Hyperkalaemia
 - Pseudohyperkalaemia
 - Haemolysis
 - Severe leucocytosis or thrombocytosis
 - Japanese Akita
 - Failure to excrete
 - ARF
 - UT obstruction
 - Hypoadrenocorticism
 - Potassium-sparing diuretics
 - Excessive Na^+ loss
 - Marked intestinal inflammation
 - Third-space fluid loss
 - Diabetes mellitus
 - Excessive intake
 - Inappropriate IVFT
- Hypokalaemia
 - Artifact
 - Lipaemia
 - Hyperglobulinaemia
 - Transcellular shift
 - Alkalosis (although not usually seen clinically)
 - Insulin treatment
 - Excessive excretion
 - Hyperaldosteronism
 - Increased DCT flow (eg: post-obstructive diuresis)
 - Renal tubular acidosis
 - Loop diuretics
 - IVFT in anorexic cats
 - Severe V \ddagger /D \ddagger



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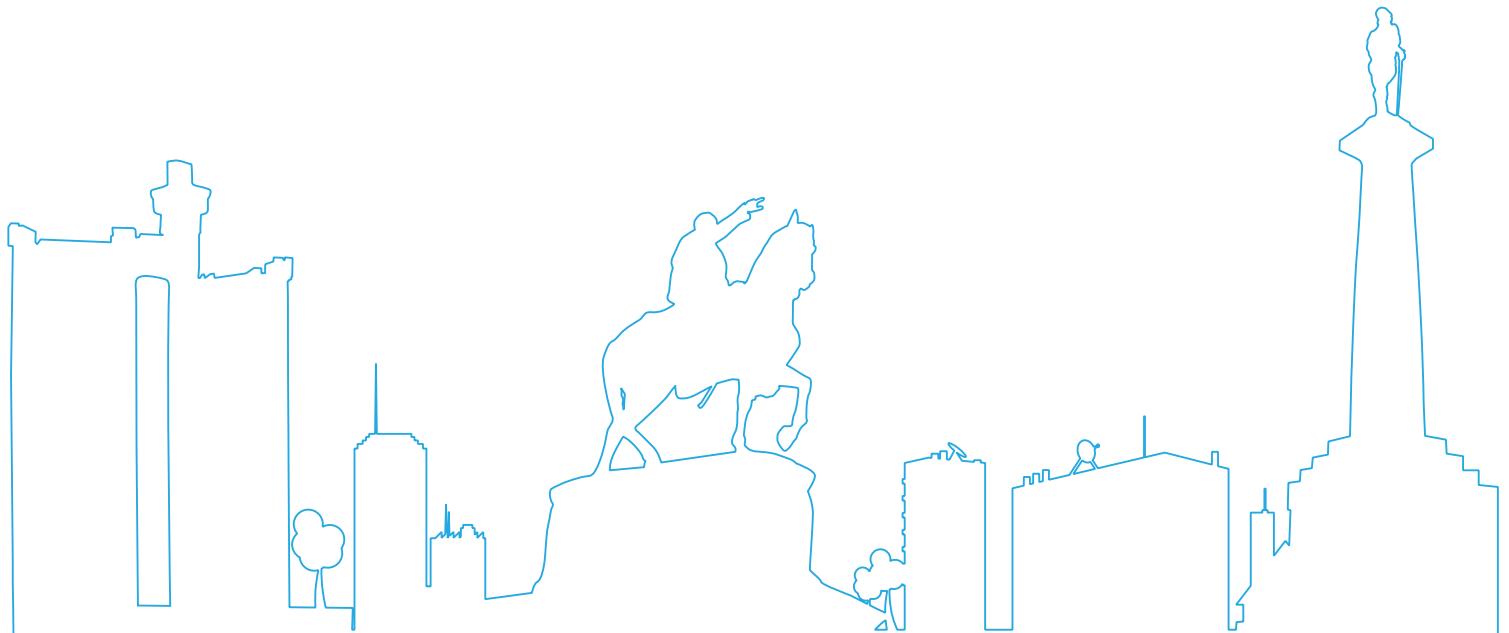
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Any questions?



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MANAGEMENT



**Elli Kalemtzaki (Greece)**

DVM, MSc, Certified Coach, NLP Certified Practitioner

EERVC 2024 Lectures

1. The Impact of Client Interactions on Veterinary Wellbeing
2. Leading with Flexibility: The Art of Situational Leadership in Veterinary Practice

Elli Kalemtzaki, DVM, MSc, Certified Coach, NLP Certified Practitioner

Elli is a graduate of the Faculty of Veterinary Medicine of the Aristotle University of Thessaloniki in Greece and holds a postgraduate degree from the National School of Public Health in Athens, Greece. She is also a Professional Coach certified by Adler International, Toronto, Canada since 2010 and a Certified Practitioner of Neuro-Linguistic Programming since 2012.

Elli comes with 20 years of experience in the pet food industry, the last 9 years in international positions during which she worked with diverse teams in countries vastly different from a cultural point of view. She has extensive experience in coaching and mentoring individuals, leading workshops and training business teams in several countries across Europe.

In 2017 she started her own business as an independent consultant originally based in Prague and later in Athens to help veterinary practitioners market their services, inspire, and motivate their teams and engage with clients. She recently joined Vet Planet as a Business Development Manager.

Elli is passionate about advancing the well-being of veterinary professionals worldwide and ensuring they work in a supportive and caring environment. She served as a member of the Board of the Hellenic Veterinary Medical Society from 2021-2024 and is co-Chair of the WSAVA Professional Wellness Committee since 2022.

THE IMPACT OF CLIENT INTERACTIONS ON VETERINARY WELLBEING

Elli Kalemzaki, DVM, MSc, Certified Coach & NLP Practitioner
kalemzaki@vetconsultancy.com

Veterinary medicine is a profession deeply rooted in compassion, expertise, and the unwavering commitment to the health and welfare of animals. However, beyond the clinical responsibilities lies an often underappreciated aspect of the job—client interactions. Client interactions are a fundamental aspect of veterinary practice, influencing not only the quality of care provided but also the emotional and mental well-being of veterinarians.^{1,2} Understanding this impact is crucial in fostering a healthier work environment and ensuring the sustainability of the profession.

Client interactions in veterinary practice are inherently dual-edged. On one hand, they can be a source of immense satisfaction, with positive client relationships leading to a sense of accomplishment and professional pride. When clients express gratitude, acknowledge the team's efforts, or provide positive feedback, it reinforces the veterinarian's purpose and can contribute to job satisfaction^{3,4}.

On the other hand, negative client interactions can be a substantial source of stress^{5,6}. These may include situations where clients are dissatisfied, uncooperative, or emotionally distressed due to the nature of their pet's condition. In some cases, clients may have unrealistic expectations, or financial constraints can lead to difficult conversations about the cost of care⁶. These scenarios can lead to feelings of frustration, helplessness, and even moral distress among veterinary professionals.

Research has shown that client interactions can directly impact the mental health and wellbeing of veterinary professionals^{1,2}. Veterinarians, veterinary nurses, and support staff are particularly vulnerable to stress, anxiety, and burnout due to the emotional labor involved in managing client relationships^{2,4}. The need to constantly display empathy and understanding, even in challenging situations, can be exhausting and lead to compassion fatigue.

Compassion fatigue, a specific type of burnout, occurs when veterinary professionals become emotionally drained from the continuous demand to care for suffering animals and support distressed clients. This condition can manifest as a reduced capacity to empathize, a sense of detachment, and a decline in job performance, ultimately affecting the quality of care provided to patients.

Moreover, the pressure to meet client expectations, especially in the face of adverse outcomes, can lead to moral distress. Moral distress arises when veterinary professionals are unable to act according to their ethical beliefs due to external constraints, such as financial limitations imposed by clients or practice policies. Over time, unresolved moral distress can contribute to a sense of disillusionment and reduce job satisfaction.

Effective communication is a key factor in managing the impact of client interactions on the wellbeing of the veterinary team. Training in communication skills can help veterinary professionals navigate difficult conversations, set realistic expectations, and build stronger relationships with clients. For example, employing techniques such as active listening, empathy, and clear explanations can help manage client emotions and foster a more collaborative approach to care⁷.

Additionally, setting boundaries is essential. Veterinary teams should be trained to recognize when a situation is becoming emotionally charged and know how to de-escalate it or seek support from colleagues. Establishing clear policies for managing client complaints and conflicts can also reduce the stress associated with these interactions.

The wellbeing of the veterinary team is not solely the responsibility of individual professionals; it is also a collective concern that should be addressed at the organizational level. Veterinary practices can play a crucial role in supporting their teams by creating a positive work environment that prioritizes mental health and work-life balance⁸.

Implementing regular team debriefings, where staff can discuss difficult cases and client interactions in a supportive environment, can help alleviate the emotional burden. These sessions can provide an opportunity for team members to share their experiences, seek advice, and gain perspective from their colleagues.

Furthermore, promoting a culture of mutual respect and understanding within the team can foster resilience. When team members feel supported by their peers and management, they are better equipped to handle the stresses associated with client interactions. Offering access to mental health resources, such as counseling services or stress management workshops, can also be beneficial.

Client interactions are an integral part of veterinary practice, but they can significantly impact the wellbeing of the veterinary team. While positive interactions can enhance job satisfaction, negative or challenging encounters can lead to stress, burnout, and compassion fatigue. By fostering effective communication, setting boundaries, and providing organizational support, veterinary practices can mitigate the negative effects of client interactions and promote the overall wellbeing of their teams. In doing so, they not only enhance the quality of care provided to animals but also ensure the long-term sustainability of the veterinary profession.

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LEADING WITH FLEXIBILITY: THE ART OF SITUATIONAL LEADERSHIP IN VETERINARY PRACTICE

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Leadership is a crucial aspect of veterinary medicine, not only for those in management positions but also for every veterinarian, technician, and team member¹. Effective leadership enhances team dynamics, improves patient outcomes, and fosters a positive workplace culture¹.

Situational leadership, introduced by Paul Hersey and Ken Blanchard in the late 1960s, is a flexible and adaptive approach that tailors leadership styles to the needs of individual team members and the demands of specific situations^{2,3}.

By understanding situational leadership, veterinary professionals can lead more effectively, inspire their teams, and navigate the complex challenges of their profession.

Core Principles of Situational Leadership

Situational leadership operates on the premise that there is no single best leadership style^{2,3}. Instead, effective leadership depends on adapting one's approach based on two primary factors: the task's demands and the team's or individual's development level. Hersey and Blanchard's situational leadership model identifies four leadership styles^{2,3,4}:

1. Directing (Telling): High directive and low supportive behavior. The leader provides specific instructions and closely supervises task execution. This style is suitable for team members who are inexperienced or new to a task and need clear guidance.
2. Coaching (Selling): High directive and high supportive behavior. The leader still provides direction but also encourages team members, explaining decisions and providing motivation. This style works well with individuals who are gaining competence but still require encouragement and instruction.
3. Supporting (Participating): Low directive and high supportive behavior. The leader facilitates decision-making and encourages autonomy, offering support rather than explicit direction. This approach is effective for team members who are competent but may need reassurance and encouragement.

4. Delegating: Low directive and low supportive behavior. The leader grants responsibility for task execution to the team members, providing minimal supervision. This style is most appropriate for individuals or teams with high levels of competence and confidence.

These leadership styles are applied in response to the "development level" of the team member, which includes both their competence (skill level) and commitment (motivation and confidence)^{2,3,4}.

In veterinary medicine, situational leadership is particularly valuable because veterinary practices consist of diverse teams, including veterinarians, veterinary technicians, support staff, and even interns or students. Each member has different levels of experience, expertise, and motivation, and the situations they face can range from routine procedures to critical emergencies⁵.

Consider a scenario involving a newly graduated veterinarian. They may have strong theoretical knowledge but limited practical experience. In this case, a directing leadership style is most appropriate. The clinic leader or senior veterinarian should provide clear instructions, establish expectations, and closely monitor their performance while they adjust to the demands of clinical practice.

As the new graduate gains experience and begins to build confidence, the leader can shift to a coaching style. Here, they can offer more detailed explanations behind decisions and allow for some autonomy, while still providing close support. Encouragement is crucial in this phase, as it helps maintain the new veterinarian's motivation during what can be a challenging transition from school to practice.

For competent yet occasionally uncertain team members, a supporting leadership style works well. The leader can involve them in decision-making, encourage their input, and provide emotional support to reinforce their confidence. This participative approach allows competent yet uncertain team members to contribute fully while feeling supported in their roles.

In contrast, for highly experienced and motivated team members who have demonstrated mastery of their tasks, a delegating style might be more suitable. Here, the leader can trust the employee to perform their duties independently, stepping in only when necessary. This autonomy not only empowers the employee but also allows the leader to focus on other priorities within the clinic.

In emergency veterinary situations, such as a critical patient requiring immediate intervention, the leader may need to revert to a directing style regardless of the team member's development level. Time constraints and the need for precise, coordinated action mean that the leader must issue clear, direct commands to ensure the best outcome for the patient. In such high-pressure environments, even experienced team members may benefit from clear, directive leadership to avoid confusion and maintain focus on the critical tasks at hand.

After the emergency has passed, the leader can debrief the team, shifting back to a supporting or coaching style to discuss what went well, what could be improved, and how the team members felt during the situation.

Situational leadership provides several key benefits in the veterinary field:

1. Improved Team Performance: By adapting leadership styles to the needs of individual team members, leaders can maximize productivity and efficiency. For example, new team members receive the guidance they need to develop their skills, while experienced staff can work autonomously without unnecessary oversight.

2. Enhanced Employee Engagement and Retention: When leaders support their teams in the right way, employees feel valued and respected. This leads to higher job satisfaction, reduced turnover, and better morale. Veterinary practices often face high stress and burnout rates, and effective leadership can mitigate these issues.

3. Better Patient Outcomes: Leadership directly influences clinical outcomes. Teams that function well under appropriate leadership are more likely to provide high-quality care, make fewer mistakes, and respond effectively in emergencies. This is especially important in veterinary medicine, where patient welfare is the ultimate priority.

4. Flexibility in Leadership: Situational leadership encourages leaders to be flexible and adaptable. This is vital in the dynamic world of veterinary medicine, where leaders must navigate changing situations, from routine checkups to life-or-death emergencies, while managing a diverse team of individuals with varying skill sets.

While situational leadership offers many advantages, it also presents challenges. It requires leaders to accurately assess the competence and commitment of their team members and be self-aware enough to shift their leadership style accordingly. Leaders may also struggle with balancing different styles for multiple team members simultaneously, especially in a busy practice where situations can change rapidly⁵.

Additionally, it can be difficult to determine the appropriate time to transition between leadership styles, particularly when dealing with subtle changes in team member development. Leaders must remain vigilant, continuously observing their teams and adapting as necessary.

Situational leadership is a powerful tool for veterinary professionals, allowing them to lead their teams effectively by adapting their leadership style to meet the specific needs of each team member and situation.

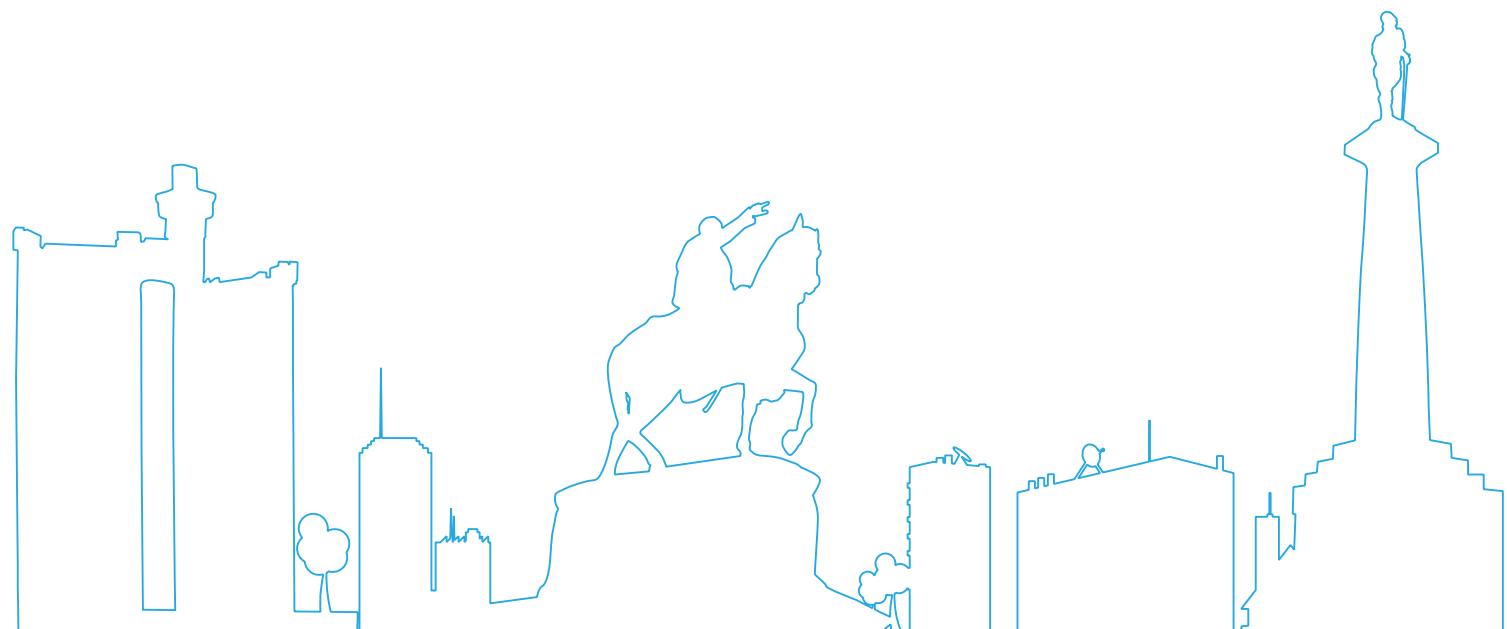
MANAGEMENT

By doing so, veterinary leaders can enhance team performance, boost employee satisfaction, and ultimately improve patient care. As veterinary medicine continues to evolve, the ability to lead with flexibility and responsiveness will become even more critical to the success of veterinary practices and the well-being of both their teams and their patients. By embracing situational leadership, veterinary professionals can foster a culture of growth, support, and excellence in their practices.

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MINIMAL INVASIVE SURGERY AND ENDOSCOPY





Antoine Adam (Switzerland)

DVM

EERVC 2024 Lectures

1. Full laparoscopic gastropexy: my 12 years journey and the technic I use now
2. PerCutaneusCystoLithotripsy (PCCL): minimvasive surgery for bladder and urethral stone diseases management. Can I imagine doing it into my practice?
3. Dog & Cat Middle Ear surgery: how endoscopy can help me?

Associate & Co-founder Vet Clinic Pacy/Eure (France) in 1994.

Founder of Vetmidi SA in 2006, veterinary clinic in St Prex (VD) and a second one in Etoy (VD) in 2017. team of 16 people: 7 vets and 9 nurses.

Vetmidi SA clinic is the most equipped of the French part of Switzerland and they have vet specialist in surgery, medicine and dermatology.

Associate in Swissvetgroup since December 2016. Group of vet clinics in Switzerland.

End of 2017 it's a team of nearly 50 people. 20 vet doctors and nearly 30 nurses.

Abstract

Gastropexy is a crucial preventive surgery for high-risk breeds prone to gastric dilatation-volvulus (GDV), a life-threatening condition where the stomach fills with air and twists. This proceeding presents a comprehensive review of laparoscopic gastropexy techniques, including Laparoscopic-Assisted Gastropexy (LAG), Total Laparoscopic Gastropexy (TLG), and Single-Incision Laparoscopic Surgery (SILS). These minimally invasive techniques significantly reduce postoperative recovery time, pain, and complication rates compared to traditional open surgery. We discuss the outcomes, complications, and success rates of these procedures in preventing GDV, highlighting their growing importance in veterinary surgery. The findings indicate that laparoscopic approaches offer high efficacy, with over 95% of cases successfully preventing GDV recurrence. These advancements in gastropexy surgery enhance patient outcomes and offer promising solutions for veterinarians managing high-risk canine patients.

Introduction

Gastric dilatation-volvulus (GDV) is a medical emergency that commonly affects large, deep-chested dog breeds such as Great Danes, Boxers, and Irish Setters, with an incidence rate of up to 45% in certain breeds. GDV occurs when the stomach fills with air and twists, cutting off the blood supply, which, if untreated, can be fatal. Prophylactic gastropexy, a surgical procedure that attaches the stomach to the abdominal wall to prevent torsion, has evolved to incorporate minimally invasive techniques, improving recovery times and reducing complications.

This proceeding examines three main laparoscopic techniques used in veterinary gastropexy—Laparoscopic-Assisted Gastropexy (LAG), Total Laparoscopic Gastropexy (TLG), and Single-Incision Laparoscopic Surgery (SILS). It compares these techniques in terms of surgical efficiency, recovery time, complication rates, and long-term efficacy in preventing GDV.

Materials and Methods

This review draws from multiple studies focusing on prophylactic gastropexy performed using various laparoscopic techniques. Patient selection was limited to large breeds prone to GDV, such as Great Danes, Boxers, and Setters. Data were collected on surgical times, complication rates, and long-term outcomes, specifically focusing on GDV recurrence and postoperative recovery.

Surgical Techniques

- 1. Laparoscopic-Assisted Gastropexy (LAG):** This technique uses a combination of laparoscopy and a small external incision for suturing the stomach to the abdominal wall. A small incision is made near the 10th or 11th rib, and the stomach is elevated and secured with non-absorbable sutures.
- 2. Total Laparoscopic Gastropexy (TLG):** TLG involves fully laparoscopic manipulation and suturing of the stomach, eliminating the need for external incisions other than trocar sites. The stomach is retracted and sutured to the right abdominal wall using non-absorbable polypropylene.
- 3. Single-Incision Laparoscopic Surgery (SILS):** SILS is a more recent advancement that involves only one incision, minimizing postoperative pain and recovery time.

Results

Across the reviewed studies, all three techniques showed a high success rate in preventing GDV, with efficacy rates of 97-100% for both TLG and LAG. SILS also demonstrated similar outcomes with a 98-100% prevention rate.

- 1. Surgical Time:** SILS had the shortest surgical time, followed by TLG and LAG.
- 2. Complication Rates:** The most common postoperative complications were minor wound infections and transient vomiting. LAG had a complication rate of approximately 10-19%, mostly consisting of self-limiting issues such as seromas and minor wound infections. TLG and SILS showed slightly fewer wound complications due to fewer incision sites.
- 3. Recovery Time:** SILS provided the fastest recovery time, with most dogs resuming normal activities within 7-10 days. LAG had a slightly longer recovery due to additional incisions.

Discussion

Minimally invasive gastropexy techniques offer significant advantages over traditional open procedures. By reducing incision size and surgical trauma, these techniques minimize recovery time and postoperative discomfort. LAG is often recommended for veterinarians transitioning from open to laparoscopic surgery, as it is easier to learn and provides excellent outcomes.

TLG, while requiring more advanced laparoscopic skills, offers even greater benefits, including reduced postoperative pain and faster recovery. SILS, although newer, has shown promise in reducing surgical time and further minimizing postoperative pain and infection risks.

Conclusion

Laparoscopic techniques for gastropexy have proven to be highly effective in preventing GDV, with success rates consistently exceeding 95% across various studies. SILS, TLG, and LAG each offer distinct advantages in terms of recovery, complication rates, and surgical efficiency. Veterinarians should consider adopting these minimally invasive approaches in their practice, particularly for high-risk breeds prone to GDV.

Review Catalog

1. Paramedian Incisional Complications after Prophylactic Laparoscopy-Assisted Gastropexy in 411 Dogs

Authors: Jessica K. Baron, Sue A. Casale, Eric Monnet, et al.

Journal: Veterinary Surgery

Overview: This review examines complications related to paramedian incisions following prophylactic laparoscopy-assisted gastropexy, focusing on 411 cases. It emphasizes the occurrence of seromas and infections, along with long-term outcomes.

2. Outcomes and Complications in a Case Series of 39 Total Laparoscopic Prophylactic Gastropexies Using a Modified Technique

Authors: Veronica Giacopella, Riccardo Grillo, Roberto Giacopella, et al.

Journal: Animals

Overview: The study outlines the outcomes and minor complications observed in 39 cases of total laparoscopic gastropexy using a modified surgical technique.

3. Perioperative Characteristics and Long-term Outcomes Following Prophylactic Total Laparoscopic Gastropexy Using a Novel Knotless Tissue Control Device in 44 Dogs

Authors: Danielle K. Fairfield, Ameet Singh, Andrea Sanchez Lazaro

Journal: CVJ

Overview: This review discusses the use of a novel knotless tissue control device during laparoscopic gastropexy, examining both short-term and long-term outcomes in 44 dogs.

4. Simplified Minimally Invasive Surgical Approach for Prophylactic Laparoscopic Gastropexy in 21 Cases

Author: Cyril Poncet

Journal: Veterinary Surgery

Overview: This article introduces a simplified minimally invasive technique for performing prophylactic laparoscopic gastropexy, focusing on its application in 21 cases.

5. Incisional Gastropexy to Prevent and Treat Canine Gastric Dilatation-Volvulus

Author: Clarence A. Rawlings

Journal: Compendium: Continuing Education for Veterinarians

Overview: This paper reviews the traditional incisional gastropexy approach, used to prevent and treat canine GDV, emphasizing its effectiveness and indications for use.

6. Total Laparoscopic Gastropexy Using One Simple Continuous Barbed Suture Line in 63 Dogs

Authors: Joel D. Takacs, Ameet Singh, J. Brad Case, et al.

Journal: Veterinary Surgery

Overview: This study reviews the use of a continuous barbed suture line in total laparoscopic gastropexy, detailing outcomes in 63 cases.

Abstract

Percutaneous Cystolithotomy (PCCL) is a minimally invasive surgical technique used for the removal of bladder and urethral stones in dogs and cats. This proceeding explores the PCCL procedure, focusing on its advantages over traditional open cystotomy, such as reduced surgical trauma, faster recovery, and fewer postoperative complications. The technique involves creating a small incision and using endoscopic tools to remove uroliths, ensuring minimal damage to the bladder and surrounding tissues. This proceeding discusses the surgical steps, case studies, and outcomes, highlighting PCCL as the gold standard for the management of urolithiasis in small animals.

Introduction

MINIMAL INVASIVE SURGERY AND ENDOSCOPY

Urolithiasis, or the formation of stones in the urinary tract, is a common condition in dogs and cats that can lead to significant discomfort and complications such as urinary obstruction, hematuria, and secondary infections. Traditionally, the removal of these stones involved an open cystotomy, a procedure associated with significant postoperative pain, risk of infection, and longer recovery times. However, the advent of minimally invasive techniques like percutaneous cystolithotomy (PCCL) has revolutionized the approach to stone removal.

PCCL, performed through a small incision using a combination of rigid and flexible endoscopes, offers a less invasive alternative to open surgery. It allows for the removal of bladder and urethral stones with reduced trauma to the surrounding tissues, minimal bleeding, and quicker recovery times.

This proceeding aims to provide an overview of PCCL, its surgical technique, and outcomes, supported by case studies of dogs and cats undergoing the procedure.

Materials and Methods

This review draws from a collection of case studies and research articles documenting the use of PCCL in dogs and cats with bladder and urethral stones. Surgical outcomes, complication rates, and postoperative recovery times were evaluated to determine the effectiveness of PCCL compared to traditional cystotomy.

Surgical Technique

The PCCL procedure involves the following key steps:

- 1. Preparation:** The patient is placed in dorsal recumbency, and the abdomen is clipped and prepared for surgery. Preoperative imaging such as X-rays and ultrasound is used to locate the stones and assess their size and number.
- 2. Incision and Access:** A 1-2 cm midline incision is made cranial to the penis in male dogs. A 5 mm trocar is inserted, allowing for the introduction of a rigid cystoscope into the bladder. In male patients, a flexible cystoscope may also be used to access the urethra.
- 3. Stone Removal:** Stones are removed using a combination of basket forceps and surgical suction. The endoscope provides real-time visualization, ensuring complete stone removal. In some cases, additional tools such as guide wires and hydrophilic catheters may be employed to aid in urethral stone extraction.
- 4. Closure:** Once the stones are removed, the bladder and abdominal incision are closed with minimal suturing, reducing the risk of postoperative complications.

Results

Case studies and clinical trials have demonstrated the advantages of PCCL over traditional open cystotomy:

- 1. Reduced Trauma:** The minimally invasive nature of PCCL causes less trauma to the bladder and urethra, resulting in faster recovery times and less postoperative pain.
- 2. Lower Complication Rates:** PCCL is associated with fewer complications, particularly with regard to bladder integrity and infection. Studies have shown a reduced incidence of stone recurrence and bladder leakage compared to open surgery.
- 3. Faster Recovery:** Patients undergoing PCCL typically experience shorter hospital stays and quicker return to normal activity compared to those who undergo open surgery.

A case study involving a 12-year-old Welsh Corgi named Merlin demonstrated the effectiveness of PCCL in removing large calcium oxalate stones without significant complications. In another case involving a dwarf rabbit named Harry, PCCL was successfully used with the assistance of a holmium laser to fragment and remove bladder stones, illustrating the versatility of the technique.

Discussion

PCCL represents a significant advancement in the management of urolithiasis in veterinary medicine. Its minimally invasive approach minimizes the risks associated with traditional open surgery, including post-operative pain, infection, and prolonged recovery. Moreover, the use of endoscopy ensures complete visualization and removal of all stones, reducing the likelihood of recurrence.

While PCCL requires specialized equipment and training, its benefits far outweigh the challenges, particularly for patients who may be at higher risk for complications from more invasive procedures. As more veterinary practices adopt minimally invasive techniques, PCCL is likely to become the standard of care for the management of bladder and urethral stones in small animals.

Conclusion

Percutaneous cystolithotomy is a minimally invasive, highly effective technique for the removal of bladder and urethral stones in dogs and cats. It offers numerous advantages over traditional cystotomy, including

reduced surgical trauma, faster recovery, and fewer postoperative complications. The technique's versatility, demonstrated in both canine and feline patients, makes it an invaluable tool in the management of urolithiasis in veterinary medicine.

Review Catalog

1. Minimally Invasive Management of Uroliths in Cats and Dogs

Author: Andréanne Cléroux

Journal: Vet Clin North Am Small Anim Pract, 2018

Overview: This review discusses the minimally invasive techniques available for managing uroliths in small animals, including PCCL and its advantages over traditional surgery.

2. Advances in Urinary Tract Endoscopy

Author: Allyson C. Berent

Journal: Vet Clin North Am Small Anim Pract, 2016

Overview: This article covers the advancements in urinary tract endoscopy, focusing on techniques like PCCL for managing urinary stones with minimal invasiveness.

3. Removal of Lower Urinary Tract Stones by Percutaneous Cystolithotomy: 68 Cases (2012-2017)

Authors: Benoît Cruciani, Catherine Vachon, Marilyn Dunn

Journal: Veterinary Surgery, 2020

Overview: A retrospective study analyzing the outcomes of PCCL in 68 cases, emphasizing its efficacy and reduced complication rates compared to open cystotomy.

4. Interventional Urology: Endourology in Small Animal Veterinary Medicine

Author: Allyson C. Berent

Journal: Vet Clin North Am Small Anim Pract, 2015

Overview: This review explores the role of endourology in managing urologic conditions in small animals, highlighting the benefits of PCCL for urolith removal.

5. Comparison of Percutaneous Cystolithotomy and Open Cystotomy for Removal of Urethral and Bladder Uroliths in Dogs: Retrospective Study of 81 Cases (2014-2018)

Authors: Chloé Job, Julie Lecavalier, Marilyn Dunn, et al.

Journal: J Vet Intern Med, 2022

Overview: This study compares the outcomes of PCCL and open cystotomy, showing that PCCL results in fewer postoperative complications and faster recovery.

Abstract

Endoscopic-assisted ear surgery has emerged as a valuable tool in improving outcomes for otologic procedures in veterinary medicine, particularly in cases involving chronic otitis media and external ear canal diseases. This proceeding explores the role of endoscopy in enhancing the accuracy of surgical interventions such as Total Ear Canal Ablation and Lateral Bulla Osteotomy (TECA-LBO), Ventral Bulla Osteotomy (VBO), and related procedures. Through detailed visualization of the tympanic cavity and surrounding structures, endoscopy allows for more precise tissue removal, reduces complications, and helps in the management of residual epithelial tissue. This proceeding discusses the surgical techniques, postoperative outcomes, and complications observed in endoscopic ear surgeries in dogs and cats. By incorporating endoscopy, veterinarians can improve both short-term and long-term surgical outcomes for their patients.

Introduction

Ear diseases, particularly chronic otitis media, are common in dogs and cats and often require surgical intervention when medical treatment fails. Surgeries like Total Ear Canal Ablation with Lateral Bulla Osteotomy (TECA-LBO) and Ventral Bulla Osteotomy (VBO) are frequently performed to alleviate the condition. However, these procedures come with the risk of complications such as facial nerve paralysis, hearing loss, and infection. Endoscopy offers an enhanced ability to visualize and treat ear canal and middle ear structures with minimal invasiveness.

This proceeding will review how endoscopy can be integrated into ear surgery techniques and compare the outcomes of traditional methods with those assisted by endoscopic tools.

Materials and Methods

A retrospective analysis of ear surgeries using endoscopic assistance was conducted on a cohort of dogs and cats suffering from chronic otitis media. Data were gathered from clinical records regarding surgical outcomes, complication rates, and postoperative recovery times. Cases involving TECA-LBO, VBO, and other otologic surgeries were evaluated. The use of rigid endoscopes (1.9 mm) was emphasized in procedures

to assess and improve tissue removal and minimize complications.

Surgical Techniques

- Total Ear Canal Ablation and Lateral Bulla Osteotomy (TECA-LBO):** In this procedure, the entire ear canal is removed, and a portion of the tympanic bulla is opened to allow drainage and debridement. Endoscopy was used intraoperatively to ensure complete removal of epithelial remnants and assess the condition of the bulla.
- Ventral Bulla Osteotomy (VBO):** Primarily used in feline patients with inflammatory polyps or chronic otitis media, this technique involves opening the bulla through a ventral incision. Endoscopic assistance allowed better access to the medial and lateral compartments of the bulla.
- Endoscopic-Assisted Myringotomy:** This procedure was performed in cases of otitis media to drain the middle ear fluid, using the endoscope to guide needle placement and avoid damaging critical structures like the facial nerve.

Results

The integration of endoscopy into ear surgery procedures provided several benefits:

- Improved Visualization:** Endoscopy enhanced the ability to view critical anatomical structures, including the facial nerve, chorda tympani, and ossicles, reducing the risk of iatrogenic injury.
- Reduced Complication Rates:** Facial nerve deficits were reported in fewer cases compared to traditional methods. Residual epithelial tissue, a common cause of postoperative infection, was significantly reduced in the endoscopic-assisted TECA-LBO group.
- Enhanced Postoperative Outcomes:** The endoscopic approach led to faster recovery times, with fewer long-term complications such as draining tracts or Horner's syndrome in cats undergoing VBO.
- Detection of Residual Tissue:** Endoscopy was effective in identifying epithelial remnants, which are often left behind in conventional surgeries. This helped in reducing the incidence of infection and fistulation postoperatively.

Discussion

Endoscopic-assisted ear surgeries offer a significant improvement over traditional techniques by providing enhanced visualization, reducing the likelihood of leaving residual disease-causing tissue, and minimizing postoperative complications. The use of endoscopy is particularly beneficial in complex cases of chronic otitis media and polyps, where accurate and thorough removal of diseased tissue is crucial for preventing recurrence.

While the learning curve for endoscopy can be steep, its benefits in improving outcomes make it a valuable tool for veterinarians performing ear surgeries. Future studies could further quantify the long-term benefits of incorporating endoscopy into routine otologic procedures.

Conclusion

Endoscopy significantly improves the outcomes of ear surgeries in veterinary practice. Its use in procedures such as TECA-LBO and VBO enhances visualization, reduces complications, and leads to better long-term results for patients suffering from chronic ear diseases. Incorporating endoscopic techniques should be considered a best practice in managing otologic conditions in dogs and cats.

Review Catalog

1. Complications and Surgical Outcome in Dogs with Otitis Media Undergoing Total Ear Canal Ablation and Lateral Bulla Osteotomy: A Retrospective Study of 37 Cases

Author: Ana Sofia Relvas Nazaré

Journal: Universidade de Lisboa

Overview: This study examines the complications and outcomes of 37 cases of TECA-LBO in dogs with chronic otitis media, focusing on facial nerve injuries and infection rates.

2. Managing Recurrent Otitis Externa in Dogs: What Have We Learned and What Can We Do Better?

Author: Tim Nuttall

Journal: JAVMA

Overview: A comprehensive review of the management of recurrent otitis externa, highlighting new treatment strategies and surgical options to improve outcomes in affected dogs.

3. Anatomic Structures of the Canine Middle Ear Visible During Endoscopic Examination Through

MINIMAL INVASIVE SURGERY AND ENDOSCOPY

a Ventral or Lateral Approach

Authors: Emily C. Viani, Caleb C. Hudson, Kristin A. Coleman, et al.

Journal: JAVMA

Overview: This paper provides an anatomical guide to the structures visible during endoscopic middle ear surgery, emphasizing the benefits of using endoscopy in both ventral and lateral approaches.

4. Auditory and Neurologic Effects Associated with Ventral Bulla Osteotomy for Removal of Inflammatory Polyps or Nasopharyngeal Masses in Cats

Authors: Brendan B. Anders, Michael G. Hoelzler, Thomas D. Scavelli, et al.

Journal: JAVMA

Overview: The study evaluates the neurologic effects and hearing outcomes in cats undergoing ventral bulla osteotomy for the treatment of middle ear masses.

5. Otoscopic Evaluation of Epithelial Remnants in the Tympanic Cavity After Total Ear Canal Ablation and Lateral Bulla Osteotomy

Authors: Meghan M. Watt, Penny J. Regier, Cassio R. A. Ferrigno, et al.

Journal: Veterinary Surgery

Overview: This review discusses the use of endoscopy in assessing epithelial remnants post-TECA-LBO and the implications for postoperative infection and healing.

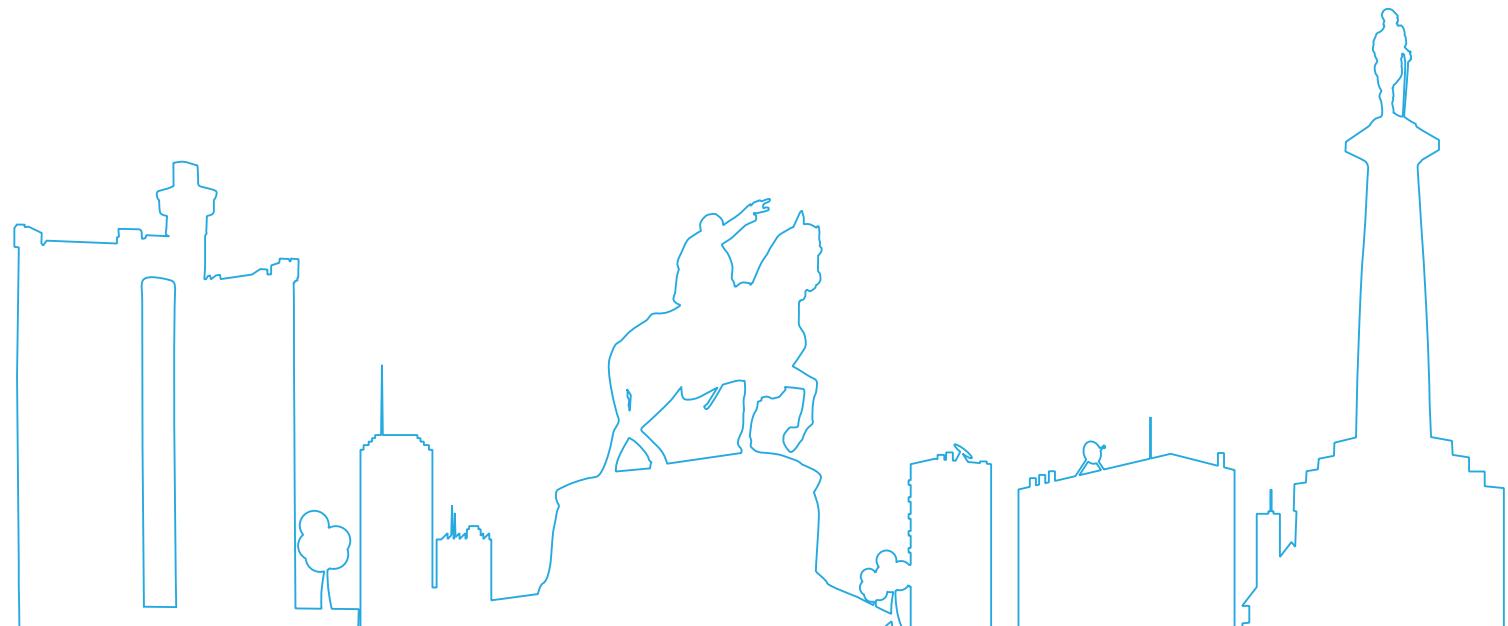
6. Effect of Empirical Versus Definitive Antimicrobial Selection on Postoperative Complications in Dogs and Cats Undergoing Total Ear Canal Ablation with Lateral Bulla Osteotomy: 120 Cases (2009-2019)

Authors: Christian A. Folk, Cassie N. Lux, Xiaocun Sun, et al.

Journal: JAVMA

Overview: This study compares the outcomes of empirical versus culture-based antimicrobial treatments in reducing postoperative complications in TECA-LBO surgeries.

NEUROLOGY



**Thomas Flegel (Germany)**

Diplomate ACVIM (Neurology), Diplomate ECVN

EERVC 2024 Lectures

1. 5 shades of intervertebral disc diseases
2. It looks like a seizure, it smells like a seizure, it tastes like a seizure, but it is not a seizure: paroxysmal dyskinesia
3. How to identify 3 common neurological presentations without being a neurologist
4. Peripheral nervous system lesions – wrongly neglected by most of us: how to recognise them?

Veterinary Training

1986-1992 – Humboldt-University Berlin, Germany

Working Experience

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FIVE SHADES OF INTERVERTEBRAL DISC DISEASE

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Canine intervertebral disc disease (IVDD) has been classified for decades based on the innovative work by Hans-Jørgen Hansen published in 1952, who differentiated between type I and II based on histological characteristics. This presentation will follow this classification as well. However, the list of types of IVDD will have to be extending including three additional types that have been discovered more recently since the availability of magnetic resonance imaging in veterinary clinical practice.

Hansen Type I (Extrusion)

Dogs of chondrodystrophic breeds are predominately affected by Hansen type I IVDD. Those dogs suffer from early degeneration of the annulus fibrosus and nucleus pulposus that is histologically detectable at less than one year of age. This degenerated intervertebral disc may not be able to withstand pressure forces that develop during physiological movements. Therefore, the annulus fibrosus may rupture and the degenerated, often mineralized nucleus pulposus extrudes into the vertebral canal compressing the spinal cord.

Most affected chondrodystrophic breeds have a relatively long back and short limbs such as Dachshunds, Pekingese, Havanese, Bassets, Shih Tzu, Lhasa Apso. However, there are other chondrodystrophic breeds with more normal body dimensions such as French Bulldogs, Cocker Spaniels or Beagles. Therefore, the long back is not the underlying pathology of an intervertebral disc extrusion but just an additional risk factor. Altogether, Dachshunds and French Bulldogs are most frequently affected, however Dachshund are usually older (mean: 6 years) than French Bulldogs (mean: 4 years) when developing clinical signs of IVDD. Clinical signs of type I IVDD are usually acute, however, they may become chronic in a few conservatively treated cases.

Hansen Type II (Protrusion)

This type of IVDD develops from a degenerated disc similar as in type I IVDD, even though the histological characteristics of both types are different, resulting a different clinical presentation. Clinical signs develop in the second half of the patient's life and they are slowly progressive in nature. Those progressive signs are caused by a weakening of the dorsal annulus fibrosus, that slowly bulges more and more into the vertebral canal finally compressing the spinal cord. Because of the slowly progressive compression of the spinal cord, the latter can compensate for the compression for quite a while, before neurological deficits become obvious. Therefore, spinal cord compression is often severe, whereas the patient may exhibit relatively mild neurological deficits only. At some point, however, the compensatory capacity of the spinal cord is exhausted and progression of clinical signs may develop within a short period of time. Successful surgical treatment in such a chronically compressed spinal cord is less likely than in acute type I IVDD with the same degree of spinal cord compression.

Most frequently affected breeds are non-chondrodystrophic breed, even though any breed can develop this type of IVDD. Sometimes both types of IVDD may coexist in one single intervertebral disc.

Type III (ANNPE)

The following types have not been described by Hansen, therefore I do not call them Hansen type III to V, but just type III to V. Some purists among neurologist refuse to keep counting and use more descriptive terms instead. Here, we will use both terminologies.

Different than in Hansen type I and II, type III IVDD develops from a normal non-degenerated intervertebral disc that contains of a fluid or gel-like nucleus pulposus. This disc disease often develops during exercise, when the annulus may develop a small fissure. The fluid-like nucleus is pushed with a high pressure and a high velocity through that fissure into the spinal canal, where it may hit the spinal cord causing a contusion and/or concussion. The compressive component can be neglected, since the fluid can disperse in the spinal canal. The acronym ANNPE stands for acute non-compressive nucleus pulposus extrusion and it describes well the underlying pathological process. Sometimes, the terms "small volume – high velocity – extrusion" or "traumatic disc extrusion" are used as well.

Non-chondrodystrophic breeds are most commonly affected by type III IVDD since it requires a non-degenerated disc to develop. Chondrodystrophic dogs suffer from early disc degeneration and therefore they are less likely to develop type III IVDD. An age predisposition does not really exist, even though it may become less likely in older dogs.

Type IV (HNPE)

Type IV IVDD develops, similar as type III, from a non-degenerated intervertebral disc. And similar, the non-degenerated nucleus pulposus extrudes through a small fissure in the dorsal annulus fibrosus. However, the fluid-like extruded nucleus is still covered by a thin membrane (most likely originating from the dorsal longitudinal ligament). That membrane prevents the fluid from dispersing in the spinal canal. Therefore, this fluid causes acute spinal cord compression similar has it happens in Hansen type I IVDD. The acronym HNPE stands for hydrated nucleus pulposus extrusion. This type of disc disease develops in dogs of non-chondrodstrophic breeds, since it requires a non-degenerated disc.

Type V (IINPE)

This type of IVDD can develop from a degenerated as well as from a non-degenerated intervertebral disc. The either fluid-like or mineralized nucleus pulposus peracutely extrudes through a rupture of the dorsal annulus fibrosus with such a tremendous velocity that it can penetrate the dura mater like a projectile causing significant intramedullary spinal cord damage (hemorrhage, laceration). Those lesions are usually associated with severe neurological deficits and they carry a poor prognosis.

Treatment of intervertebral disc disease

In general, IVDD can be treated conservatively (exercise restriction, physiotherapy, pain medication) or surgically. The latter is recommended in cases, where IVDD did result in a non-ambulatory status, in cases with severe spinal pain is non responsive to pain medication or for cases, where conservative treatment failed. However, surgery is indicated for those types of IVDD only that cause spinal cord compression (types I, II, IV). In contrast, type III IVDD is not treated surgical independent of the severity of clinical signs, since it does not cause spinal cord compression. Studies have shown that the compressive type IV IVDD can be treated surgically or conservatively with a similar success rate, even though it is a compressive type.¹ In type V IVDD surgical removal of the intramedullary disc material can be attempted, however, surgery may cause additional parenchymal damage and therefore a surgical treatment should be critically discussed with the owner.

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IT LOOKS LIKE A SEIZURE, IT SMELLS LIKE A SEIZURE, IT TASTES LIKE A SEIZURE, BUT IT IS NOT A SEIZURE: PAROXYSMAL DYSKINESIA

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Paroxysmal dyskinias (PD) are characterized by a sudden onset of abnormal limb movements lasting for just a few minutes. For decades, those episodes have been interpreted as partial epileptic seizures, before this entirely different disease, the PD, was identified by veterinary neurologist more recently. Clinicians should be aware of differences in the clinical signs between partial seizures and PD, since diagnostic work-up and therapeutic interventions are different for both diseases.

Introduction

PD are characterized by sudden, paroxysmal, abnormal motor activity (decreased or increased) in patients with normal or nearly normal mentation, which are lacking typical electroencephalographic abnormal activity that is characteristic of seizures.¹ Without an EEG, it can sometimes be challenging to differentiate between partial epileptic seizures and PD just based on clinical sign. However, there are some characteristics that should raise your suspicion that a patient may be suffering from PD:

- only a few muscle groups of one or two limbs are affected
- normal mentation during those episode
- no autonomic signs
- no pre or post ictal signs
- variable duration (seconds to minutes)
- self-limiting
- large variation in frequency in one patient (several times a day to just a few in a year)
- no neurological deficits between episodes

Specific breed-specific clinical signs have been described for the following canine breeds: Bichon Frisé, Border Terrier, Boxer, Cavalier King Charles Spaniel, Chinook, German Pointer, Jack Russel Terrier, Labrador Retriever, Maltese, Markiesje, Norwich Terrier, Scottish Terrier, Sheltie, Soft-coated Wheaton Terrier, Welsh Terrier und Yorkshire Terrier. However, based on the increased number of reports in recent years, it is probably safe to assume that any breed can be affected.

The cause of PD is most likely a genetic defect in most dogs, however structural intracranial diseases can trigger PD as well. Therefore, diagnostics mainly aims at exclusion of structural intracranial pathologies, since genetic defects are known for a few breeds only (Soft-coated Wheaton Terrier, Cavalier King Charles Spaniel, Markiesje).

In the following typical clinical signs are described for breeds, where PD has been studied more extensively:

Epileptoid Cramping Syndrome in Border Terriers

Episodes of PD in affected dogs usually develop at an age of less than 3 years (range: 6 weeks to 7 years). The duration of PD episodes varies between 2 and 30 minutes (less commonly hours) at a very variable frequency. Clinical signs may include generally increased extensor tone in all limbs and in neck muscles, often in combination with chorea, tremor, and athetosis. The mentation is usually normal, even though patients appear to be irritated by what's going on. Some clients report increased borborygm, diarrhea and/or vomiting as well. In half of those patients, gastrointestinal signs appear in close time relationship to the episodes of PD.² Antibodies against transglutaminase 2 and gliadin can be found in serum of affected dogs. Therefore, gluten sensitivity has been suspected as underlying pathology.³ Feeding a gluten-free diet does result in complete resolution of clinical signs in many dogs, whereas it does decrease the frequency of PD episodes in others.

Episodic Falling in Cavalier King Charles Spaniels

PD episode in Cavalier King Charles Spaniel start at an age between 3 months and 4 years. They are often induced by stress. Frequency and duration can be very variable. Clinical signs during those episodes include progressive hypertonicity of front and rear limbs, as well as neck muscle. Clinical signs can be so severe that dogs suddenly fall over resulting leading to the descriptive name of the disease: episodic falling. Dogs may cross their front limbs over their head once they have fallen.⁴ Normal mentation and a lack of cyanotic membranes may help to differentiate those episodes from seizures and syncopes. A genetic defect affecting the brain specific proteoglycan brevican has been identified in affected dogs. A commercial genetic test is available and it can be used to diagnose the disease as well for breeding purposes.⁵ A generally effective treatment is not known. However, improvement of clinical signs can be achieved in some dogs using clonazepam or azetazolamide.

Paroxysmal dyskinesia in Maltese

Episodes of PD start in young adult dogs at an average age of 5 years (range 1 – 11 years). The frequency of episodes is very variable. Some dogs show clusters within days, followed by periods of months without any event. Episodes can develop from situations of rest as well in moments of stress at any time of the day. Half of the clients report some abnormalities that sound similar as a preictal period in epileptic seizures such as: seeking attention of owner, before typical symptoms of PD start: hypertonicity of one or several limbs, generalized tremor, which interferes with the ability to walk normally during those episodes. One quarter of affected dogs exhibits increased salivation as well.⁶ Azetazolamide or fluoxetine may decrease the frequency of PD episodes, even though not all dogs may respond to those drugs. Gluten-free diet may help to decrease the frequency and severity.

Paroxysmal dyskinesia in Norwich Terriers

PD episodes start at an average age of 3 years (range 9 months – 6 years). They can happen at any time of the day, but most of them start if the dog is stressed or if it is excited. In some patients, changes in weather conditions seem to trigger PD episodes. Most owners believe that their dogs experience some abnormal

sensation right before the onset of an episode. Clinical signs consist of increased muscle tone of one or both rear limbs. In about 2/3 of dogs the front limbs or abdominal muscle or tail muscles are affected as well. Rear limbs are often flexed underneath the abdomen. Dogs exhibit rear limb swaying, whereas front limbs are affected less frequently by this sign. Half of the dogs are so severely affected that they lose their ability to maintain a standing position. The duration is variable, but episode usually last between 2 and 30 minutes. Mentation is usually normal during those episodes. Neither any medication nor dietary changes seem to influence the frequency.⁷

Scotty Cramp in Scottish Terriers

PD episodes usually start at an early age, often between 6 and 8 weeks of age. Dogs exhibit exercise induced increased extensor muscle tone of rear limbs. Clinical signs are so severe in some dogs that they are not able to ambulate anymore. An autosomal recessive genetic defect has been suspected, but has not been identified yet. In mildly affected dog oral treatment using methysergide may decrease severity of clinical signs. Diazepam can be tried alternatively.

Diagnostics

There is an increasing number of clinical reports in many different canine breeds. In several of those dogs, PD seems to be associated with a gluten hypersensitivity as described for Border terriers. In a recent paper, gluten antibodies were described in 18 canine breeds.⁸ There are more breeds developing PD in association with gluten antibodies based on our own experiences. Therefore, testing serum for antibodies against transglutaminase 2 and gliadin should be one of the first diagnostic steps in every dog with suspected PD. In cases, where clinical signs do not allow to differentiate between PD and partial seizures, an electroencephalogram (EEG) is indicated. Ideally, EEG is performed during one of those episodes, but typical EEG abnormalities can be found in 29 % of patients with seizures even in the interictal period.⁹ A complete diagnostic work-up similar as in a dog with seizures is recommended in any dog, that does not have a breed specific genetic defect, were gluten serum antibodies cannot be found or were a gluten free diet does not result in any clinical improvement.

Treatment

The following medication can be tried in cases of suspected PD. However, the response to any of those drugs is difficult to predict:

fluoxetine:	2 – 4 mg/kg SID
levetiracetam:	20 mg/kg TID
acetazolamide:	4 mg/kg BID to TID
clonazepam:	0,5 mg/kg BID to TID

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HOW TO IDENTIFY 3 COMMON NEUROLOGICAL PRESENTATIONS WITHOUT BEING A NEUROLOGIST

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Many clinicians capitulate before to complexity of the nervous system and therefore they try to avoid dealing with patients with neurological diseases. Those cases are frequently referred to specialized neurological referral centers. That is a valid approach. However, this talk aims at convincing you that some neurological conditions can be diagnosed and treated by virtually everybody, even if you do not have any detailed knowledge of the nervous system pathophysiology, the neurological examination or the treatment. We will use a pattern recognition approach in three different scenarios of clinical signs.

Peracute, highly lateralized loss of motor function in rear limbs in a dog

The following clinical presentation is very indicative for a **Fibrocartilaginous Embolism** (FCE):

- peracute loss of motor function in rear limbs
- neurological deficits are very lateralized, so that one limb can barely be moved and the other is close to normal
- signs developed suddenly during exercise, when patient cried out in pain
- dog is not painful anymore during your examination

FCE causes peracute to acute paralysis by occluding arterial or venous blood vessels within the spinal cord by fibrocartilaginous material leading to sudden ischemia and consequently to neurological deficits. Depending on size and location of the affected blood vessels different neurological symptoms may develop. FCE has a high prevalence in young adult to middle aged dogs of larger breeds (80% weigh 20-25 kg or more), even though it may affect any breed at nearly any age. However, two smaller breeds, the Sheltie and the Miniature Schnauzer, may be more often affected than other small breed dogs.

The typical clinical history includes sudden paralysis during exercise (playing, jumping, running). Many patients cry out in pain in the initial moment, but they are non-painful once they are presented to the veterinarian. Neurological signs may progress within the first 24 hours in some cases, but progression is usually restricted to the first minutes or the first hour. Depending on the location of the embolism along the spine, only rear limbs or all four limbs may be affected. The most frequent localisations are: Th3-L3: 33%, L4-S3 32%. In many cases, neurological signs are asymmetric to a degree, which would not be likely with any compressive spinal cord lesion. The severity of neurological deficits may vary from mild ambulatory paresis to paraplegia without deep pain perception.

There is no test to diagnose FCE with certainty *in vivo*. However, ruling out other potential causes may allow a very likely presumptive diagnosis. The one single test to diagnose the disease would be spinal MRI, where a typical intraspinal signal hyperintensity can be seen on T2 weighted images. However, for establishing a strong presumptive diagnosis MRI may not really be necessary. It might be sufficient to rule out any compressive lesion using myelography in a patient with typical signalment and clinical history. Therefore, the following approach can be recommended. FCE is very likely if the following criteria are fulfilled: young adult to middle aged large breed dog, acute paralysis during exercise, no progression of clinical signs beyond the first hour, no pain on spinal palpation, no extradural compression on myelography. The major differential diagnosis in those case would be acute non-compressive nucleus pulposus extrusion.

There is no specific treatment beside supportive care and physiotherapy. Anecdotic, FCE has been treated using propentofylline. In general, prognosis is usually fair with two thirds of dogs recovering. Significant improvement is usually seen within two weeks. Negative prognostic indicators are: bilateral neurological deficits, involvement of lower motor neurons, plegia without deep pain perception.

Droopy lower jaw in a dog

The following clinical presentation is very indicative for an **Idiopathic Trigeminal Neuropathy**:

- owners report inability to eat and drink
- inability to actively close the mouth
- no resistance to passive closure of the mouth
- sometimes in combination with complete loss of facial sensation
- excessive salivation

The trigeminal has three branches that supply motor function to masticatory muscles as well as sensation to most of the face. Sensory deficits may go along unrecognized, whereas bilateral loss of masticatory motor function interferes with eating and drinking. In about 90% of dogs with trigeminal nerve deficits, those clinical signs are caused by idiopathic trigeminal neuropathy.¹ All of those dogs exhibit bilateral loss of masticatory muscle function (however, an unrecognized number of dogs might have unilateral disease only), whereas 35 % have additional sensory deficits. A small number of dogs may experience facial nerve deficits as well Horner's syndrome (loss of sympathetic innervation of the eye). There is no specific treatment known, except bandaging the patient's mouth to substitute for masticatory muscle function in order to allow the dog to take in food and water. Glucocorticoids do not shorten the course of the disease. The average time to spontaneous recovery is about 3 weeks.

Vestibular syndrome plus Horner's syndrome and/or loss of facial sensation

The following clinical presentation is very indicative for a **Middle/Inner Ear Disease**:

- vestibular syndrome (ataxia, head tilt, nystagmus) in combination with one or both of the following:
 - o in the eye of the lower side (head tilt): smaller pupil, droopy upper eye lid, third eye lid protrusion
 - o loss of all facial movements (blinking, lip retraction)

Vestibular deficits are a common presenting complaint in small animal practice. Those patients are rewarding for the "neurological beginner" since they are easy to identify. Therefore, the first step in establishing the suspected diagnosis of a vestibular lesion is usually straight forward, if a patient is presented with generalized lack of limb coordination, head tilt, nystagmus. The second and may be more important step, however, localizing the problem to the peripheral (ear) or to the central parts (brain) of the vestibular system, can be slightly more demanding. However, the combination of clinical signs provided above will clearly indicate a peripheral vestibular lesion, where the underlying pathology has to be expected in the inner ear. Two nerves, the sympathetic nerve and the facial nerve travel in very close proximity to the inner and middle ear and therefore, they can be affected in inner/middle ear pathologies.

Loss of sympathetic innervation of the eye does cause clinical signs of a Horner's syndrome: miosis (smaller pupil), ptosis (droopy upper eye lid), enophthalmus (sunken in eye ball), and third eye lid protrusion, whereas facial nerve deficits cause a loss of facial muscle function that are necessary for facial expressions (loss of menace response, loss of palpebral reflex, droopy lip and loss of lip retraction).

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PERIPHERAL NERVOUS SYSTEM LESIONS - WRONGLY NEGLECTED BY MOST OF US: HOW TO RECOGNIZE THEM?

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Patients with lesions affecting the peripheral nervous system can look quite similar as patients with spinal cord lesions. In addition, most clinicians are more familiar with spinal cord pathologies than with those affecting the peripheral nervous system and therefore those are suspected first if a patient is presented with neurological deficits resulting in an inability to walk. However, it is crucial to be able to differentiate between both, since differential diagnoses, diagnostics work-up, treatment and prognosis differ significantly between both groups of pathologies.

This presentation aims at recognizing signs of peripheral nervous system diseases. As in most diseases, clinical signs in peripheral nervous system disease can vary significantly based on the severity of the disease. We will focus on the most severe cases, in which patients lost their ability to ambulate due to peripheral nervous system disease.

Patients presented for peripheral nervous system (PNS) lesions exhibit normal mentation and behavior. Cranial nerve examination may reveal deficits in several patients with generalized PNS lesions (since cranial nerves are peripheral nerves to) as well as in patients with intracranial diseases and therefore, those deficits cannot be used for differentiation between a disease of the central or peripheral nervous system. The optic nerve is an exception. This nerve is rather an extension of the brain carrying more characteristics of the central nervous system than of a peripheral nerve. Therefore, cranial nerve II (optic nerve) is usually not affected by generalized PNS diseases, whereas involvement of other cranial nerves may lead to dysphonia, laryngeal paralysis, difficulties prehending and swallowing food as well as facial nerve deficits.

Patients with generalized PNS lesions may show a combination of sensory and motor deficits affecting their limbs: ataxia, conscious proprioceptive deficits and paresis/plegia. Diseases purely affecting the sensory nerves cause ataxia and conscious proprioceptive deficits without paresis, whereas diseases purely affecting the motor nerves causing paresis/plegia. In most cases sensory and motor deficits coexist in peripheral nervous system disease. Motor deficits in generalized peripheral neuropathy can be pronounced, often being much more severe than in spinal cord disease. Paresis/plegia is usually flaccid. Neurogenic muscle atrophy can commonly be appreciated within one week. All those described above are relatively unspecific can be seen with lesions in different parts of the nervous system.

However, there are other clinical signs that will guide you towards a peripheral nervous system disease. Most generalized peripheral nervous system diseases affect all limbs based on the nature of the underlying pathology. However, clinical signs often start in the rear limbs and progress to the front limbs within a few days. Such a progression of clinical signs from pure rear limb deficits to a tetraparesis or tetraplegia is difficult to be explained by any type of spinal cord pathology. Therefore, this progression is very indicative for a peripheral nervous system disease. Testing segmental spinal reflexes should be the next diagnostic step in those cases. Many patients with peripheral nervous system diseases may have generalized reduced segmental spinal reflexes in all limbs, with the flexor reflexes often being most significantly affected. Therefore, the following sentence is crucial for recognizing peripheral nervous system disease: **Tetraparesis or tetraplegia with loss of segmental spinal reflexes in all four limbs is nearly always caused by a peripheral nervous system pathology.** However, it does not have to involve all segmental spinal reflexes, but you should find at least one reduced reflex in the rear limbs and in the front limbs as well in order to raise the suspicion of a peripheral nervous system pathology.

In addition, owners of dogs with peripheral nervous system disease may report a voice change, a loss of voice or hoarseness of their pet. That is caused by a laryngeal paralysis due to recurrent laryngeal nerve involvement. Sometimes, this hoarseness may be the first clinical signs of peripheral nervous system disease, since those pathologies may affect the longest nerves first and the recurrent laryngeal nerve is one of those.

In cases, where neurological deficits affecting the sensory or motor function are not convincingly enough, taking chest radiographs might be helpful, because patient that are tetraparetic or tetraplegic because PNS disease may develop a megaesophagus, whereas those with spinal cord disease usually don not. Screen chest radiographs with megaesophagus carefully for secondary aspiration pneumonia, which is most frequently seen in the right middle lung lobe.

The two most common differential diagnoses for dogs with PNS disease are botulism and polyradiculoneuritis, even so a large variety of other differentials may have to be considered: hypothyroidism, hyperadrenocorticism, intoxication (organophosphates, carbamates, algae), fulminant myasthenia gravis. Rabies may present with a similar ascending paralysis starting in rear, even though it is ultimately affecting the central nervous system.

Botulism

Botulism is an often fatal disease characterized by muscular paralysis. It develops after ingestion of pre-formed botulinum toxin produced by the anaerobic bacterium *Clostridium botulinum* (less common *Clostridium baratii*, *Clostridium butyricum*). Toxins produced have different antigenic properties allowing differentiating between 7 different subtypes (A-F). In human beings, botulism is mainly caused by serotypes A, B, E whereas in dogs it is caused by serotype C and rarely type D.

The bacterium is ubiquitous and it can be found in soil, water and in the gastrointestinal tract of mammals and fish. In dogs, botulism may develop after ingesting carcasses or inadequately stored food or by drinking from contaminated ponds. The toxin is absorbed from the small intestine by endocytosis. It enters the lymphatic systems and it is distributed via the blood stream. It binds rapidly and irreversibly to the neuronal surface of the pre-synaptical terminal of the neuromuscular junction. After being internalized, it modifies the SNARE-protein, which is responsible for acetylcholine release at the neuromuscular endplate. The impairment of acetylcholine release causes the typical flaccid paralysis.

Clinical symptoms

Botulism is characterized by a rapidly progressing paralysis, often starting in the rear limbs. The more rapidly the symptoms develop, the more severe the disease tends to be. Clinical signs may occur within hours to several days following ingestion of toxin. Clinical signs reflect a progressive, symmetrical disorder, ranging from mild weakness to severe flaccid tetraplegia with absent spinal reflexes. Those signs may be accompanied by weakness in muscles of the face, jaw, pharynx, and esophagus resulting in dysphonia, dysphagia, facial paralysis, and megaesophagus. Mydriasis may be present. Pain perception remains normal and there is no evidence of hyperesthesia. Muscle atrophy is not seen. In severe cases, respiratory musculature might be affected resulting in a predominately diaphragmatic respiration.

Sometimes, signs of autonomic dysfunction (mainly parasympathetic) such as changes in heart rate, mydriasis, keratoconjunctivitis sicca, urinary retention and constipation can be seen.

Diagnosis

History and clinical symptoms are often suggestive of botulism. Definitive diagnosis is based on toxin detection early in the disease (i.e. within 24 h) in blood, feces or gastrointestinal tract contents. Often a mouse biological assay is used for toxin detection. Due to the nature of the test, it may take several weeks to obtain a result. Alternatively, ELISA and PCR can be used but those are not generally available yet in veterinary medicine.

Electrodiagnostic tests can be used to support a tentative diagnosis. EMG changes are rather mild and are dominated by fibrillation potentials developing after 2 weeks of disease. M-wave amplitudes may be markedly reduced, whereas motor nerve conduction velocity is normal or only mildly reduced. Most helpful are changes on repetitive nerve stimulation. A slight decrement can be seen after low frequency stimulation (< 5 Hz), whereas an increment may be induced after high frequency stimulation (50 Hz). The latter is highly suggestive of botulism.

Therapy and prognosis

Therapy is mainly supportive: soft bedding, urinary catheter, avoiding pressure sores, feeding. Antitoxin application is discussed controversially. It will bind circulating toxin and therefore should be given early in the disease. The available trivalent antitoxin, however, acts against subtypes A, B and E. There is no commercially available antitoxin against the C, the one causing botulism in dogs. The prognosis is usually favourable in dogs, with recovery occurring within 1 to 3 weeks, although some affected dogs are euthanized due to other clinical complications or respiratory failure. Therefore, careful monitoring for signs of respiratory insufficiency is indicated.

Idiopathic Polyradiculoneuritis

Polyradiculoneuritis, generalized inflammation of nerve roots, may actually be a group of various etiopathological different diseases. In the context of this presentation, we will focus on Idiopathic Polyradiculoneuritis. It is mainly seen in dogs (much less common in cats). It is a suspected immune mediated disease, most likely triggered by different immunological stimuli, one of those presumably being raccoon saliva. Lesions involving nerve roots (more obvious in ventral than in dorsal roots) and the most proximal part of peripheral nerves are composed of segmental demyelination and mononuclear interstitial infiltration.

The disease affects dogs of any breed, both sexes, and usually of adult age. In the original descriptions of this disease, clinical signs frequently appeared 7 to 11 days after an encounter with a raccoon. Presence of circulating antibodies against raccoon saliva has been demonstrated in some dogs using ELISA assays. Nowadays, it is thought that different immunological stimuli may trigger the immune response leading to polyradiculoneuritis.

Onset is marked by weakness and pelvic limb hyporeflexia, although thoracic limb involvement may sometimes be the initial and dominant clinical sign. Paralysis progresses rapidly, resulting in a flaccid symmetric tetraplegia; however, milder forms without paralysis can occur. The duration of paralysis varies from several weeks to 2 or 3 months. Motor impairment is more pronounced than sensory changes, although many dogs appear to be hyperesthetic to sensory stimuli. Bladder and rectal paralysis are not usually observed. In severely affected animals, there may be complete absence of spinal reflexes, facial weakness, loss of voice, inability to lift the head, and labored respiration. Megaeosphagus seems to be much less common than in botulism.

Respiratory insufficiency may be seen if phrenic nerves and/or intercostal nerves are affected as well.

Diagnosis

EMG changes are more pronounced than those seen in botulism. There is extensive spontaneous muscle activity (fibrillation potentials, positive sharp waves) within 5 to 7 days after the onset of clinical signs. Those findings are sometimes more obvious in distal limb muscles, which has been explained by the preferential involvement of longer nerves associated with their greater chance of being affected by a multifocal demyelinating process. Motor nerve conduction velocity may be markedly reduced and M-wave temporal dispersion can be seen in many dogs. F-waves can be altered (e.g., prolonged F-wave latencies and F-wave dispersion, decreased F-wave amplitudes, increased F-wave ratio), depending on clinical signs and duration of disease reflecting that the primary pathology is located in nerve roots.

Cerebrospinal fluid analysis may reveal mild albumin-cytological dissociation (normal nucleated cell count, increased total protein concentration). Nerve and muscle biopsies taken from routine peripheral biopsy sites are usually unremarkable or may exhibit unspecific findings. In order to establish a diagnosis based on a biopsy, a sample has to be taken from a nerve root, which is a rather invasive procedure.

The following infections should be ruled out as underlying pathology of infectious polyradiculoneuritis in a given case (especially in those with mild clinical symptoms): toxoplasmosis, neosporosis, borreliosis.

Therapy and Prognosis

Therapy is mainly supportive similar as in botulism. Glucocorticosteroids do not seem to alter course of the disease. Patients with obvious hyperesthesia may benefit from specific medication for neurogenic pain:

- Gabapentin: 5-10 mg/TID
- Pregabalin: 2-4 mg/kg BID

Intravenous application of human immunoglobuline (0.5 g/kg once or 0.5 g/kg on 3 consecutive days) can hasten recovery but may be cost prohibitive in most dogs.¹

Prognosis is usually favorable, but dogs with severe axonal degeneration may die from respiratory paralysis or may have protracted, incomplete recoveries. Some animals may not show any clinical improvement. The overall course of the disease may be 4-8 weeks. However, recovery may take up to 6 months. Relapses are possible but rare.

References

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EERVC 2024 Lectures

1. No need to panic – confident neurological emergencies management
2. Easy peasy bladder squeezy – diagnosis and management of lumbosacral disease
3. Ace of spines – diagnosis of acute non-surgical myelopathies in first opinion practice
4. Epilepsy beyond seizures and drug treatment

Holger is currently Professor of Small Animal Diseases and the Head of Department of Small Animal Medicine and Surgery, University of Veterinary Medicine Hanover, Honorary Professor of Veterinary Neurology and Neurosurgery, Royal Veterinary College, London and Affiliate Professor of Veterinary Neurology, University of Copenhagen. He graduated from the University of Veterinary Medicine Hanover in 2001, where he also did his PhD in Neuropharmacology studying basic mechanisms of drug-resistant epilepsy. He then completed his specialist clinical education doing an internship and a residency in Neurology and Neurosurgery at the Royal Veterinary College (RVC). The RVC also provided him with the chance to not only excel academically and clinically, but also in his leadership skills, going through the reigns from lecturer to head of service, clinical director of the Small Animal Referral clinic and last as head of department of clinical science and services. Holger is internationally known for his work in the field of SARS-CoV-2 Medical Scent Detection Dogs, neuropathic pain and epilepsy. He was President of the European College of Veterinary Neurology and active in the Executive Board of Veterinary Specialisation as treasurer. He has been a recipient of several Jim Bee educator excellent in teaching awards, Gerhard-Domagk-Award, Bourgelat Award from BSAVA, the International Canine Health Award from the Kennel Club, and the RCVS International Award.

NO NEED TO PANIC – CONFIDENT NEUROLOGICAL EMERGENCIES MANAGEMENT

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Here we will discuss about some of the neurological emergencies we have not covered yet today. Head and spinal traumas are not uncommon. They appear to be very challenging and are difficult cases to assess as a complete physical and neurological examination is usually not possible. Manipulating the patient is not advised as it is often not immediate apparent if the animal has a single or multiple injury such as a neck and thoracolumbar spine fracture.

Traffic accidents are the most common cause for head trauma, but they can also occur secondary to a fall, horse kick, dog or cat bite or penetrating objects...

Understanding pathophysiology can help to manage these cases.

There is primary and secondary damage. The primary damage is caused by the impact of the trauma and has happened when you see the animal. The animal might have a skull fracture, contusions, laceration or intracranial haemorrhages.

This starts the process of secondary damage. This is a very complex process, but is also the target for treatment. In general, secondary damage is a cascade of events associated with progressive hypoxic / ischaemic brain injury which lead to increase in intracranial pressure (ICP).



The ischaemia leads to cytotoxic and vasogenic oedema which then cause a more severe ischaemia and so on. The blood and oxygen supply to the brain is dependent on the cerebral perfusion pressure. The cerebral perfusion pressure is dependent on the mean arterial pressure and ICP. Thus, make sure you monitor the blood pressure in animals with head trauma. The same applies for dogs in which you suspect an infarct.

In these cases the mean arterial blood pressure can increase to improve blood brain perfusion. Monitor also the dog for an increase in ICP. The increase in ICP will cause the brain to herniate caudally and will cause neurological deficits which you can pick up in your neuro exam.

Assess and Re-assess

- Assess & Reassess.
- Mentation.
- Pupils.
- Oculovestibular Reflex.
- Posture & Motor Function.
- Deep pain sensation.
- Blood pressure and heart rate.

1. Mentation

Mentation will change with an increase in ICP. The animal will become first obtunded, then stuporous (rousable by testing deep pain) and finally comatous.

2. Pupil size

Assess pupil size and function doing the papillary light reflex. Make sure that your lightsource is as bright that you have a dazzle reflex when shining the light in your own eye.

3. Occulovestibular reflex

The occulovestibular reflex is using information from the vestibular system and proprioceptive information from the cervical region to maintain the eye's position at first. The eye muscles become then stretched and this proprioceptive information will cause a jerky movement towards the side of head movement. This reflex is a **brainstem reflex**. The projection fibres between the vestibular system and the nuclei for the cranial nerves is deep in the brainstem and therefore an indicator for deeper brainstem damage.

4. Posture and Motor function:

There are different levels of function loss. First the animal will lose its proprioceptive functions, then motor function and then finally deep pain sensation. In an **unconscious patient** do not manipulate the spine or head position as this could cause further trauma.

The patient might have a fracture you have not yet identified. If the animal is **recumbent** watch out for voluntary movement and test the spinal reflexes. If the animal is **ambulatory** test the postural reactions.

5. Deep pain perception

Test deep pain perception if the animal is non-responsive and has a lack of movement. It makes no sense to test deep pain perception in an animal that can walk. It will only cause them harm.

6. Blood pressure and heart rate

Monitoring blood pressure is not only important to ensure a good perfusion of the brainstem, but also to assess the brainstem function. A brainstem dysfunction can cause the Cushing response. Cushing's response (a compensatory response that attempts to provide adequate cerebral perfusion pressure in the presence of rising ICP) presents as a rising systolic pressure, a widening pulse pressure, and bradycardia and is a late presentation of brainstem dysfunction. The next level of dysfunction will be a change in respiratory pattern in addition to the Cushing response.

Use the modified Glasgow Coma Scale for monitoring initially every 30 minutes. Look out for trends in level of consciousness, brainstem reflexes and motor activities.

1. Assess if the animal is cardiovascular stable (ACB, Airway, Breathing, Circulation).
2. Give oxygen & place IV.
3. Treat seizures as they can also increase your ICP.
4. Neurological assessment and Glasgow Coma Scheme.
5. Monitor blood pressure and heart rate.
6. Complete your physical examination (Polytrauma assessment).
7. Get a history from the owner (when did it happen? how did it happen? Any improvement or deterioration?).

Diagnostics

- PCV / TP
- Pulse Ox/Blood Gases
- Blood Pressure
- Survey Radiography
- Ultrasound
- Skull Radiography
- Computed Tomography

Look at the CT on the right side. Good or bad prognosis?

Imaging does not prognosticate assess the animals' function (Glasgow Coma Scheme).

Medical management

As we discussed earlier the primary impact is in the past, but the cascade causing secondary damage can be tackled. The ultimate aim is to treat and prevent further secondary brain injury (i.e. ICP).

HYPOXAEMIA and HYPOTENSION are Strongly Correlated with ↑ICP · Aim for normotension and normal oxygenation.

- FLUID THERAPY (Volume limiting is strictly contraindicated and will worsen brain and spinal cord injury.)

OXYGEN THERAPY

o Monitor:

- Pulse oximetry – $\text{SaO}_2 > 95\%$.
- Arterial blood gases - $\text{PaO}_2 > 80 \text{ mmHg} (> 100 \text{ mmHg cats})$.
- Ventilate the animal if necessary:
 - See graph on the right-side cerebral blood flow in respect of O_2 , CO_2 and blood pressure.
 - Keep $\text{CO}_2 < 35 \text{ mmHg}$ to decrease blood volume and therefore ICP.
- HEAD ELEVATION. This facilitates:
 - Displacement of CSF to cervical spine.
 - Venous drainage.
 - Decreases excessive cerebral blood flow. o 30° Optimal. o Maximal ↓ in ICP. o Minimal compromise in CBF.
- If increased ICP o Give MANNITOL.
 - Mechanism of action:
- Osmotic diuretic.
- Multiple proposed mechanisms that ↓ICP.
- Reflex vasoconstriction.
- Osmotic action.
- Free-radical scavenging. · ↓CSF Production.
 - Dose:
- 0.5-1.0g/kg over 10-20mins.
- q3-6hrs.
- (max 3/24hrs).
- Monitor:
 - Hydration.
 - Electrolytes
 - Osmolality if possible (Keep <320mOsm/L).
- NO CORTICOSTEROIDS.

But more later on....

Consider surgical management if animals get worse despite medical management and animal is not stable for transport.

Spinal trauma

Spinal trauma has a lot of similarities to head trauma. There is the initial impact (primary damage) and the subsequent following secondary damage. The aim is again to limit the secondary damage.

Assume that a non-ambulatory animal hit by a car coming to your practice has an unstable spine. Do your initial assessment as described before, but make sure it does not move. As soon you can de-mobilize the animal on a spinal board. It does not have to be anything fancy, but x-ray beams should be able to penetrate it. On the left you can see a dog with a C3 fracture after running into a tree. It is quite common that when dogs run into an object that they fracture their higher cervical spine (especially greyhounds and lurchers).

Neurological assessment and prognostication

1. Determine the neuroanatomical **localization**.
2. **Severity.** I. Pain only
 - II. Pain and ambulatory paresis/ataxia
 - III. Non-ambulatory paresis
 - IV. Plegia with deep pain perception
 - V. Plegia with loss of deep pain perception

Grade V has a grave prognosis.

Primary injury

The primary injury has completed at the time of presentation. In spinal trauma similar to brain trauma the trauma causes laceration, contusion and compression. The velocity or the impact of the trauma is the most important factor for trauma. The degree of compression and the time of compression is not as important.

Secondary injury

The same as in brain injury:

The aim therefore is to reduce the secondary damage. Consider decompression and stabilization.

Action plan

1. Assess if the animal is cardiovascular stable (ACB, Airway, Breathing, Circulation).
2. Give oxygen & place IV.
3. Complete your physical examination (Polytrauma assessment).
4. Neurological assessment (when animal is cardiovascular stable).
 - a. Consider multiple lesions (be conscious about the "weak" points of the spinehigh cervical, thoracolumbar and lumbosacral).
 - b. Lower motor neuron lesions can mask upper motor neuron lesions.
 - c. Give opioids after assessing deep pain perception.
5. Monitor blood pressure.
6. Get a history from the owner (When did it happen? How did it happen? Any improvement or deterioration?)

Diagnostics

- PCV / TP
- Pulse Ox/Blood Gases
- Blood Pressure
- Survey Radiography
 - o **DO NOT TURN PATIENT** o Perform conscious
 - o Radiograph entire spine – multiple lesions
 - o **Radiographs do not prognosticate – your neuro-exam does**
- Computed Tomography
 - o Not all lesions are easily visualized on radiographs. We CT the whole spine and hip nowadays as your neurological exam is limited by the amount you can manipulate the patient. Therefore, always consider multiple lesions

Medical management:

- Aim for normotension and normal oxygenation
- **Corticosteroids are no longer recommended**

Surgical management:

- Decompression
- Rigid Fixation:
 - o Advantages
 - No casts/bandages
 - Physiotherapy

Status epilepticus

Status epilepticus (SE) is a life-threatening neurologic emergency characterized by prolonged seizure activity. Histopathological brain damage can be seen in humans and animals after a 30 minutes seizure activity. However, we know from human medicine that a generalized seizure activity which lasts longer than 10 minutes is unlikely to stop on its own without medical treatment. Cluster seizures are defined as two or more seizures in a 24 hours period.

Prolonged seizure activity of 30 minutes causes primary brain damage and secondary complications can occur. Primary brain damage is caused by multiple causes such as excitotoxicity, hypoxaemia and ischaemia. Excitotoxicity is secondary to excessive activation of NMDA receptors causing an excessive influx of calcium ions. This leads to activation of the apoptosis cascade. The described events lead to brain necrosis and cerebral oedema, which can lead to herniation.

After 30 minutes of SE dogs develop hypertension, increased cerebral blood flow, hypoxaemia, hypercarbaemia, hyperglycaemia and lactic acidosis. If the seizure activity continues the blood pressure drops, hyperthermia, hypoglycaemia, myolysis, cardiac arrhythmias, and hyperkalaemia can occur. This can be even further aggravated by aggressive treatment with antiepileptic drugs, which decreases the blood pressure and reduces heart and respiratory rates. This can lead to organ failure. If there are clinical signs of increased intracranial pressure mannitol 1g/kg over 20 minutes is indicated.

Pharmacokinetic considerations during SE

The prolonged seizure activity can affect both the peripheral and central pharmacokinetic of drugs. Especially the fall in pH of the blood has an effect on the ionization of drugs affecting their half-lives and blood brain barrier permeability. Blood pH decreases to a greater degree than brain pH; this facilitates the movement of weak acids from the blood to the brain such as PB. Furthermore, the blood brain barrier has been described to be more leaky during an SE.

The goals of treatment:

1. **Stop the seizure!**
2. **Protect the brain!**
3. **Think about the future!**

Stabilise the patient.

First airways, breathing and circulatory support has to be established. The animal's blood oxygenation should be assessed with a pulse oximeter and/or arterial blood gas analysis. Bloods should be tested for electrolytes (sodium, potassium, calcium, chloride), blood glucose levels, PCV and total protein. We also routinely send bloods away for CBC, biochemistry and drug serum level (if indicated). The animal should be oxygenated, the blood pressure measured, treated with i.v. fluids such as 0.9% NaCl 10 ml/kg/hr. The rectal temperature should be closely monitored and hyperthermia should be minimised.

We start with diazepam (0.5 mg/kg IV [1 mg/kg if on PB]). This can be repeated three times and should never exceed a total dose of 3 mg/kg. High doses of diazepam aggravate the hypoxaemia. In cases where an iv catheter is difficult to place diazepam can be applied rectally 1 mg/kg (2 mg/kg if on PB). Diazepam works nearly immediately.

PB can take up to 20 minutes to be efficacious. Diazepam's effect lasts around 30 minutes. We therefore often start the animal on PB straightaway. Start drug-naïve animals: Load up to 20 mg/kg in 24 hrs (this can be divided into multiple doses and should not exceed 100 mg/min). If the animal is on PB, administer 1 mg/kg for each µg/ml of desired increase in the patient. Do not increase the dose too rapidly. We try to increase PB by 5 µg/ml each time.

Alternatively, one can use a midazolam continuous rate infusion, which has less cardiorespiratory side effects than diazepam.

Risk with benzodiazepine usage:

Diazepam adverse effects have to be known to be mastered. It also has to be considered that the adverse effect can last longer than the anticonvulsant activity: Respiratory depression, hypotension and cardio-respiratory collapse.

Maintenance therapy

Institute maintenance PB treatment for sustained antiepileptic effect.

If the animal did not respond to PB, we recommend starting the dog on potassium bromide or the cat on levetiracetam (see also chapter above for more details).

Treat recurrent seizure activity

If the seizures continue despite the aforementioned treatment, it becomes difficult to find the right drug. After 30 minutes the GABA receptors change their subunit composition leading to drug refractoriness; for example there is a lack of the benzodiazepine receptor. Furthermore, diazepam binds to GABA receptors and increases the potency of GABA. Thus, without GABA diazepam has no effect. After prolonged seizure activity GABA is often depleted. Phenobarbitone works for longer as it increases GABA's efficacy. Also drugs like phenytoin do not work after sustained SE and is not as GABA dependent actively open the GABA channel. Levetiracetam has a unique mechanism of action and was therefore proposed for refractory SE. Human studies however have been not promising.

The NMDA antagonist ketamine is most likely the most promising alternative.

One can try Diazepam CRI – 0.5 mg/kg/hr or the less cardiovascular compromising drug midazolam CRI – 0.3 mg/kg/hr. One can also try propofol: 4-8 mg/kg i.v. slow to effect followed by 4-12 mg/kg/hr or Levetiracetam: 60 mg/kg i.v. then 20 mg/kg TID. The dose for ketamine is 5 mg/kg i.v. bolus then 5 mg/kg/hr CRI.

The difficulty for all drugs mentioned is how does one determine the cessation of seizure activity. The only reliable method is EEG. Otherwise the animal has to be very well observed.

Be aware when you use propofol, that when it is weaned off too quickly contractures can occur, which can be mistaken with seizures.

Prolonged anaesthesia

When everything has failed, one can only try to anaesthetise the animal for a prolonged period (minimum 12 hrs).

We have recently published a consensus statement about SE treatment where you can find many more information: Charalambous M, Muñana K, Patterson EE, Platt SR, Volk HA. ACVIM Consensus Statement on the management of status epilepticus and cluster seizures in dogs and cats. J Vet Intern Med. 2024 Jan-Feb;38(1):19-40. doi: 10.1111/jvim.16928



In summary:

In conclusion, SE is a serious, life-threatening disorder that requires rapid intervention. A preemptive treatment protocol will stop the SE and help to improve the seizure frequency in the future.

EASY PEASY BLADDER SQUEEZY – DIAGNOSIS AND MANAGEMENT OF LUMBOSACRAL DISEASE

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Lumbosacral disease is very common in large breed dogs. The first sign the owner usually notice is reluctance of the dog to jump into the car or go upstairs. Dogs can also show paresis ("lameness"), which should be considered when orthopaedic disease has been ruled out (see further details below).

Lumbosacral disease is characterised by a stenosis of the vertebral canal and/or intervertebral foramina and/or the related vasculature. The cauda equina (L6-7, S1-3, Cd1-5) and/or nerve roots leaving via the vertebral foraminae can be compressed by various abnormalities:

1. Hansen type II disc degeneration and protrusion at the lumbosacral junction.
2. Thickening and in-folding of the interarcuate ligament.
3. Subluxation of the articular facets.
4. Epidural fibrosis.
5. Thickened lamina and pedicles.
6. Spondylosis, proliferative degenerative changes of the articular facets.
7. Instability and misalignment between the last lumbar vertebra and the sacrum.

Large breed dogs, especially German Shepherd dogs, are most commonly affected. Breed specific anatomic conformation and abnormalities in lumbosacral motion are suspected to cause lumbosacral disease.

Diagnosis

Lumbosacral stenosis is most common in large breed dogs (GSD), with a female to male ratio of 2:1. The most common presentation is spinal pain, difficulties rising or jumping. On neurological examination a sciatic nerve lesion can be often identified, leading to a reduced withdrawal (hock flexion decreased) and pseudo-hyperreflexia of the patella reflex. Pseudohyperreflexia: The patella reflex looks increased due to

the fact that the opposing caudal limb muscles innervated by the sciatic nerve are reduced in tone. The tail can be floppy in some cases. Faecal and urinary incontinence (lower motor neuron bladder) can occur and have been associated with a poorer outcome.

Some dogs also show signs of a nerve root signature such as lameness or lifting the limb.

Foraminal stenosis can also cause intermittent neurogenic claudication, as blood vessels cannot swell during exercise.

Radiographs of the lumbosacral area can reveal spondylosis deformans, disk space narrowing, and end-plate sclerosis. There may be evidence of lumbosacral fracture/luxation, osseous neoplasia, diskospondylitis, or congenital lumbosacral stenosis. In dogs with lumbosacral osteochondrosis, a radiolucent defect occurs in the dorsal aspect of the affected end-plate along with one or more bone fragments in the vertebral canal and sclerosis of the dorsal part of the end-plate. Stress radiography, such as dynamic flexion/extension studies, may accentuate the lumbosacral instability.

Epidurography and diskography can help to identify the compression in around 90% of the cases. Myelography is not as useful as the dural sac is physiologically already elevated at the LS junction.

Advanced imaging such as CT or MRI are probably the diagnostic procedures of choice. MRI can clearly reveal soft tissue, such as cauda equina, epidural fat, and intervertebral disk. CT scans on the other hand are useful to evaluate the vertebral foramina. However, the degree of compression seen on MRI is not necessarily correlated to the clinical presentation.

Electrophysiological studies can give further functional insight into the severity of the disease, but can also be normal in dogs with L/S disease.

Treatment

Dogs with urinary and faecal incontinence have a poor prognosis. Otherwise the treatment has to target the observed pathology. In general, conservative management (pain relief, rest and later physiotherapy) can stabilize the condition. We start in most of the cases with rest for at least eight weeks, before we introduce slowly lead exercise again. If this fails we then consider surgical decompression of the cauda equina or foraminectomy. Often dogs are significantly better after surgery, however if not rested appropriately after surgery, soft tissue proliferation can occur causing a secondary compressive lesion.

ACE OF SPINES – DIAGNOSIS OF ACUTE NON-SURGICAL MYELOPATHIES IN FIRST OPINION PRACTICE

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Spinal diseases can be roughly divided into diseases, which are either myelopathic or nonmyelopathic. Non-myelopathic diseases present usually as painful conditions without causing neurological deficits. Which structures surrounding the spinal cord can cause pain? Please colour the structures, which you think could be painful if they were affected by a tumour or inflammation/infection.

Primary bone tumours in the spine are rare. Secondary tumours such as locally invasive tumour (such as prostatic carcinoma) or metastasis (such as lymphoma or osteosarcoma) are more common.

Inflammatory conditions of the articular facets are normally associated with a generalised polyarthritis. See below for a more detailed description of some diseases which can present without causing a myelopathy. Differentiating between causes of gait abnormalities in practice can be challenging. However, by initially defining the problem and the system involved, a list of further appropriate diagnostic tests can be performed. Despite the recent advances in diagnostic imaging, the neurological and orthopaedic examinations remain the foundation of localising the lesion and help identify severity. The majority of cats and dogs that present with a thoracic or pelvic limb lameness will have an underlying orthopaedic condition, but it is important to recognise that neurological disorders can present with similar clinical signs. Neurological disorders will more commonly present as decreased voluntary movement (paresis) or lack of voluntary movement (plegia), but again, these clinical signs are not synonymous with neurological disorders. Once the location of the lesion has been defined, a list of differential diagnoses can be formulated based on

onset, clinical course and clinical features such as pain and asymmetry of clinical signs (five finger rule) + signalment. Each individual case has its own challenges, and any purely rule-based system is likely to result in mistakes. We will discuss various cases and have a live on stage discussions which we hope will help you tackle these challenging cases better in the future.

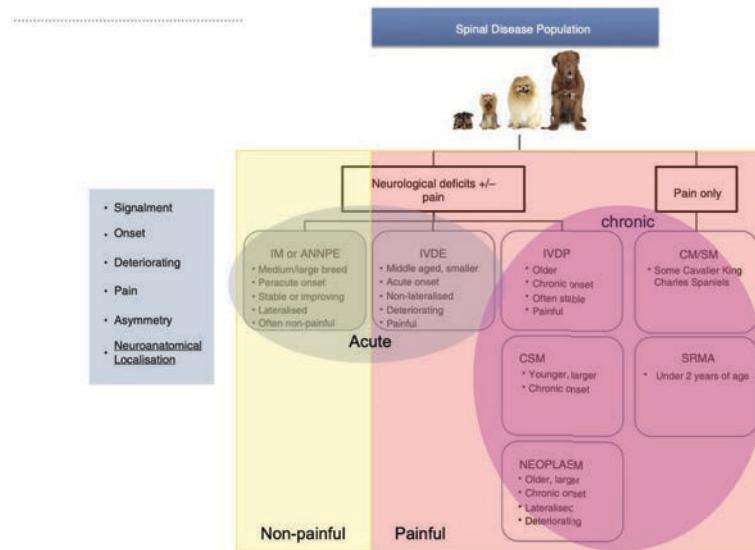
The majority of patients who present with gait abnormalities will have abnormalities that primarily and structurally affect either the musculoskeletal or the nervous systems. As a result, lesion localisation will concentrate on lesions affecting the musculoskeletal and neurological systems. Unlike the cardiovascular, metabolic and respiratory body systems, there is no simple laboratory or diagnostic test that can be performed to differentiate between the nervous and musculoskeletal body systems. The differentiation between the two systems is based on the clinician's physical examination. Most animals with neurological disorders will present with paresis, while those with orthopaedic disorders will present with lameness.

As aforementioned for neurological diseases it is important to differentiate between diseases which cause only pain and no neurological deficits (painful non-myelopathic spinal diseases) and diseases which cause neurological deficits (myelopathic spinal diseases). **Non-myelopathic spinal diseases-** Animals that solely present with back pain and do not show neurological deficits need to have a thorough orthopaedic examination, as polyarthritis has to be considered. Other differentials are inflammatory, infectious and neoplastic diseases. If the animal presents with a history of trauma, luxation and fractures need to be considered. As aforementioned, syringomyelia is an exception and can present as a painful condition without causing neurological deficits.

Myelopathic spinal diseases - The five-finger rule (onset, progression, pain, lateralisation and neuroanatomical localisation) can be used to effectively differentiate between myelopathies these myelopathies. A couple of examples are listed as follows:

- Patients who present with peracute, non-progressive or improving, largely non-painful and lateralised neurological deficits (most often in T3-L3 spinal cord segments) have a 98% chance of having an ischaemic myelopathy such as fibrocartilaginous embolism (FCE) or acute nucleus pulposus extrusion (high velocity but low volume disc extrusion/traumatic disc).
- Hansen type-I disc disease (intervertebral disc extrusion) is best characterised as an acute onset, deteriorating, painful and occasionally lateralised myelopathy (often at T3-L3 spinal cord segments). Ninety percent of patients presenting with these clinical signs will have Hansen type-I disease.
- In contrast, Hansen type-II (intervertebral disc protrusion) has a more chronic onset, is often stable, but still painful.
- Meningo(encephalo)myelitis of unknown aetiology (MUA) can present with an acute onset, deteriorating painful myelopathy. MUA is four times more likely to present as a multifocal neuroanatomical localisation (multiple spinal cord segments and/or brain). Many of the animals will also have mentation changes and cranial nerve deficits.

These examples demonstrate that thinking pathophysiologically and using the five-finger rule can refine the differential list significantly. If you then also take demographics and signalment into account, you have a very high chance of identifying the most likely diagnosis before embarking on diagnostics. Many of the neurological conditions will require advanced imaging and/or CSF analysis, but funds are limited, and the aforementioned approach can provide you with the framework to narrow down diagnostics to the most essential or provide the owner with a presumptive diagnosis.



Differentials for spinal cord diseases (A = adult & aged dog; Y=young dog)

Category	Acute nonprogressive	Acute progressive	Chronic progressive
Degenerative		Type I disc disease (A, Y)	Cervical Spondylomyelopathy (A,Y) Type II disk disease (A) Degenerative myelopathy (A) Spondylosis deformans (A) Demyelinating diseases (Y) Axonopathies and neuronopathies (Y) Extradural synovial cysts (A) Breed specific myelopathies (such as Afghan hound myelopathy (Y) Storage disease (Y)
Anomalous			Chiari-like malformation& Syringomyelia (Y, A) Vertebral anomalies (Y) Atlantoaxial luxation (Y) Spinal dysraphisms (Y)
Neoplastic		Primary (A) Metastatic (A) Skeletal (A)	Nephroblastoma (Y) Primary (A) Metastatic (A) Skeletal (A)
Nutritional			Hypervitaminosis A (Y,A)
Inflammatory / infectious		Distemper (Y,A) FIP (Y) Protozoal (Y) GME (A) Bacterial myelitis (Y,A) Discospondylitis (Y,A)	Distemper (Y,A) FIP (Y) Protozoal (Y) GME (A)
Traumatic	Fractures (Y,A) Luxations (Y,A) Contusions (Y,A) Traumatic disk, ANNPE,HNPE (Y,A)	Traumatic disk (Y,A)	
Vascular	Infarction (FCE; Y,A) Septic emboli (Y,A) Hemorrhage (Y,A) Vascular malformations (Y)		

EPILEPSY BEYOND SEIZURES AND DRUG TREATMENT

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The most common chronic neurological brain disease in both people and dogs is epilepsy, with an estimated prevalence of 0.4-1% (Cowan, 2002; Sander and Shorvon, 1996) in humans and 0.6-2% in dogs (Schwartz-Porsche, 1986; Kearsley-Fleet et al., 2013; Heske et al., 2014). Over twenty percent of human patients with epilepsy will continue to experience seizures despite chronic medical treatment with two adequate antiepileptic drugs (AEDs) (Picot et al., 2008). In the case of dogs with suspected idiopathic epilepsy, the remission rate varies from 20-80%, depending on the examined dog population (hospital vs. primary care) (Heynold et al., 1997; Berendt et al., 2002; Arrol et al., 2012; Boothe et al., 2012). Approximately 20-30% of these dogs will remain inadequately controlled (with less than a 50% reduction in seizure frequency) despite adequate treatment with standard AEDs such as phenobarbital (PB) and/or potassium bromide (KBr) (Trepanier et al., 1998; Schwartz-Porsche et al., 1985; Podell and Fenner, 1993).

Epilepsy is more than just a pure seizure disorder (De Risio et al., 2015). Dogs suffering from epilepsy, especially drug-refractory epilepsy characterized by a high seizure frequency and cluster seizures, are at an increased risk of developing behavior changes, cognitive dysfunction, premature death, reduced quantity of life, and a diminished owner-perceived quality of life (QoL) (Berendt et al., 2007; Shihab et al., 2011; Chang et al., 2006; Wessmann et al., 2012; Packer & Volk, 2015). Behavioral changes, the effects of recurrent seizure activity, and anti-epileptic drugs can significantly impact the QoL of affected dogs, leading to issues such as injury, disorientation, fear, anxiety, restlessness, polyphagia, and polydipsia/polyuria. Moreover, there is potentially a high burden on the owners of dogs with epilepsy, negatively affecting their QoL as well. This burden includes dealing with unpredictable and uncontrollable seizure activity, administering medications multiple times a day, and making emergency vet visits (Wessmann et al., 2012; Chang et al., 2006). The owner's perception of the dog's QoL is especially affected by a high seizure frequency and when the dog is on more than two drugs (Wessmann et al., 2016). On the other hand, the owner's QoL is mainly influenced by the sedation and ataxia level of their AED-treated dog with epilepsy. It is not surprising, but relevant, that reductions in perceived dog QoL scores are associated with reductions in owner QoL, and vice versa. Understanding what influences QoL is of utmost importance. Podell et al. (2016) state in the ACVIM Small Animal Consensus statement on seizure management that the most important outcome measure is QoL, as this will ultimately affect the owner's decision-making regarding the care of a pet with epilepsy or, ultimately, whether the pet's life is worth living. Owners may choose euthanasia if caring for a pet with epilepsy becomes too emotionally stressful, financially challenging, or impacts the owner's own psychosocial environment. Some owners describe having an epileptic pet as "living with a ticking time bomb" (Pergande et al., 2020). Therefore, it is important to understand how different management options are tolerated and how they influence the QoL of the affected pet and owner.

Key strategies for reducing stress and improving QoL for both the owner and the dog include considering the influence of the environment, diet, comorbidities, and antiepileptic treatment. Some reports have indicated the importance of diets, such as the ketogenic diet, hypoallergenic diet, and fatty acid supplementation, as new or alternative treatment strategies for canine epilepsy. Food supplementation with Omega-3 has shown inconclusive results (Matthews et al., 2012). There is some anecdotal evidence that a hypoallergenic diet might improve seizure control in dogs with gastrointestinal hypersensitivity (Lujan et al., 2004). However, a traditional high-fat, low-carb/protein ketogenic diet failed to improve seizure control (Patterson et al., 2005). A more promising ketogenic diet is based on medium-chain triglycerides, which has improved seizure control in the majority of cases (Law et al., 2015).

Another key to successful epilepsy management is pet owner education. The better educated the pet owner is about epilepsy, its comorbidities, and antiepileptic treatment (including side effects, pharmacodynamics, and pharmacokinetics), the better they will be equipped to live with the condition successfully and assist in the care of the patient. This includes actively monitoring seizure frequency, which can be done using a paper or electronic seizure diary with an app (Bhatti et al., 2015). It's worth noting that only one

in five owners administers medication correctly (Booth et al., 2021), which may partially explain less successful outcomes. Owners can also help reduce environmental stress factors and assist with tailoring drug treatment to the dog's seizure status (Packer et al., 2015). The principle of comprehensive epilepsy care is that "every little helps" to increase the seizure threshold and, therefore, improve epilepsy management.

Conceptually, epilepsy is not a single disease with a unique and simple pathophysiology. The term encompasses a heterogeneous group of chronic conditions with seizures as their clinical manifestation. This complexity arises from the intricate structure of the brain, which can only function and dysfunction in certain limited ways. Hence, a wide variety of disturbances in brain structure and function can result in seizures and epilepsy. Similar pathophysiological pathways are thought to contribute not only to epilepsy but also to other neurodevelopmental and neurobehavioral disorders (Johnson and Shorvon, 2011; Shihab et al., 2011). These disorders share pathophysiological commonalities at various levels. Understanding both the shared and distinct pathophysiological pathways can improve our understanding of these diseases and guide appropriate diagnosis, monitoring, and treatment. Furthermore, characterizing these disorders pharmacologically and understanding why treatment fails in some cases can enhance our knowledge of the mechanisms behind drug resistance and how to overcome them. This, in turn, can deepen our understanding of their pathophysiology and natural clinical course. Moreover, understanding comorbidities of epilepsy, such as behavioral abnormalities, might offer new treatment angles, which could improve the QoL of affected dogs and potentially enhance seizure control.

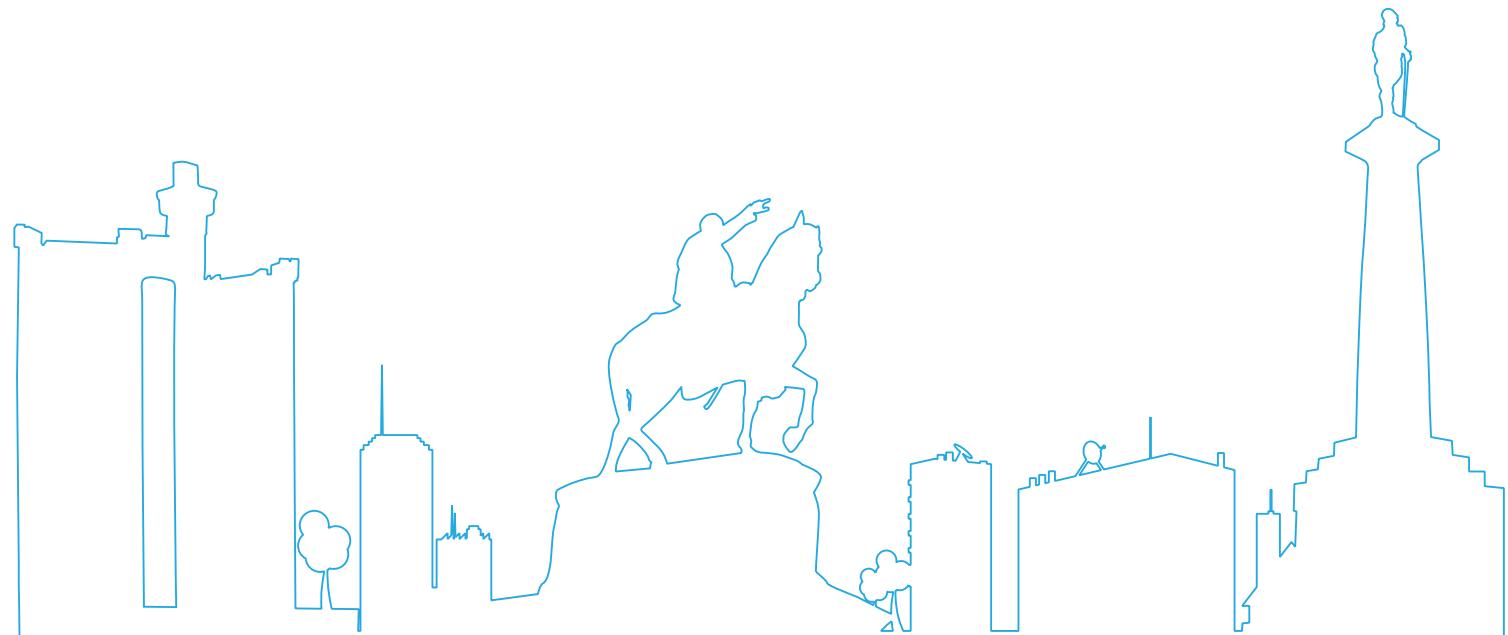
Shihab et al. (2011) were the first to describe an increase in fear/anxiety and defensive aggression behavior in dogs with idiopathic epilepsy. In the Lagotto Romagnolo dog, a benign "childhood" or juvenile epilepsy form has been described, with the first seizures occurring around 6 weeks of age (Jokinen et al., 2007). Nearly all affected dogs go into remission at around 10 weeks of age without receiving medical treatment. However, Jokinen and colleagues (2015) found that despite these dogs remaining seizure-free, they exhibited increased inattention and excitability/impulsivity behavior compared to controls. ADHD-type behavior has also been described in a recent study from the UK (Packer et al., 2016), which could be partially improved with a medium-chain triglyceride diet.

Drug-resistant epilepsy continues to be a major challenge in veterinary medicine, and further studies are needed to unravel its pathophysiology, identify clinical and behavioral factors that may provide new treatment approaches, and develop individually tailored treatment strategies to maximize responses to AEDs. AEDs will remain the mainstay of epilepsy management, but environmental factors, diet management, and treatment of comorbidities may also need to be considered to ultimately improve the QoL of both the patient and the owner.

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ONCOLOGY



**Gillian Dank (Israel)**

D-ACVIM -Oncology, D-ECVIM- Ca-Oncology

EERVC 2024 Lectures

1. Canine Osteosarcoma
2. Canine soft tissue sarcoma
3. Canine hemangiosarcoma
4. Feline lymphoma – different grades and locations with different prognosis

Gillian Dank, DVM, is a board certified veterinary oncologist and senior lecturer at the Koret School of Veterinary Medicine, Robert H. Smith Faculty of Agriculture, Food and Environment of the Hebrew University of Jerusalem. She earned her Doctor of Veterinary Medicine from the Koret School in 1998 and completed her residency in oncology at the University of California, Davis in 2002.

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CANINE OSTEOSARCOMA

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Osteosarcoma accounts for than 80% of the malignant bone tumors in dogs. The tumors are believed to arise from the medullary cavities, usually at the metaphysis, and expand outward, destroying cortex and disrupting periosteum.

Epidemiology:

- Increased incidence- Large to giant breeds with a body weight of more than 20 kg.
- Age- middle aged to older animals with a median of 8 years. The tumor can affect dogs of any age, and occurs in giant breed dogs at a young age.
- No sex predilection
- Breed- increased risk- Rottweilers
- OSA has been associated with sites of bone infarcts, healed fractures or internal- fixation devices- chronic irritation may play a role. In addition, OSA may be caused by radiation therapy.

Location and behavior:

- Most commonly affects the appendicular skeleton, metaphyseal region.
- Common sites: distal radius, proximal humerus (away from the elbow), proximal tibia, and distal femur (toward the knee).
- Forelimbs> hindlimbs

Clinical Presentation:

Lameness- intermittent in the early course of disease becoming chronic until the limb can no longer bear weight. The early fluctuating course is believed to be due to subperiosteal bleeding and microfractures of the weakened cortex.

Initially- there may be no clinically apparent lesion on palpation and radiographs may show only subtle changes.

As the disease worsens- swelling and lameness rapidly worsen and the lesion may be painful to the touch. If untreated progressive erosion of the cortex may cause pathologic fractures of the affected limb.

Duration- one to three months.

Staging and Diagnosis:

1. Minimum database: CBC, Serum Chemistry Profile. Urinalysis
2. Radiographs:
 - primary lesion- lytic, productive or mixed appearance, tumor extension and mineralization form periosteal spicules in the surrounding soft tissues: sunburst appearance (should not cross the joint)
 - Thoracic radiographs- 3 view
3. Ultrasound guided fine needle aspirate
4. Histopathology- biopsy from the center of the lesion using a Jamshidi bone marrow biopsy needle.

Prognosis:

- Dogs between the ages of 7 and 10 years had the longest survival times, both young and old dogs fared less well.
- Prognostic factors that did not influence survival: gender, site of the tumor, whether a presurgical biopsy was performed.
- ALKP has been shown to be a prognostic indicator
- Once OSA metastases are clinically or radiographically evident, good response to chemotherapy is rare. Pulmonary metastectomy seems to prolong survival if the dog is more than 300 days after the initial diagnosis, and if fewer than 3 nodules are found radiographically. Median survival after metastectomy was 176 days.

Treatment:

1. Amputation- eliminates the primary tumor, which provides pain relief. Palliative, but rarely increases survival. Median survival with amputation alone- 126 days. 10.7% were alive 1 year after surgery.
2. Limb Sparing procedure- ideal for dogs that are poor candidates for amputation (very large dogs, neurologic or orthopedic problems). Not an option if the lesion involves more than 50% of the bone, tumors that invade adjacent soft tissue or tumors of the hind limb.
3. Chemotherapy- significantly prolongs survival times (with removal of the primary tumor). Carboplatin, Doxorubicin, Doxorubicin + Carboplatin, and cisplatin are currently in use. With a median survival time of 10-12 months.
4. Palliative radiation- for dogs that cannot have amputation or a limb sparing procedure performed. Palliative pain relief.

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SOFT TISSUE SARCOMAS DOGS

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Incidence and Risk Factors

- Most sarcomas are solitary in the older dog and cat
- Etiology- sarcomas have been associated with radiation, trauma, foreign bodies, and parasites
- Dogs- tend to be over reported in larger breeds (flat coated retriever)

Pathology

- Develop from mesenchymal tissues
- Do not include tumors of hematopoietic or lymphoid origin

- Often considered collectively due to their similarity in clinical behavior and similarity in histologic features.

Important Common Features

- Tend to appear pseudoencapsulated soft to firm tumors but have poorly defined histologic margins or infiltrate through and along facial margins- locally invasive
- Local recurrence after conservative surgery - common
- Metastasis- hematogenous, in up to 20% of cases
- Regional LN metastasis are unusual (except synovial)
- Histopathologic grade is predictive of metastasis
- Resected tumor margins predict local recurrence
- Poor response to chemotherapy and radiation therapy for measurable/ bulky disease

History and clinical signs

- Slowly growing non painful mass
- Rapid cellular proliferation, intratumor hemorrhage or necrosis may result in a rapid increase in size
- Symptoms are related to the site of involvement, invasiveness of the tumor
 - Leiomyosarcomas can rarely have hypoglycemia

Diagnostic Techniques and Workup

- Fine needle aspirate
 - To rule out lipomas, seromas, inflammation, abscess
 - Problems-
 - Poor cellularity- don't exfoliate well
 - Resemblance between reactive tissue and benign and malignant tumors.
 - FNA from areas of necrosis- false negative
 - Biopsy-Necessary for a definitive diagnosis!!
 - Planned well- to be excised with surgery later
- Imaging- Regional and Thoracic
 - Radiographs
 - CT
 - Ultrasound
 - MRI
- CBC and Chemistry Panel

Treatment

- Determined based on location, clinical stage, histologic subtype, grade, completeness of surgery.
- Surgery is the treatment of choice!
- Local recurrence after surgery can develop in between **17% and 75%** of all patients
- It is well accepted that wide or radical excision margins of STS will provide good local tumor control in most patients. With treatment, many dogs with STS can experience prolonged survival with **median survival times ranging from 480 to 1796 days**

Take Home Points

- Diagnose every mass before removal
- Image/ CT every mass that is diagnosed as a soft tissue sarcoma
- Consider incisional biopsies for grading
- Only after imaging- discuss treatment options

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CANINE HEMANGIOSARCOMA

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Hemangiosarcoma- a malignant tumor of endothelial cells

Epidemiology:

- Increased incidence- German Shepherds
- Other breeds affected- Pointer, Boxer, Labrador Retriever, Golden Retriever, English Setter, Great Dane, Poodle, Siberian Husky.
- Age- mean- 8-10 years.
- No sex predilection No etiologic agent has been identified
- Cutaneous hemangiosarcoma- more common in females. Dogs with lightly haired, poorly pigmented skin are predisposed: whippets, salukis, bloodhounds, and English pointers. May be sunlight induced.

Location and behavior:

- Primary- Spleen, Cardiac (right atrial appendage or right atrium), liver, lungs, kidney, skin (SQ), bone, oral cavity, muscle, urinary bladder, peritoneum.
 - Metastasis: most often- liver and lungs. In addition- kidney, skeletal muscle, peritoneum, omentum, lymph nodes, mesentery, brain, spinal cord, urinary bladder, adrenal gland, diaphragm.
- Due to widespread metastasis identification of the primary tumor may be difficult.

Clinical Presentation:

Depends on the primary tumor site and the organs affected: progressive or intermittent lethargy, anorexia, collapse, weight loss, exercise Intolerance, dyspnea, pallor, acute hypovolemic shock can be seen due to rupture of the tumor.

Splenic Hemangiosarcoma- In addition, the dogs may have a palpable abdominal mass, abdominal distension, hemoperitoneum (fluid wave on palpation).

Cardiac Hemangiosarcoma- may be associated with syncope, ataxia, cyanosis, cardiac arrhythmias or signs of blood flow obstruction including peripheral edema, hepatomegaly, dyspnea and pleural effusion.

Hemangiosarcoma of the bone: clinical signs characteristic of malignant bone tumors- lameness, pain and soft tissue swelling.

Cutaneous Hemangiosarcoma- may have concurrent hemangiomas. Hemangiosarcomas are more likely to affect the dermis than the subcutis and have a predilection for ventral abdominal skin. The overlying epidermis is thickened and ulcerated.

Laboratory Finding:

Hematologic Disorders:

1. normocytic, normochromic, regenerative anemia (if early, a non regenerative anemia) with increased reticulocytes, nucleated rbc's, polychromasia, poikilocytosis, anisocytosis, schistocytes.
2. Neutrophilic leukocytosis

Hemostatic Abnormalities:

DIC- low platelet counts, high levels of fibrin degradation, prolonged PT and PTT

Staging and Diagnosis:

1. Minimum database: CBC, Serum Chemistry Profile. Urinalysis
2. Radiographs: pulmonary metastasis (characterized by a widely disseminated nodular pattern), abdomen (a large intraabdominal mass of soft tissue density in the mid-abdomen).
3. Ultrasonography: enlarged spleen, peritoneal effusion, sites of abdominal metastasis
4. Echocardiogram: examine right atrial appendage, pericardial effusion
5. Bone hemangiosarcoma- bone lysis with minimal periosteal reaction
6. Definitive diagnosis requires histopathology because cytology is problematic due to the heterogeneous nature of the tumor- hematomas, fibrotic areas, extramedullary hematopoiesis. In addition, the risk of bleeding is high.

Prognosis:

Splenic Hemangiosarcomas: Some believe that clinical staging is of prognostic value. Regardless, the prognosis is poor.

1. Stage 1- confined to the spleen with no evidence of metastasis
2. Stage 2- may have a ruptured spleen, with or without regional lymph node involvement
3. Stage 3- large invasive tumors with distant metastasis

Cardiac Hemangiosarcomas: most either die or are euthanized at or shortly after diagnosis because of the high rate of metastasis.

Cutaneous Hemangiosarcoma: solar elastosis in the skin adjacent to the hemangiosarcomas is related to long survival- solar induced hemangiosarcomas may be less aggressive.

Treatment:

1. *Splenic Hemangiosarcomas:*
 - Surgery- treatment of choice. Relieves abdominal distention and provides palliation by stopping bleeding. Does little to prolong survival.
 - Chemotherapy- Doxorubicin/ Doxorubicin and cyclophosphamide. Average survival times – 6 months.
2. *Cardiac Hemangiosarcoma:*
 - Surgery- only possible if the tumor is confined to the right auricle. Mean survival times in one study- 4 months. All dogs developed disseminated metastatic disease.
 - Chemotherapy-
3. *Hemangiosarcoma of the bone:*
 - Surgery- amputation
4. *Cutaneous Hemangiosarcoma:*
 - Surgery- treatment of choice
 - Chemotherapy- In light of the long remission with surgery alone the contribution is hard to assess.

Conclusion:

Canine Hemangiosarcoma is a rapidly progressive, malignant, fatal disease. Present methods of treatment have limited success on a long term basis. Advanced diagnostics that allow early detection of disease and chemotherapy may be the key to improved survival.

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FELINE INTESTINAL LYMPHOMA-LARGE AND SMALL CELL. DIFFERENT DISEASES WITH DIFFERENT PROTOCOLS AND DIFFERENT PROGNOSIS

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Lymphoma is a cancer of the lymphocytes. Lymphocytes are cells involved in the immune system and travel throughout the body in the blood and lymphatic vessels; therefore, lymphoma is always considered to be a systemic, not localized, disease. Lymphoma is seen frequently in cats, and accounts for approximately 30% of new feline cancer diagnoses. Common sites of lymphoma in cats include:

1. **Intestinal lymphoma.** This term describes lymphoma that affects the gastrointestinal tract. This is, by far, the most common type of lymphoma in cats, accounting for 50-70% of feline lymphoma cases. It is most common in older cats, with the average age at diagnosis ranging from 9-13 years old.
2. **Mediastinal lymphoma.** In this form of lymphoma, lymphoid organs in the chest (such as the lymph nodes or the thymus) are affected. Mediastinal lymphoma is often seen in young cats, with an average age of onset of 5 years old.
3. **Renal lymphoma.** Lymphoma in the kidney can lead to signs of kidney failure, as functional kidney cells are replaced by cancer cells.
4. **Nasal lymphoma-** difficulty breathing or facial deformity.

What are the clinical signs of lymphoma? They depend on the effected organ.

In the intestines-clinical signs are often similar to other intestinal diseases. Affected cats often develop weight loss, vomiting, and diarrhea. Appetite varies; some cats have a decreased appetite, some have an increased appetite, while others have no change in appetite.

Mediastinal lymphoma occurs within the chest and is often associated with respiratory difficulties. Fluid often accumulates around the tumor, making it more difficult for an affected cat to inflate the lungs fully.

In cats with renal lymphoma, signs associated with kidney failure may be seen. These signs often include decreased appetite, weight loss, increased thirst, and vomiting. These changes are all associated with a buildup of toxins in the bloodstream that the kidneys cannot filter effectively.

How is lymphoma diagnosed?

Fine needle aspirate of an enlarged lymph node, enlarged kidney, thickened region of intestine, or fluid present within the chest. If a fine needle aspirate is inconclusive or impractical due to the location of the lesion, then either endoscopy or a surgical biopsy is performed. If lymphoma is diagnosed via biopsy, the pathologist can also determine whether your cat possesses **high-grade or low-grade lymphoma**.

Treatment- Lymphoma is usually treated with chemotherapy.

- Low-grade lymphoma is treated with prednisone (a steroid) and chlorambucil (an oral chemotherapy agent). Low-grade lymphoma in cats is more likely to respond to chemotherapy, and chemotherapy often results in longer periods of remission.
- High-grade lymphoma is treated using one of several injectable chemotherapy protocols. Cats tolerate chemotherapy much better than humans; they rarely lose their hair or appear sick. The most common side effects include vomiting, diarrhea, and decreased appetite. However, even these effects are seen in only about 10% of patients.

- Surgery and/or radiation may be appropriate for lymphoma confined to one area, such as nasal tumors or abdominal masses, but this is uncommon. Most cases cannot be successfully treated with surgery or radiation and will require chemotherapy.

What is the prognosis? The prognosis for lymphoma depends on:

1. the location of the lymphoma
2. the grade of the lymphoma
3. how sick the cat is at the start of treatment
4. how quickly the disease is diagnosed and treated.

Most cases of **gastrointestinal lymphoma** are **low-grade lymphoma**. With treatment, approximately 70% of cats with low-grade lymphoma will go into remission. Lymphoma is never truly cured, but remission is a term used to describe the temporary resolution of all signs of lymphoma. The average remission for low-grade lymphoma is two to three years, meaning two to three years without any signs of disease.

High-grade gastrointestinal lymphoma, however, does not respond as well to treatment. Only 25-50% of cats with high-grade lymphoma achieve remission with treatment. Typically, this period of remission lasts only 6-9 months, and then cats become ill again. Cats that achieve a complete remission live longer than those achieving a partial remission and a small percentage can live more than 2 years. Prognosis includes 6 months of treatment with a combination of injectable and oral chemotherapy drugs. Some cats with gastrointestinal lymphoma benefit from surgery or radiation therapy in addition to chemotherapy. Large cell lymphoma can also be treated with an oral drug called CCNU; median survival time for responding cats is approximately 6 months.

Mediastinal lymphoma - These cats show an average survival time of 9-12 months, with the initial response to treatment often indicating survival time.

Renal lymphoma, unfortunately, carries a poor prognosis. Average survival with this type of lymphoma is only 3-6 months, though there are isolated reports of cats surviving far longer. Renal lymphoma tends to spread to the brain and central nervous system; this occurs in approximately 40% of renal lymphoma cases and worsens the prognosis for this disease.

Nasal lymphoma-Cats with nasal lymphoma have a prognosis of approximately 1 year with treatment (radiation and/or chemotherapy)

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**Antonio Giuliano (Hong Kong)**

DVM, MS, GpCert(SAM), PgCert(CT), ECVIM (onc), MRCVS European, RCVS and HK specialist in Companion Animals

EERVC 2024 Lectures

2. Fine needle aspirate (fna) and biopsy tips in oncology; mastering techniques for precise diagnoses
3. Urothelial carcinoma from diagnosis to treatment options
4. Mast cell tumours in dogs, what's new?
5. Understanding mammary tumours in dogs and cats

Dr. Antonio Giuliano graduated from the University of Messina in 2007. While working in general practice, he completed a master's degree in Small Animal Oncology from the University of Pisa, a post graduate certificate in cancer therapeutic at Barts Cancer Institute and the GpCert (SAM) in internal medicine. He completed a rotating internship in a busy referral practice in UK and an oncology internship and residency at the Queen Veterinary School Teaching Hospital (QVSH), at the University of Cambridge.

After successfully completing the residency, he worked for one year as oncology clinician and supervisor at the QVSH. In the 2019 he moved to Hong Kong (HK) working as oncologist at CityU VMC, the largest multi-disciplinary referral Hospital in HK.

Dr Antonio Giuliano is a recognised European, RCVS and HK specialist in Companion Animals (Oncology). He published numerous articles in international peer reviewed journals and presented his research in many international conferences.

He has a broad interest in cancer research, with particular focus on finding new drugs/therapeutic approaches to improve survival time and quality of life of pets. He is also interested in preclinical and clinical investigations into the development of rapid tests to predict prognosis and/or early diagnosis of cancer.

FINE NEEDLE ASPIRATE (FNA) AND BIOPSY TIPS IN ONCOLOGY; MASTERING TECHNIQUES FOR PRECISE DIAGNOSES

Antonio Giuliano DVM, MS, PgCert (CT), GpCert (SAM), AiCVIM (onc), ECVIM(onc), MRCVS

Fine needle aspirate (FNA) is the most common technique used in veterinary medicine to sample lumps and bumps in the skin. It is also used to sample mass located in the abdomen or thorax via ultrasound (US)-guided or more rarely CT-guided techniques. FNA is minimally invasive with a very low risk of complications, it is cheap, with a fast result turnover. FNA can be fully evaluated in-house or sent directly to an external laboratory.

It is always advisable, before sending the cytology to the lab, to have a look in-house to evaluate the quality of the sample.

Is it the sample cellular enough? Is it representative of the lesion/diagnostic? This will avoid annoying inconclusive results and waste of time and money.

There are different techniques for taking FNAs often depending on the suspected cancer type (different tumour texture and fragility of the cells; Lymphoma VS sarcoma).

As a general rule, it is better to start taking FNA using a small gauge needle to avoid blood contamination and if not enough cells are collected, switch to a larger gauge.

When using a US-guided technique, it is important to select the most "solid" and less cavitate or vascularised areas of the tumour, so to avoid sampling necrotic/cystic areas that are unlikely to be diagnostic and avoid excessive blood contamination.

Despite the complications rate of FNA sampling being very low, the author suggests caution in certain scenarios, for example, large cavitate mass in the spleen, cavitate masses in the lungs or patients with coagulation problems. Thrombocytopenia is unlikely to be a significant risk factor unless severe.

In certain cases, impression smear or scraping of superficial masses/lesions can also be used (es. mucosal oral lymphoma), but in general, these methods are less likely to give a diagnosis.

When a final diagnosis cannot be reached by FNA or when establishing tumour grading is needed (like in some cases of mast cell tumours), biopsies can be performed.

Different biopsy methods can be used based on the location, appearance of the mass and the clinical suspect.

Tru-cut biopsies are used for large subcutaneous masses or internal organs via ultrasound or CT-guided techniques. The use of Tru-cut is considered safe but caution needs to be applied during sampling of internal organs like liver or lung masses. Tru-cut biopsy of the spleen should be always avoided.

Punch biopsy can be used for masses in the skin or mucosae, like the oral cavity. However, as the punch biopsy can only take a superficial sample, attention needs to be paid to collecting a representative sample of the mass avoiding areas of inflammation or necrosis.

The best portion or area of the mass to biopsy is difficult to establish as mostly depends on a case-by-case basis. However certain general rules apply to different cancer types. For example, sampling the centre of the tumour is advisable in osteosarcoma, while in FISS the centre needs to be avoided as often it is necrotic and likely to result in a non-diagnostic sample.

Surgical biopsies are usually taken when other less invasive techniques are not feasible (splenic masses) and they can be done by excisional or incisional biopsy depending on the cases (STS in dogs, versus FISS). Various clinical case scenarios along with visual aids such as pictures and video illustrations will be presented during the conference talk!

UROTHELIAL CARCINOMA FROM DIAGNOSIS TO TREATMENT OPTIONS

Antonio Giuliano DVM, MS, PgCert (CT), GpCert (SAM), AiCVIM (onc), ECVIM(onc), MRCVS

Canine transitional cell carcinoma (TCC), also referred to as urothelial carcinoma (UC), is the most common malignant neoplasm of the urinary tract in dogs, accounting for about 1.5 to 2% of all canine cancers^{1,2}. The term urothelial carcinoma encompasses both the carcinoma affecting the prostate and the urinary bladder, TCC is the most common type of carcinoma in the urinary bladder while in the prostate, adenocarcinoma is the most common tumour type. However often it is difficult to differentiate bladder from prostate carcinoma as bladder tumours infiltrate the prostate and vice versa.

Certain breeds, such as Scottish terriers, Shetland sheepdogs, and West Highland white terriers are predisposed to TCC^{1,3}. UC is a locally aggressive cancer, that invades the urinary bladder wall, and often metastasizes to the regional lymph nodes and distant organs, with the metastatic disease found in more than half

of the patients at necropsy^{4,5}. However, most dogs are usually euthanized due to the progression of the local disease and/or lower urinary tract obstruction⁶. The clinical signs of TCC are nonspecific and include hematuria, dysuria, pollakiuria, stranguria, and urinary obstruction^{2,4}.

Diagnosis is usually obtained by traumatic catheterization or biopsy sample via direct visualization of the cancer by cystoscopy, while percutaneous ultrasound-guided FNA is often contraindicated to avoid needle-track seeding of the tumour.

The prognosis of canine UC is generally poor, with most patients surviving less than a year despite treatment^{7,8}. The most common treatment options for canine UC consist of chemotherapy often with mitoxantrone, carboplatin, gemcitabine, doxorubicin and vinblastine⁹⁻¹⁰. Non-steroidal anti-inflammatory drugs (NSAIDs) with cyclooxygenase-2 (COX-2) inhibiting properties, especially piroxicam and meloxicam are often used alone or in combination with chemotherapy. The role of surgery in the treatment of UC is still controversial.^{4,9} Most canine UCs are located in the trigonal region of the urinary bladder where surgery is usually not feasible¹⁰. Even when the tumour is located away from the trigone and resected with complete margins, recurrence is often seen due to urinary bladder field cancerization. Despite surgery alone is unlikely to be effective, a combination of surgery and chemotherapy has shown some promising results^{2,4,9}.

In addition to conventional chemotherapy agents, metronomic chemotherapy has emerged as a promising alternative or adjunctive treatment for canine TCC. In a prospective study, chlorambucil showed 70% clinical benefit, with a good safety profile, as only 23% of dogs developed mild adverse events¹¹.

New findings on preliminary research conducted by the author, new therapeutic approaches and examples of cases will be presented during the presentation!

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MAST CELL TUMOURS IN DOGS, WHAT'S NEW?

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Mast cell tumour (MCT) is one of the most common types of cancer in dogs(Dobson et al., 2002). It can have variable clinical appearance and presentation. MCT can look like a skin tag, a papule, a small nodule or a large mass. MCT can resemble a fluid-filled cyst or a soft and movable subcutaneous mass like a lipoma.

Diagnosis of MCT is really important to plan further treatment and FNA can easily achieve it. However, achieving a diagnosis of MCT is not sufficient, because the prognosis and treatment will depend on the histological grade (Kiupel et al., 2011).

Predicting the biological behaviour of MCT is important, but it can be challenging. Histopathology grading and mitotic index are probably the most important ways to predict the biological behaviour of MCT, but it is not always enough(Kiupel et al., 2011; Romansik et al., 2007). Some immunohistochemistry stains or molecular features can be requested to have more comprehensive prognostic information in ambiguous cases(Thamm et al., 2019).

The clinical appearance and stage of the MCT are also important for predicting prognosis and treatment planning(Stefanello et al., 2015)

The need to perform staging in a dog with MCT depends on a case-by-case, but most dogs with MCT do not need to be staged.

MCTs usually have predictable biological behaviour and metastatic patterns, infiltrating the regional lymph nodes first, followed by the liver and spleen. When needed, a sufficient baseline staging can be easily performed by FNA of the regional lymph node, abdominal ultrasound and FNA of the liver and spleen or just the spleen(Brown et al., 2022; Pecceu et al., 2018).

CT scan is often not necessary, unless for surgical planning of large masses. Both ultrasound and CT scan of the liver and spleen can appear completely normal despite significant MCT infiltration. Cytology of apparently normal-looking spleen and liver is often necessary to confirm or rule out organ involvement(Pecceu et al., 2018). Thoracic radiography is rarely included in the staging of MCT, as this tumour very rarely metastases to the lungs. However, ruling out comorbidities in the lung/heart in an old dog before going for extensive surgery is often a good idea.

Surgery is the most effective treatment for low-grade/low-risk MCT and it is often curative.

For high-grade/high risks MCT adjuvant treatment, local or systemic or both are often necessary. Various local treatments can be used for incompletely resected MCTs, like scar resection, radiotherapy, electrochemotherapy or even just monitoring.

There are a few chemotherapy treatments for MCTs; vinblastine and prednisolone are probably still the most effective treatment (in particular in microscopic diseases). Other available chemotherapies like lomustine and chlorambucil, or various combinations can also be used. Tyrosine kinase inhibitors (TKI) are effective in the treatment of MCT and have been extensively studied(Weishaar et al., 2018). Both TKIs, masitinib and toceranib have similar response rates, but slightly different safety profiles.

In summary, Most MCTs will be treated and often cured just by surgical resection. Establishing which case will need staging/chemotherapy/other adjuvant treatment will depend on the accurate evaluation of multiple factors like the history, clinical appearance, grading, staging, and sometimes molecular markers or histopathology review/second opinion.

An interactive clinical case will be presented at the end of the talk!

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UNDERSTANDING MAMMARY TUMOURS IN DOGS AND CATS

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Mammary tumours are one of the most common cancers affecting both dogs and cats, however, the incidence varies widely between countries. The risk of developing mammary tumours in both dogs and cats is related to the neutering status, countries with a low neutering rate have a much higher incidence of mammary tumours compared to countries with high neutering rates. However, it is not only the neutering status, but the time of neutering that is even more important. Time of neutering significantly influences the risk of developing mammary tumours, for example, for dogs neutered before the first oestrus, the risk is only 0.5%, but the risk increases to 8 % between 1st & 2nd oestrus and 26% between 2nd & the 3rd oestrus. While neutering after 2.5 years of age shows no reduction in the risk of developing mammary tumours¹. The most common tumour affecting the mammary gland is adenocarcinoma with various possible different subtypes (mixed, solid, papillary etc).

Staging is important to plan treatment and give an accurate prognosis. Basic staging involving thoracic radiographies plus /minus abdominal ultrasound and clinical evaluation of the regional lymph nodes (axillary/inguinal) are often enough for small mammary tumours in dogs. While in cats or dogs with large masses/enlarged regional lymph nodes, histologically aggressive/high-grade tumours an abdominal and thoracic CT scan is preferable.

The treatment of choice for mammary tumours is surgery in both dogs and cats. While in dogs a simple mastectomy of the affected gland is often curative (around 75% of cases are cured) in cats a bilateral total mastectomy (bilateral mammary strip) is necessary for long-term control of the disease, and it is still considered the most effective treatment option (around 90% of feline mammary tumours are malignant and a lower percentage of cases can be cured by surgery,~40%)².

The role of adjuvant chemotherapy is controversial and should only be reserved in cases with multiple negative prognostic factors (advanced stage/size, high grade, aggressive histotype).

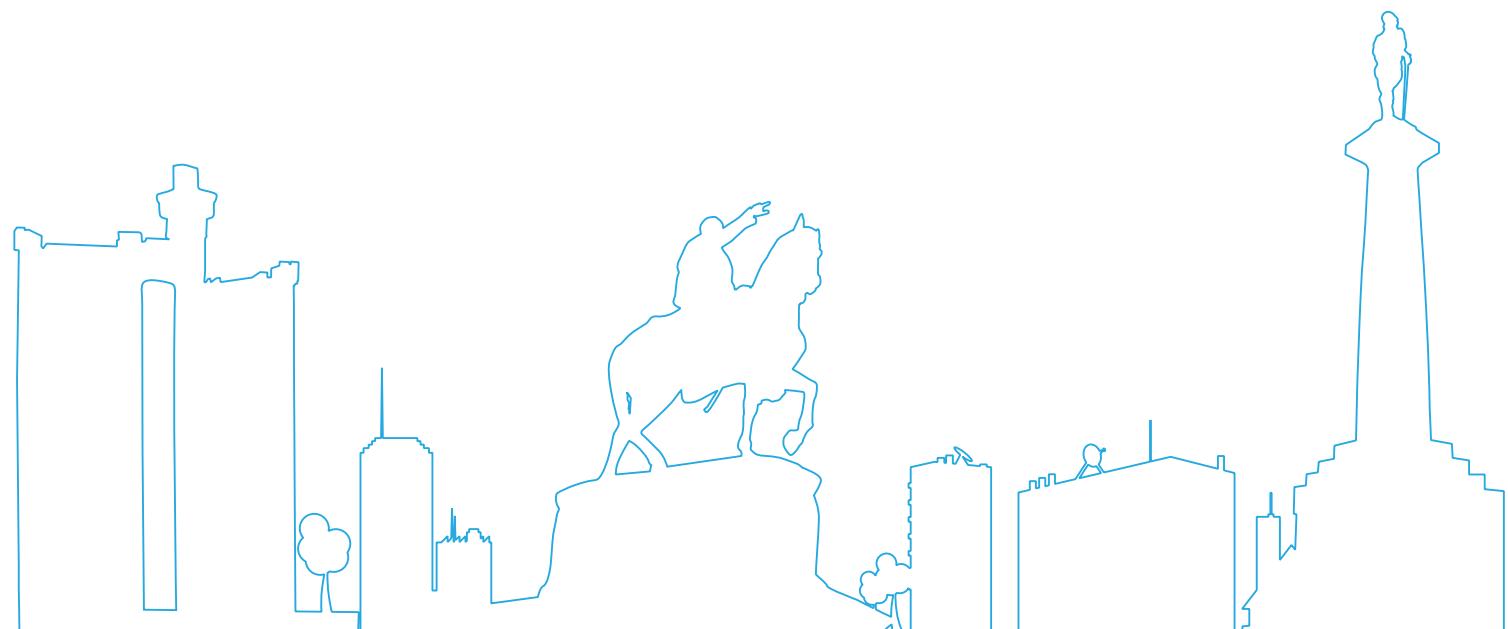
The prognosis in both dogs and cats varies widely depending on the size of the tumour, stage, grade, histotype and histology parameters (lymphatic invasion/mitotic index) and treatment^{3,4}.

In dogs the majority of mammary tumours are cured by surgery, however, some uncommon, but aggressive types of carcinomas like anaplastic, comedone and squamous cell carcinoma tend to have a more aggressive behaviour and higher metastatic rate. Inflammatory carcinoma is one of the most aggressive types of carcinomas in dogs that invariably carry a very poor prognosis and surgery is rarely indicated⁵.

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ORTHOPEDICS





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EERVC 2024 Lectures

1. Why do people believe exercise harms joint development?
2. Can joint supplements do more harm than good?
3. The hidden dangers of unlicensed painkillers
4. The ideal diet for arthritic dogs

Mike Farrell graduated from the Royal Veterinary College in 1997 and completed internships at Bristol and Edinburgh Universities. He's worked in the UK and Australia as a general practitioner and in the USA and Switzerland as a veterinary anaesthetist.

Mike completed a surgery residency at Glasgow University in 2006 and gained his European Diploma in Small Animal Surgery in 2007. He's an EBVS and RCVS board certified specialist in small animal surgery. Mike's open access educational YouTube channel, www.youtube.com/vetlessons, helps veterinary practitioners and animal lovers make difficult choices for their pets.

IS EVERYTHING WE'VE BEEN TOLD ABOUT EXERCISING PUPPIES WRONG?

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About the Author

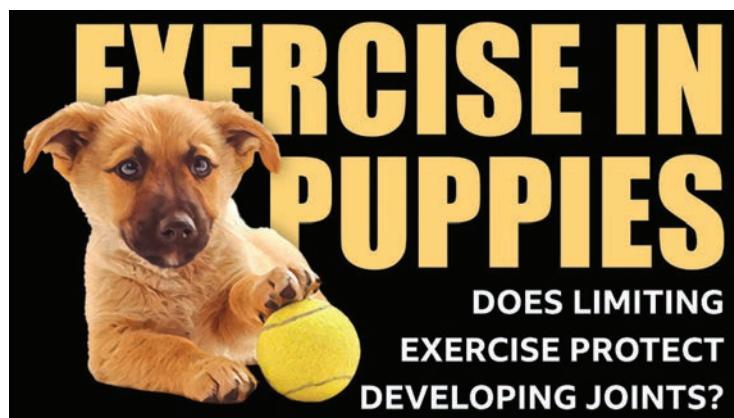
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Free Online Lecture

Separating facts from fiction can be a daunting task. I've done the hard work for you by reviewing dozens of scientific studies relevant to this subject. In this free online lecture, I'll share the key findings using plain language.



Take-Home Messages

- **If exercise were a supplement,** it would be hailed as a wonder drug. Its ability to limit chronic inflammation, optimise musculoskeletal health, and lay a strong foundation for a long life is nothing short of miraculous.
- **How should we tell the truth?** Knowing the truth is not enough. It's the truths we share that create change. Dogs rely on animal lovers to speak out, so when you know the truth, please share it.
- **What's the critical message?** Overfeeding puppies dramatically increases their risk of developmental joint disease, chronic pain and a shortened lifespan. The take-home message couldn't be simpler: We must defend puppies' healthiest hobby and refocus public attention on the true culprit, overfeeding.

Introduction

Picture a doctor diagnosing a little girl with hip dysplasia. The child's parents are referred to a physiotherapist who explains the problem:

"Your daughter's hips have weak stabilisers. Her joint capsules are weak, so her secondary stabilisers must work harder."

He explains that the overworked hip stabilisers form part of her core. Everything's making perfect sense until he suggests a programme to weaken her core. Here it is:

- Ten minutes of slow crawling three times daily
- Feed the recommended amount for a normal child even though she isn't allowed to exercise. Okay, so she might pack on some puppy fat, but what harm can that do?
- Restrict play because it's bad for her developing joints

None of this is good advice. Play, for example, does not harm developing joints. It's how young mammals learn how to deal with the unexpected. When they run, jump, and chase one another, they alternate between losing and regaining control both physically and emotionally. In effect, they're deliberately teaching themselves how to deal with the physical and emotional challenges of life.

Supporters of restricted exercise programmes employ a common defence. In their opinion, restricted exercise programmes are "*a sensible guideline created to discourage irresponsible exercise.*" At first glance, this seems like a smart argument. At second glance, it's anything but. It's a perfect example of a *slippery slope fallacy*.

How would it sound if we applied the same logic to diet? Imagine someone suggested feeding your dog one carrot per day per month of age. When you counter that a 100% carrot diet is unbalanced, they'd say: "It's a sensible guideline to stop you feeding grapes and chocolate."

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2. A 1975 study funded by the NIH found that crate-confined German shepherd dog puppies were less likely than unrestricted puppies to develop hip dysplasia. The author's actual conclusion - that [a wide-based stance limits dysplasia](#) - might have evolved into the fallacy that exercise restriction prevents hip dysplasia.
3. Incline treadmill running for 4km/day, 5 days/week for 15-weeks produced subtle [improvements in cartilage quality in beagle puppies](#).
4. Puppies who ran a daily marathon 5 days per week for a year showed [no evidence of developmental joint disease](#). The only difference between marathon-running puppies and a control group was focal superficial cartilage matrix change.
5. The Rarámuri, whose children and teenagers participate in a long-distance running tradition, have [significantly lower rates of joint disease](#) compared to Australian Aboriginal communities where children and adolescents are much less active.
6. If exercise caused hip dysplasia, wolves wouldn't have [survived for 750,000 years](#) and domestic dogs wouldn't exist.
7. Guide dog charities' success reducing joint disease prevalence owes everything to careful breeding and [nothing to limited exercise](#).
8. A study of 325 Swedish Labradors provides [no evidence](#) that chasing sticks and balls causes abnormal joint development.
9. Slippery surfaces have [no significant effect](#) on the prevalence of hip dysplasia.
10. A team of Norwegian researchers suggest that young puppies should not be allowed access to stairs [without providing any evidence](#) that stair exercise causes harm.
11. The decline of play and the [rise of psychopathology](#) in children and adolescents.
12. Puppies deprived of the opportunity to play are [significantly more likely to display emotional extremes](#) in stressful situations.
13. The commonest cause of death in dogs under 3-years-old is [euthanasia due to behavioural problems](#).

A SCEPTIC'S GUIDE TO COMPANION ANIMAL JOINT SUPPLEMENTS

Mike Farrell BVetMed CertVA CertSAS DipECVS MRCVS

clinically proven /'klinɪkəlɪ 'prvən/ verb **1.** A treatment which has undergone a rigorous testing process involving one, or ideally several, clinical trials which are published in peer-reviewed scientific journals. **2.** A treatment supported by unpublished data and testimonials; e.g. YuMove®

Concorde effect /'kɔːnkrd ɪ'fkt/ noun A compulsion to continue investing time, money, or effort into a failing strategy purely because we've already made a significant personal investment. From the doomed supersonic passenger jet which ultimately cost British and French taxpayers over £4 billion.

correlation /kɔː'releɪʃən/ noun A statistical relationship; e.g. "a strong correlation was found between the number of library assistants working in Alabama and arson incidence in the same state (2002-2022), $r=0.773$ "

or "a correlation was found between dogs who were born in autumn and the development of hip dysplasia."

causation /kɔː'seɪʒən/ noun The act or process of causing. Correlation does not imply causation; e.g. there is no actual evidence that Alabaman library assistants are arsonists, and there is no evidence that increased exercise causes hip dysplasia.

endowment effect /ɛ'ndmənt ɪ'fkt/ noun Barring major complications, human nature compels us to believe that our personal choices are objectively superior than the choices we rejected. Also known as the 'vain brain' phenomenon, endowment bias is one of many reasons to be sceptical of testimonials.

numerically superior /nu'mrɪklɪ su'iħrɪər/ adj An expression used by researchers who are (a) industry funded, (b) determined to accept their hypothesis, and (c) unable to prove their point using statistical tests.

opportunity cost /aapər'uwwnət̩i kast/ noun 1. The difference between the profit we make for our choice less the profit we would have made if we'd chosen a better option, 2. The price paid by painful animals who are given joint supplements instead of actual painkillers.

pure natural halo /pjʊr 'nɪtʃərəl 'hhloʊ/ noun A sense of inherent 'goodness' which protects supplements (but not prescription painkillers) from critical appraisal.

repeated measures /ri'iytɪd 'mɜːrz/ verb A tactic favoured by industry funded researchers to increase the odds of a positive outcome. If we throw one dart and hit the bullseye, the outcome probably wasn't a fluke. If we throw fifty darts and one hits the bullseye, the odds of it being a fluke are much higher. It is common practice for researchers to report bullseye results without mentioning the misses (or pretending the misses are unimportant).

Russell's teapot /russell's, 'tpt/ A hypothetical teapot proposed by Bertrand Russell that is orbiting the Sun between Earth and Mars to make the point that not all claims that cannot be proven false should be accepted as true; i.e. absence of evidence is not evidence of absence.

study limitations /'stdi ,lɪmɪ'eyeɪʃənz/ noun 1. Inconvenient truths which are routinely under-played, while weak positive findings are routinely over-played. 2. Also known as critical study flaws.

Schrödinger's painkiller /'pɔːnklər/ noun Any therapy which is simultaneously powerful and lacks the potential to cause severe side-effects; e.g. anti-nerve growth factor antibodies, joint supplements.

sharpshooter fallacy /'ʃrpʃʊtər 'flæsi/ The common practice of selecting the criteria for success specifically to match the results that are already known. See 'repeated measures'.

"there's conflicting evidence" /ðer ɪz kən'flktn̩ 'ehvædəns/ See "we need more studies".

treatment pyramid /'trtmənt 'præmɪd/ noun A visually appealing but misleading paternalistic decision aid.

unicorn /'ynɪ,əoɔrn/ noun 1. A mythical therapy capable of producing a profound positive impact but incapable of producing a profound negative impact. 2. A 'disease-modifying' or 'chondroprotective' joint supplement. See Schrödinger's painkiller.

unpublished data /ə'nɒplɪʃt 'dtə/ noun Supportive data which (a) helps sell a product, (b) cannot be verified, and (c) should not be confused with 'scientific proof'. See 'clinically proven'.

veterinary armchair quarterback /'vtrɪə,nri 'aar,mtʃr k'wrtər,aeæk/ noun 1. A layperson who criticises a veterinarian or offers veterinary advice, with or without the prefix, "I'm not a vet" 2. Someone who sincerely believes that 'doing your research' is equivalent to 'doing research'.

"we need more studies" /wi nɪd mɔːr 'stdiz/ A popular statement used to defend weak arguments from overwhelming scientific evidence; e.g. "You say the Earth is spherical. I say we need more studies." or "You say joint supplements are not 'disease modifying'. I say we need more studies."



Mike Farrell graduated from the Royal Veterinary College in 1997. He's a diplomate of the European College of Veterinary Surgeons and a board-certified specialist of the European Board of Veterinary Specialists and the Royal College of Veterinary Surgeons. Mike has a passion for companion animal pain management, and is well-known for creating VetLessons, a free online resource that simplifies complex orthopaedic problems for veterinary professionals and pet owners. In his spare time, Mike enjoys fashioning false moustaches from moss he finds on trees.

THE RISKS AND BENEFITS OF UNLICENSED PAINKILLERS FOR DOGS

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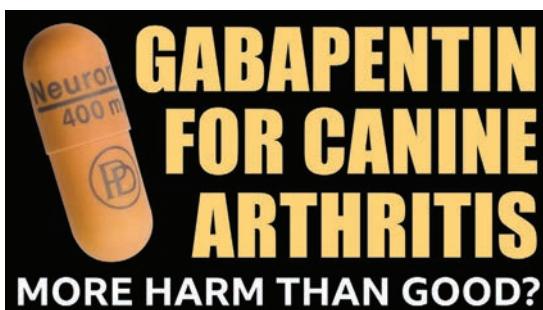
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Free Online Lecture

How does gabapentin help or harm arthritic dogs? In this lecture, we'll delve into the drug's mechanism of action, potential side effects, and overall effectiveness in managing canine joint pain.



We'll also cover the risks and benefits of unlicensed opioids and cannabidiol (CBD).

Take-Home Messages

- Gabapentin is not anti-nociceptive. This is scientific jargon for "gabapentin is not a painkiller".
- Gabapentin is a pain *modulator* which slows nerve firing rate. This explains why it's a popular choice for sedating anxious or aggressive animals.
- There are many excellent reasons why gabapentin is not used to treat human arthritis.

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IS EVERYTHING WE'VE BEEN TOLD ABOUT EXERCISING PUPPIES WRONG?

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Free Online Lecture

If we want to make good choices, we must first separate facts from fiction. This can be a very daunting task when the subject is nutrition because we're 'fed' so much misinformation. I've done the hard work for you by reviewing dozens of scientific studies relevant to this subject. In this free online lecture, I'll share the key findings using plain language.

**Take-Home Messages**

- **Food substitutes are not food**, and completeness is no longer the most important pet food metric
- **Excuses don't make pets healthier:** The most popular excuses for failed weight loss are not backed by science.
- **No pain, no gain:** Significant improvement requires significant investment. Like it or not, there's no such thing as a free lunch

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**Ignacio Carlos Calvo Bermejo (Spain)**

CertSAS DipECVS PhD MRCVS

EERVC 2024 Lectures

1. Feline joint luxations. Anything different to dogs?
2. Carpal an tarsal arthrodesis in the feline patient. Tricks and tips
3. Common failures in feline fracture fixation. Lean from my mistakes
4. Feline OA, what we need to know?

Nacho graduated from the University of Cordoba (Spain) in 2000. After a formative period at Madrid Vet School, he then completed an internship in Small Animal Medicine and Surgery at the University of Glasgow, Scotland, followed by completion of a 3-year Small Animal Surgery Residency at the same institution. Nacho obtained the RCVS certificate in small animal surgery in 2009 (CertSAS), the European Diploma in Small Animal Surgery (Dipl ECVS) in 2012 and his PhD for studies into the Tibial Tuberosity Advancement procedure in 2016. Before joining Hospital Animal Bluecare in Malaga (Spain) as medical director and head of orthopaedics in July 2018, Nacho held the positions of Lecturer in Small Animal Surgery at University College Dublin (UCD), Lecturer in Small Animal Orthopaedic Surgery at Glasgow Vet School, Senior Clinician in Orthopaedics at Fitzpatrick's Referrals and Senior Lecturer and Head of Orthopaedics at the Royal Veterinary College in London. Nacho is an active faculty and European board member of AOVET. Nacho is also the Musculoskeletal disorders and orthopaedics section editor of BMC veterinary research.

COMMON FAILURES IN FELINE FRACTURE FIXATION. LEARN FROM MY MISTAKES

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Fracture fixation in feline patients is a pivotal aspect of veterinary orthopedic practice, involving specific challenges attributable to the unique anatomical and physiological characteristics of cats. This comprehensive review synthesizes the current understanding and clinical observations regarding common fracture fixation failures in cats, examining both intrinsic and extrinsic factors that contribute to these adverse outcomes. Despite significant advancements in fracture repair techniques and materials, fixation failures remain a significant concern, influencing recovery trajectories and overall quality of life in feline patients.

Intrinsic Factors:

Intrinsic factors involve the inherent biological, mechanical and anatomical attributes of the feline species that can impact fracture healing. Cats have distinct bone characteristics compared to similar-sized dogs, which affects the integration and stabilization of fixation devices. Additionally, the type and location of the fracture significantly influence the potential for fixation failure. Certain fractures, such as those in the tibia, inherently challenge stabilization due to the complex forces and biological environment involved.

Extrinsic Factors:

Among the extrinsic factors, the choice of fixation method and materials is paramount to successful fracture management. Common techniques encompass internal fixation with plates and screws, external fixation devices, and intramedullary pins. Each method presents distinct advantages and potential pitfalls. Internal fixation, although often providing superior stability, can lead to complications such as screw loosening, plate breakage, and soft tissue irritation, particularly if subjected to inappropriate stress. The selection of device size and type, which must be tailored to address specific fracture configurations, is crucial. Misjudgement in these factors frequently underpins fixation failures.

Post-operative management and the natural behaviour of cats are critical considerations. Cats' agile nature and tendency towards high mobility can inadvertently increase stress on healing fractures, thereby risking fixation failures. Thus, ensuring adequate immobilization and regulated movement post-surgery is essential.

Errors in surgical technique, such as improper alignment or insufficient reduction of the fracture site, contribute significantly to fixation failures by leading to malunion or nonunion conditions. These occur when the bone heals incorrectly or not at all, respectively. Additionally, infection at the surgical site presents a notable risk, often necessitating the removal of fixation devices and revision surgeries that further complicate recovery.

Innovations and Future Directions:

Recent advancements in fixation technology offer promising avenues to mitigate these challenges.

To effectively reduce the incidence of fixation failures in feline patients, a multifaceted approach is recommended, involving copious preoperative planning, precise execution of surgical technique, appropriate fixation method selection, and diligent post-operative care. Continuous professional development and research are essential for veterinary orthopedic surgeons to remain well-informed about the latest techniques and materials available, ultimately enhancing clinical outcomes for feline patients.

Efforts must also concentrate on generating prospective studies and clinical trials focused on understanding feline-specific responses to different fixation devices and techniques. Enhanced biomechanical testing and the development of strategies specifically tailored to cats could significantly lower the prevalence of fracture fixation failures in this distinct patient population. Through such an understanding, veterinary orthopedic practices can more effectively tailor interventions to improve recovery rates and overall patient welfare in cats experiencing fractures.

In conclusion, while addressing the myriad factors contributing to fracture fixation failures in feline patients remains complex, the integration of emerging technologies and a deeper understanding of species-specific needs offers a pathway to more successful outcomes in feline orthopedic care. This review underscores the importance of targeted research and the development of feline-specific orthopedic protocols to enhance the effectiveness of fracture fixation in veterinary practice.

FELINE OA. WHAT WE NEED TO KNOW?

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Principio del formulario

Feline osteoarthritis (OA) is an increasingly recognized condition, affecting a substantial portion of the domestic cat population, particularly as they age. This comprehensive review aims to consolidate current knowledge on the diagnosis and management of OA in feline patients, addressing the challenges and advancements in understanding this pervasive disease. Osteoarthritis in cats poses unique diagnostic and therapeutic challenges due to their distinct physiology and behavior, which often complicates early detection and intervention.

Diagnosis of Feline OA:

Diagnosing OA in cats is inherently challenging because of their ability to mask pain and behavioral changes. Traditional indicators of arthritis in other species, such as lameness, are not as overt in cats due to their tendency to modify activity patterns subtly rather than exhibit pronounced limping. Consequently, the diagnosis often relies on a combination of owner observations, physical examinations, and advanced imaging techniques.

Owners may notice changes such as reluctance to jump, decreased activity, or alterations in grooming habits. A thorough physical examination by a veterinarian can help identify pain or discomfort in the joints. However, definitive diagnosis often requires the use of owners questionnaires and imaging techniques such as radiography, which can reveal joint changes typical of OA. Advanced imaging modalities, including MRI and CT scans, can offer more detailed insights into the joint structure and disease progression but may not always be practical in general practice settings.

Management Strategies:

Management of feline OA focuses on multimodal approaches that incorporate pharmacological treatments, dietary adjustments, environmental modifications, and complementary therapies. The goal is to alleviate pain, improve joint function, and enhance the quality of life.

1. Pharmacological Management:

- **Monoclonal antibodies:** New kid on the block, very effective in controlling OA pain, initial data shows to be very safe and efficacious, probably first line of treatment particularly in older patients with comorbidities that makes NSAIDs not a possibility.
- **Nonsteroidal Anti-Inflammatory Drugs (NSAIDs):** NSAIDs are commonly used to reduce pain and inflammation but require careful dosing and monitoring due to potential side effects, particularly on renal function.
- **Analgesics:** Medications like gabapentin might offer additional pain relief, particularly for chronic pain management or in neuropathic pain.
- **Disease-Modifying Osteoarthritis Drugs (DMOADs):** These include glucosamine and chondroitin sulfate supplements that aim to support joint health, although their efficacy is questionable.

2. Dietary Management:

- obesity exacerbates OA.

3. Environmental and Lifestyle Modifications:

- Ensuring the cat's environment accommodates their condition, such as using ramps to help access favorite resting spots and providing warm, comfortable bedding to alleviate joint stiffness.
- Regular, controlled exercise helps maintain mobility and muscle mass, though it needs to be tailored to the individual cat's capabilities and pain levels.

4. Physical Rehabilitation and Alternative Therapies:

- Physical therapy exercises, laser therapy, and acupuncture may support joint function and pain management, though the availability and acceptance of these treatments in feline practice can vary.
- Hydrotherapy is less commonly used in cats compared to dogs, but innovative approaches continue to evolve.

5. Surgical Interventions:

- In severe cases where medical management fails, surgical options such as joint arthrodesis or even joint replacement might be considered, although these are less common in cats and require specialized expertise.

Challenges and Future Directions:

Due to the often-subtle presentation of OA in cats, enhancing awareness among pet owners and veterinarians is crucial for early detection and intervention. Continued research into effective diagnostics, such as biomarker development or non-invasive imaging techniques, may provide breakthroughs in early disease identification.

Additionally, cultivating a collaborative approach between veterinarians, researchers, and pet owners will advance the management of OA in feline patients. This partnership is essential to refine treatment protocols, optimize multimodal management strategies, and ultimately improve the quality of life for cats suffering from osteoarthritis.

In conclusion, while the management of feline OA is challenging, ongoing advancements in diagnostics and therapeutics are promising. By integrating a holistic approach that considers pharmacological, dietary, and lifestyle modifications alongside emerging therapies, veterinarians can better manage this condition, offering cats longer, more comfortable lives. The need for comprehensive research and dedicated clinical trials remains paramount to unlocking future treatments and improving the standard of care in feline OA.

FELINE JOINT LUXATIONS. ANYTHING DIFFERENT TO DOGS

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1. Hip Luxations:**A. Etiology and Pathophysiology:**

- Typically result from trauma such as falls or vehicular accidents.
- Can also be secondary to degenerative joint diseases or congenital abnormalities.

B. Clinical Signs:

- Sudden hind limb lameness.
- Difficulty bearing weight on the affected limb.
- Altered gait and posture.

C. Diagnosis:

- **Clinical Examination:** Assessment of limb position and pain response.
- **Imaging:** Radiographs are essential to confirm dislocation and determine direction (craniodorsal vs. caudoventral).

D. Treatment:

- **Closed Reduction:** Non-surgical manipulation under anesthesia to reposition the femoral head. Questionable success in cats
- **Open Reduction and Internal Fixation (ORIF):** Surgical intervention for irreducible or recurrent luxations. Hip toggle and Ilio-femoral suture
- **FHO (Femoral Head Osteotomy):** Removal of femoral head; considered in chronic cases or when osteoarthritis is present.

E. Prognosis and Recovery:

- Generally good with proper treatment, though complications like re-luxation or arthritis can occur.

2. Elbow Luxations:**A. Etiology and Pathophysiology:**

- Often caused by direct trauma
- lateral displacements is the most common.

B. Clinical Signs:

- Acute forelimb lameness and inability to flex the elbow.
- Swelling and presence of joint deformity.

C. Diagnosis:

- **Clinical Examination:** Palpation for abnormal joint position.
- **Imaging:** Radiographic evaluation to confirm luxation and identify associated fractures.

D. Treatment:

- **Closed Reduction:** Initial attempt at manual repositioning. High percentage of relaxation
- **Surgery:** Farrell technique

E. Prognosis and Recovery:

- Depend on promptness of treatment; prognosis is variable with potential for complications like joint stiffness.

3. Sacroiliac Luxations:

A. Etiology and Pathophysiology:

- Primarily result from high-impact trauma, such as falls or traffic accidents.
- Involves separation of the pelvis from the sacrum.
- Careful if sacral fracture is present

B. Clinical Signs:

- Pelvic limb lameness, asymmetry of the pelvic area.
- Pain response upon manipulation of the pelvis.

C. Diagnosis:

- **Clinical Examination:** Observing limb use and palpating the pelvis.
- **Imaging:** Radiographs assess the degree of displacement.

D. Treatment:

- **Conservative Management:** Rest and pain management for minor cases. With unilateral disease and little displacement
- **Surgical Intervention:** Lag screw luxation or transiliac pin to stabilize the joint

E. Prognosis and Recovery:

- Prognosis is generally favorable with appropriate intervention, although pelvic alignment issues may persist.

Conclusion:

1. Summary of Key Points:

- Importance of early diagnosis and tailored treatment plans.
- Emphasizing rehabilitation and monitoring for complications to enhance recovery outcomes.

2. Future Directions:

- Need for further studies on surgical techniques and long-term outcomes.
- Development of preventive measures and owner education programs.

CARPAL AND TARSAL ARTHRODEIS IN THE FELINE PATIENT, TRICKS AND TIPS

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Carpal arthrodesis

Carpal arthrodesis, a surgical procedure that involves the fusion of the carpal joints, is occasionally required in feline veterinary practice to treat conditions resulting in severe, irreversible joint damage. Cats, being agile creatures, rely heavily on the mobility and flexibility of their limbs for movement, balance, and other essential activities. Therefore, significant carpal joint injuries or diseases can severely impact their quality of

life. While such procedures are more common in dogs, particularly larger breeds, cats can also benefit from arthrodesis in cases of severe carpal trauma or debilitating disease.

This abstract will explore the various aspects of feline carpal arthrodesis, including the relevant anatomy, indications for the surgery, preoperative considerations, surgical techniques, postoperative care, potential complications, and prognosis. The goal is to provide a comprehensive understanding of this procedure to veterinarians, veterinary students, and cat owners considering carpal arthrodesis for their feline patients or pets.

Feline Carpal Anatomy

To fully understand the surgical procedure of carpal arthrodesis in felines, it is crucial to first comprehend the anatomy of the carpal joint. The feline carpus is composed of several small bones arranged in two rows, located between the distal radius and ulna (the bones of the forearm) and the metacarpal bones. These bones are stabilized by ligaments and tendons that allow a wide range of motion.

The key components of the carpal joint in cats include:

1. **Radial Carpal Bone (RCB):** Located on the medial (inside) side of the wrist.
2. **Ulnar Carpal Bone (UCB):** Positioned laterally (outside) on the wrist.
3. **Accessory Carpal Bone:** This bone is located on the caudal aspect of the carpus and serves as the attachment point for flexor tendons.
4. **Proximal and Distal Rows of Carpal Bones:** The carpus is divided into two rows, the proximal and distal rows, comprising several small bones (e.g., the intermedioradial carpal bone, the ulnar carpal bone, and smaller carpal bones).
5. **Carpal Ligaments and Tendons:** The wrist joint is stabilized by multiple ligaments, including the collateral ligaments (medial and lateral), palmar carpal ligament, and various smaller ligaments. Flexor and extensor tendons cross the carpal joint to control wrist movements.

The carpal joint in cats allows for flexion, extension, and limited side-to-side movement. Given the complexity and delicacy of this joint, any significant trauma or disease affecting the carpus can greatly compromise the function of the limb.

Indications for Feline Carpal Arthrodesis

Arthrodesis of the carpal joint is indicated in cases where conservative treatments, such as splinting or non-steroidal anti-inflammatory drugs (NSAIDs), have failed, or when the condition of the joint is irreversibly compromised. The most common conditions that may necessitate carpal arthrodesis in cats include:

1. **Severe Trauma:** Fractures or dislocations of the carpal bones or ligament injuries, such as hyperextension injuries, are the leading causes of carpal instability. In hyperextension injuries, which are relatively common in cats, the palmar ligaments and fibrocartilage are disrupted, causing collapse of the carpal joint.
2. **Degenerative Joint Disease (DJD):** Cats may suffer from osteoarthritis or other degenerative joint diseases affecting the carpus. In advanced stages, these conditions can cause significant pain, inflammation, and decreased mobility.
3. **Immune-Mediated Polyarthritis:** In some cases, immune-mediated conditions, such as rheumatoid arthritis, may result in chronic inflammation and joint destruction that is not manageable through medication alone.
4. **Infectious Arthritis:** Infections in the carpal joint, whether bacterial or fungal, can lead to irreversible joint damage. If the infection cannot be eradicated with medical management and causes significant pain or instability, arthrodesis may be necessary.
5. **Neoplasia:** Tumors affecting the carpal bones or surrounding soft tissues may require resection of the joint, resulting in the need for arthrodesis to restore limb function.
6. **Congenital Deformities:** Rare congenital deformities of the carpus that result in instability or pain may also be corrected through surgical fusion.

Preoperative Considerations

Before performing carpal arthrodesis in a cat, several preoperative assessments and considerations are necessary to optimize the outcome of the surgery.

1. **Physical Examination:** A thorough physical examination should be conducted to assess the overall health of the cat and confirm the extent of the carpal joint pathology. The surgeon must evaluate whether the condition is isolated to the carpal joint or involves other parts of the limb or body.

2. Diagnostic Imaging:

- **Radiographs (X-rays):** Preoperative radiographs are essential to visualize the affected carpal joint and assess the severity of the damage. Radiographs help determine the extent of bone and soft tissue involvement and are useful in planning the surgical approach.
- **Advanced Imaging (CT or MRI):** In more complex cases, or when the cause of the problem is not fully visible on X-rays, advanced imaging techniques such as computed tomography (CT) may be used to provide a more detailed view of the joint and surrounding structures.

Surgical Techniques

The primary goal of carpal arthrodesis is to achieve a stable, pain-free joint through the surgical fusion of the affected carpal bones. The procedure involves removing the damaged cartilage and immobilizing the bones with surgical implants to promote bone fusion.

1. Approach and Preparation

- **Positioning:** The cat is placed under general anesthesia, and the affected limb is prepared aseptically. The cat is positioned in dorsal recumbency with the affected limb facing upwards. A sterile surgical field is created around the carpal joint.
- **Incision:** A dorsal approach to the carpus can be used, depending on the specific anatomy and nature of the condition. The dorsal approach is more commonly used because it provides direct access to the carpal bones and is more familiar to most surgeons. A skin incision is made over the carpal joint, and soft tissues are carefully retracted to expose the carpal bones.

2. Debridement and Joint Preparation

- **Cartilage Removal:** The surgeon removes all articular cartilage from the carpal bones using a combination of curettes, rongeurs, and power burrs. The goal is to expose subchondral bone to create a raw surface that will facilitate bone fusion. Complete cartilage removal is essential for successful arthrodesis.
- **Joint Realignment:** The bones of the carpal joint are aligned in a functional position that mimics normal limb alignment. The degree of carpal extension or flexion is carefully controlled to ensure that the limb will be positioned appropriately after fusion.

3. Internal Fixation

Internal fixation is critical to maintain the stability of the joint while bone fusion occurs. Several methods of fixation may be employed, including:

- **Plates and Screws:** Bone plates, typically hybrid locking compression plates (LCPs), are commonly used to stabilize the carpal bones. The plate is positioned on the dorsal or palmar surface of the carpus and secured with screws inserted into the carpal bones. The plate provides rigid fixation, which is essential for successful bone healing.
- **Pinning:** In some cases, intramedullary pins may be used to provide additional stabilization of the carpal bones. However, pinning alone is generally insufficient for maintaining stability, so it is often used in combination with plates and screws.
- **Bone Grafting:** Autologous bone grafts (harvested from the cat's own body) or allografts (donor bone grafts) may be used to enhance bone healing. Bone grafts provide a scaffold for new bone growth and improve the chances of successful fusion, especially in cases where there is significant bone loss or damage.

4. Closure

Once the internal fixation has been completed and the carpal bones are securely stabilized, the soft tissues are closed in layers. The skin is sutured, and a sterile bandage is applied to protect the surgical site.

Postoperative Care

Postoperative care is critical for the success of carpal arthrodesis. The recovery period can be lengthy, and careful attention is required to ensure proper healing.

Potential Complications

As with any surgical procedure, carpal arthrodesis carries potential risks and complications, including:

1. **Infection:** Postoperative infections can occur at the surgical site or around the implants. Signs of infection include swelling, redness, and discharge. Infections may require antibiotic therapy and, in severe cases, removal of the implants.

2. **Implant Failure:** Plates, screws, or pins may loosen or break, particularly if the cat is not adequately restricted during the healing phase. Implant failure can lead to non-union of the bones and may require revision surgery.
3. **Delayed Union or Non-union:** In some cases, the bones may not fuse as expected, leading to delayed union or non-union. This can result from poor blood supply, infection, or excessive movement. Bone grafting or revision surgery may be necessary.
4. **Limb Dysfunction:** While most cats adapt well to the loss of mobility in the carpal joint, some may experience ongoing discomfort or difficulty using the limb. Persistent lameness may require further evaluation.

Prognosis

The prognosis for cats undergoing carpal arthrodesis is generally favorable, especially when the procedure is performed correctly, and postoperative care is diligently followed. Most cats experience a reduction in height of jump.

Conclusion

Feline carpal arthrodesis is a valuable surgical option for treating severe carpal injuries and diseases that result in joint instability, pain, and loss of function. By fusing the carpal bones and eliminating joint movement, the procedure can restore limb stability and improve the cat's quality of life. While the surgery is complex and carries potential risks, careful preoperative planning, meticulous surgical technique, and diligent postoperative care can lead to successful outcomes in most cases.

Veterinarians considering carpal arthrodesis for their feline patients must weigh the benefits of the procedure against the potential risks and complications, while also ensuring that owners are well-informed about the recovery process and long-term expectations.

Feline Tarsal arthrodesis

Feline tarsal arthrodesis is a surgical procedure performed to stabilize and fuse the tarsal joint, typically in cases where the joint is severely damaged, unstable, or painful due to trauma, degenerative diseases, or other conditions. The tarsal joint, located in the hind limb, is essential for a cat's mobility, balance, and weight-bearing capabilities. Cats are agile animals that rely heavily on their limbs for running, jumping, and climbing, so significant tarsal injuries can have a profound impact on their quality of life.

While tarsal arthrodesis is less commonly performed in cats than in dogs, it can be a highly effective procedure for addressing chronic or severe conditions that affect the stability of the tarsus. This essay provides a comprehensive overview of feline tarsal arthrodesis, exploring the anatomy of the tarsal joint, indications for the procedure, preoperative considerations, surgical techniques, postoperative care, potential complications, and long-term prognosis.

Feline Tarsal Anatomy

To fully appreciate the complexities of tarsal arthrodesis in felines, it is important to understand the anatomy of the tarsal joint and surrounding structures. The tarsus, or hock joint, is located between the tibia and fibula (bones of the lower leg) and the metatarsal bones (bones of the foot). It is a complex joint composed of several smaller articulations, which together allow for flexion, extension, and slight rotational movements. The tarsus plays a critical role in weight-bearing and locomotion.

Key components of the tarsal joint in cats include:

1. **Talus:** The talus, also known as the astragalus, is the bone that forms the primary connection between the tibia and the tarsal bones. It articulates with the tibia and fibula at the tibiotarsal joint, which is the main site of motion in the tarsus.
2. **Calcaneus:** The calcaneus is the largest bone of the tarsus and forms the heel of the cat's hind leg. It serves as the attachment point for the Achilles tendon (common calcaneal tendon), which plays a crucial role in the extension of the tarsus during movement.
3. **Central Tarsal Bone (CTB):** Located centrally in the tarsus, this bone helps to distribute forces across the joint.
4. **Other Tarsal Bones:** The tarsus is composed of several smaller bones, including the lateral and medial malleolus, the tarsal cuboid, and the metatarsal bones. These bones form multiple articulations that contribute to the stability and function of the hind limb.
5. **Ligaments and Tendons:** The tarsal joint is stabilized by multiple ligaments, including the medial and lateral collateral ligaments, which help maintain joint integrity. The Achilles tendon is one of the most prominent tendons in the tarsus, contributing to the extension and propulsion of the hind limb during movement.

The tarsus in cats is a high-motion, high-impact joint, especially during jumping and landing. Therefore, damage to any of the bones, ligaments, or tendons in the tarsus can lead to significant pain and dysfunction.

Indications for Feline Tarsal Arthrodesis

Tarsal arthrodesis is typically indicated in cases where conservative management has failed or when the joint is irreparably damaged. The goal of arthrodesis is to fuse the bones of the tarsus, eliminating joint motion and providing stability to the hind limb. While this results in the loss of normal joint mobility, it can significantly reduce pain and improve the cat's ability to use the affected limb.

The most common indications for tarsal arthrodesis in cats include:

1. **Severe Trauma:** Trauma is one of the leading causes of tarsal joint instability in cats. Fractures, dislocations, or luxations of the tarsal bones or ligamentous injuries, particularly those affecting the collateral ligaments or the Achilles tendon, can result in chronic instability. Hyperextension injuries, often caused by falls from heights (e.g., the "high-rise syndrome" common in urban cats), can lead to collapse of the tarsus.
2. **Degenerative Joint Disease (DJD):** Osteoarthritis, a common degenerative joint disease, can affect the tarsal joint, particularly in older cats or those with a history of trauma or joint disease. In advanced cases, the cartilage deteriorates, causing chronic pain, stiffness, and loss of function. Arthrodesis can provide long-term relief by eliminating the painful joint motion.
3. **Infectious Arthritis:** Infections within the tarsal joint, whether bacterial or fungal, can cause significant damage to the joint structures. When medical management fails to control the infection or the joint is left severely compromised, surgical fusion may be necessary.
4. **Immune-Mediated Polyarthritis:** Cats can develop immune-mediated conditions, such as rheumatoid arthritis, that result in chronic inflammation of multiple joints, including the tarsus. In cases where the joint is permanently damaged by the inflammatory process, arthrodesis may be required to alleviate pain and restore stability.
5. **Chronic Achilles Tendon Rupture:** Injury or rupture of the Achilles tendon can lead to a loss of function in the tarsal joint. In cases where the tendon cannot be repaired or when instability persists, arthrodesis may be required to provide long-term stability to the joint.
6. **Congenital Deformities:** Rare congenital deformities of the tarsal joint that lead to abnormal joint function or pain can sometimes be corrected with arthrodesis. These deformities may involve abnormal alignment of the bones or instability of the joint structures.

Preoperative Considerations

Before performing tarsal arthrodesis, several key assessments and considerations are required to ensure a successful outcome and minimize the risk of complications.

1. Physical Examination

A thorough physical examination is essential to assess the overall health of the cat and determine the severity of the tarsal joint condition. The veterinarian should evaluate the cat's gait, limb function, and level of pain. It is also important to determine whether the problem is isolated to the tarsal joint or whether other joints or parts of the limb are involved.

2. Diagnostic Imaging

- **Radiographs (X-rays):** Standard radiographs are a crucial part of preoperative planning for tarsal arthrodesis. They provide detailed images of the bones and joints, allowing the veterinarian to assess the extent of the damage or deformity. Radiographs can help identify fractures, joint luxations, degenerative changes, and bone infections.
- **Advanced Imaging (CT):** In more complex cases, such as those involving trauma, neoplasia, or chronic degenerative changes, advanced imaging techniques like computed tomography (CT)

Surgical Techniques for Feline Tarsal Arthrodesis

The surgical technique for tarsal arthrodesis involves removing the joint cartilage and stabilizing the bones with internal fixation to promote fusion. The procedure is designed to achieve a stable, pain-free joint by eliminating motion in the tarsus.

1. Surgical Approach and Preparation

- **Positioning:** The cat is placed under general anesthesia, and the affected limb is prepared aseptically. The cat is typically positioned in dorsal recumbency with the affected limb facing upwards. A sterile surgical field is created around the tarsus.

- **Incision:** A dorsal approach to the tarsal joint is commonly used, depending on the specific anatomy of the injury or condition being treated. A skin incision is made over the tarsal joint, and the soft tissues are carefully dissected to expose the underlying bones and joint structures.

2. Joint Debridement and Cartilage Removal

- **Cartilage Removal:** The articular cartilage from the bones involved in the tarsal joint is removed using curettes, rongeurs, or power tools. Complete removal of the cartilage is essential for achieving bone fusion. The surgeon scrapes away the cartilage to expose the subchondral bone, which will serve as the surface for bone fusion.
- **Realignment:** After debridement, the bones of the tarsal joint are realigned in a functional position. The angle of the tarsal joint is carefully controlled to ensure proper limb alignment and to prevent abnormal stress on the surrounding joints. The goal is to place the tarsus in a weight-bearing position that allows for optimal function after fusion.

3. Internal Fixation

Internal fixation is critical for maintaining stability during the healing process. Several types of internal fixation may be used for tarsal arthrodesis, including:

- **Plates and Screws:** Bone plates, particularly locking compression plates (LCPs), are commonly used to stabilize the tarsal joint. These plates are designed to provide rigid fixation by securing the bones in place with screws. The plate is positioned along the lateral or dorsal surface of the tarsus and is attached to the bones with screws. The use of locking screws improves stability, especially in smaller bones.
- **Bone Grafting:** Bone grafts, either autografts (harvested from the cat's own body) or allografts (donor bone), may be used to enhance fusion. Bone grafts provide a scaffold for new bone growth, which is critical for achieving successful arthrodesis, particularly in cases with significant bone loss or poor bone quality.

4. Closure

After the internal fixation is secured and the bones are in the correct position, the soft tissues are closed in layers. The skin is sutured, and a sterile bandage is applied to protect the surgical site and reduce swelling.

Postoperative Care

Postoperative care is essential for a successful outcome in tarsal arthrodesis. Recovery can be a prolonged process, and diligent care is required to ensure proper healing and minimize complications.

Restricted Activity

Strict confinement is critical for several weeks after surgery. Cats should be confined to a small, safe area or crate to prevent running, jumping, or other activities that could compromise the surgical repair. Gradual reintroduction of controlled activity can begin once healing has progressed.

Follow-up Radiographs

Radiographs are typically taken 6–8 weeks after surgery to assess bone healing and the integrity of the implants. Additional radiographs may be required at later stages if healing is delayed or if there are concerns about implant failure. Bone fusion can take several months to complete.

Potential Complications

As with any major orthopedic surgery, feline tarsal arthrodesis carries certain risks and potential complications. Some of the most common complications include:

1. **Infection:** Postoperative infections at the surgical site or around the implants can occur. Signs of infection include swelling, redness, discharge, and pain. Infections may require antibiotic therapy, and in severe cases, implant removal may be necessary.
2. **Implant Failure:** Plates, screws, or pins may loosen or break, particularly if the cat is too active during the recovery period. Implant failure can lead to non-union or delayed union of the bones and may require additional surgery.
3. **Delayed Union or Non-union:** In some cases, the bones may fail to fuse properly, resulting in delayed union or non-union. This can be caused by poor blood supply to the area, infection, or excessive movement. Bone grafting or revision surgery may be required in such cases.
4. **Persistent Lameness:** While many cats regain functional use of the limb, some may experience ongoing lameness or discomfort. This may be due to residual soft tissue damage, improper alignment of the joint, or complications with the implants.

- 5. Limb Dysfunction:** Some cats may have difficulty adapting to the loss of tarsal joint motion, leading to altered gait or limb dysfunction. However, most cats adjust well over time.

Prognosis

The prognosis for cats undergoing tarsal arthrodesis is generally positive, provided that the surgery is performed correctly and postoperative care is closely followed. Most cats experience significant relief from pain and regain functional use of the limb, despite the loss of tarsal joint mobility. However, the success of the procedure depends on several factors, including the extent of the initial injury or disease, the cat's overall health, and adherence to postoperative care.

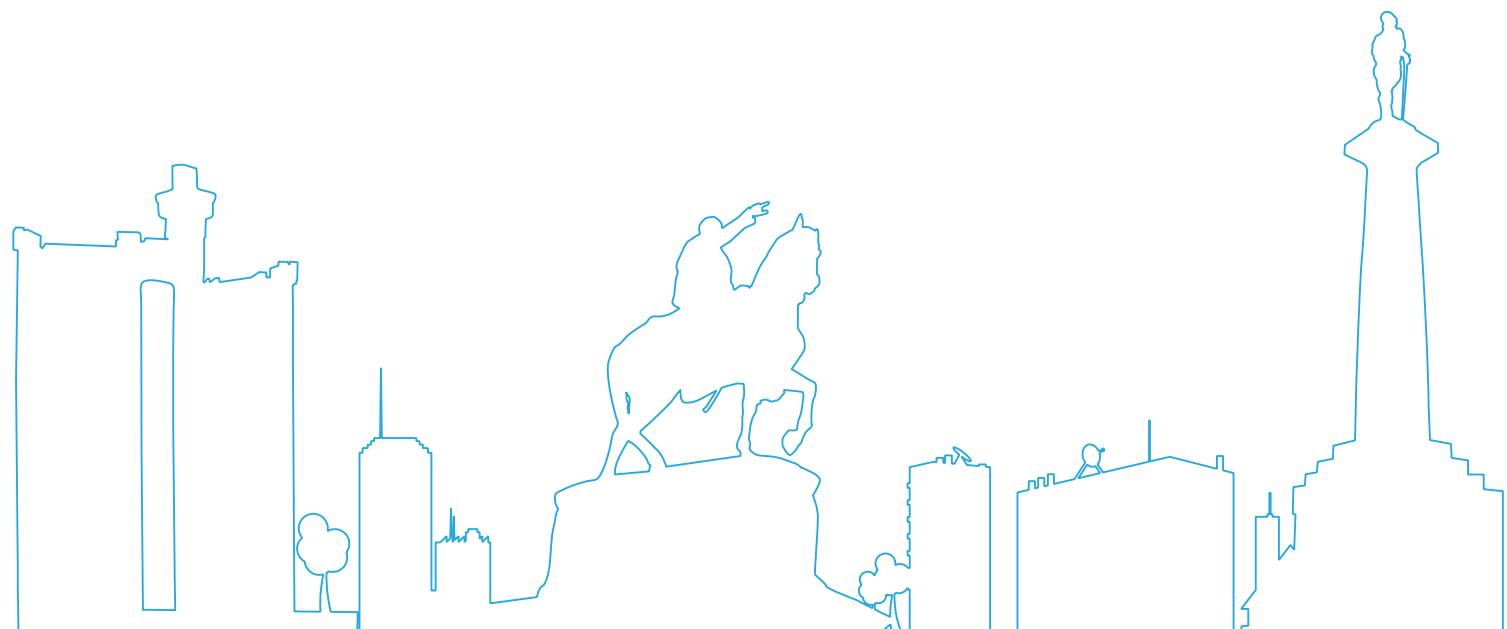
With appropriate care and management, many cats can return to normal activity levels and enjoy a good quality of life after tarsal arthrodesis.

Conclusion

Feline tarsal arthrodesis is a valuable surgical option for addressing chronic or severe tarsal joint instability, pain, and dysfunction. By fusing the bones of the tarsal joint, the procedure provides long-term stability and pain relief, enabling cats to regain functional use of the affected limb. While the surgery is complex and requires careful preoperative planning, meticulous surgical technique, and diligent postoperative care, the outcome can be highly successful for most cats.

Veterinarians must weigh the benefits of tarsal arthrodesis against the potential risks and complications, and ensure that cat owners are fully informed about the recovery process and long-term expectations.

SOFT TISSUE SURGERY



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EERVC 2024 Lectures

1. What to do with lymph nodes in surgical oncology?
2. Surgery of facial tumours
3. What can you learn from my complications?
4. Getting started in thoracic surgery
5. Easy tips and tricks for wound closure
6. Surgery of prostatic diseases

Laurent graduated from Paris' vet school, the Ecole Nationale Vétérinaire d'Alfort, in France in 1995 and was assistant instructor in the anatomy department the following year. He then qualified for a 2-year surgical internship in the same school and later completed a Master of Science in Biology and Physiology of Circulation and Respiration, as well as a university degree in Experimental Surgery and Microsurgery.

After having worked for 3 years in a large private practice outside Paris, he spent 4 years at the Centre Hospitalier Vétérinaire Frégis, near Paris, where he completed an ECVS residency. He became a diplomate of the European College of Veterinary Surgeons in 2008 and was recognized as a specialist in small animal surgery by the Royal College of Veterinary Surgeons in 2012. Laurent worked at VRCC Veterinary Referrals in Essex, United Kingdom, where he was one of the full-time soft-tissue surgeons and directors from 2006 to 2014. He moved to the Oncology and Soft Tissue Surgery department of Fitzpatrick Referrals in October 2014. Laurent is a member of several professional boards and committees, regularly contributes to international publications and book chapters, and gives lectures and presentation in many countries. His fields of interest include surgical oncology, reconstructive surgery and general soft tissue surgery.

WHAT TO DO WITH LYMPH NODES IN SURGICAL ONCOLOGY

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The role of lymph nodes in veterinary oncology has become increasingly crucial in the last decade. A comprehensive understanding of their significance is essential for effective management in surgical oncology. The status of lymph nodes (LNs) holds significant prognostic value in the staging of various cancers in both human and veterinary medicine. Resection of metastatic nodes has shown to enhance outcomes in specific cancer types.

The interested reader is referred to a recent review of the current approach to lymph nodes in veterinary oncology[1].

STAGING

The sentinel lymph node(s) (SLN) is/are the first lymph node(s) draining a tumour. Sentinel Lymph Nodes are pivotal in determining the metastatic status of tumours that spread through the lymphatic route, such as carcinomas, mast cell tumours, and melanomas. For some of these tumours, we know that if the sentinel lymph node is negative, the tumour is very unlikely to have metastasised elsewhere. Their identification is critical, as a negative SLN often indicates a low likelihood of metastasis in specific tumours.

The sentinel lymph node (i.e., the lymph node effectively draining a particular tumour in a particular patient) is often different from the regional lymph node (RLN; 28–63% of cases), which is the tumour logically expected to drain the area of the tumour based on anatomical knowledge. This is why determining which is the sentinel lymph node is important on an individual basis. Accurate identification of the true sentinel lymph node is essential for precise staging.

Preoperatively, the SLN can be identified by several methods, including lymphoscintigraphy, radiographic lymphography, CT lymphography, contrast-enhanced ultrasound, with success rates varying from 40 to 100%. Intraoperatively, several methods exist to identify the SLN and/or help finding draining LNs, including the injection of methylene blue, fluorescein, near infrared lymphography (ICG). Anchors are another aid in finding lymph nodes identified on preoperative imaging.

It's noteworthy that normal-sized lymph nodes do not necessarily guarantee a tumour-free status. For instance, one study showed that 42% of dogs with head and neck tumours had seemingly normal, but metastatic, lymph nodes.

For radiographic lymphography, a lipophilic contrast agent can be used. It accumulates in lymph nodes and therefore radiographs are typically taken a few hours after the injection[2]. Alternatively, a recent study showed good results with a hydrophilic contrast agent, with all lymph nodes identified on radiographs taken 3 mins after injection.[3]

For CT lymphography, a hydrophilic contrast agent is used, and images taken within minutes of injection. For contrast ultrasound, a specific contrast medium and software are required. [4]

TREATMENT

The therapeutic benefits of removing metastatic lymph nodes are increasingly evident in various tumour types. This has most notably been shown for mast cell tumours and anal sac carcinomas.

Illustrating this, a study involving 149 dogs with Stage II Mast Cell Tumours (MCTs) demonstrated that those undergoing lymphadenectomy had significantly lower risks of tumour progression and death compared to those without the procedure.

CONCLUSION

The key takeaway is that Sentinel Lymph Nodes are often distinct from Regional Lymph Nodes. Sampling SLNs enhances staging accuracy and excising them correlates with improved prognoses in selected tumours. This underscores the importance of tailored approaches in veterinary surgical oncology, recognizing the nuanced nature of lymph node involvement.

A comprehensive grasp of the role of lymph nodes in veterinary surgical oncology significantly impacts patient management and outcomes. Keeping abreast of evolving techniques for lymph node identification and acknowledging individual variability ensures a nuanced and effective approach in veterinary oncology.

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SURGERY OF FACIAL TUMOURS

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A wide variety of tumours are encountered in all portions of the face. Surgically, they often constitute a technical challenge because of the frequent proximity of important structures, which make seeking wide margins not always possible, and because of the lack of spare tissues nearby to reconstruct large defects. They are also more difficult to treat because of the natural repulsion that owners (and practitioners) can have when the disease or its treatment causes significant distortion of the face, which we humans use to read feelings and emotions. As a consequence, surgeries are more impressive when involving the face, regardless of their real, objective impact on the patient's function and quality of life.

The degree of self-awareness of animals is unknown, but they do not seem to suffer in any way from cosmetic degradations, contrary to human beings, and a large number of facial surgeries do not cause any long-term functional deficits. It is therefore important to be sufficiently familiar with the treatments available and to evaluate as objectively as possible their consequences, placing the patient first, and his family second.

TUMOURS OF THE FACE: WHAT CAN BE REMOVED?

The most common tumour types encountered on the face of dogs and cat include mast cell tumours, squamous cell carcinomas, soft tissue sarcomas and melanomas. Tumours of the facial bones (e.g. osteosarcoma, chondrosarcoma, multilobular tumour of bone (MLTB, formerly multilobular osteochondrosarcoma, MLO)) are also an indication for facial mass resection and reconstruction.

Non-specifically, the preoperative work-up of facial mass include biopsy (fine-needle aspirates, core-needle biopsies or surgical biopsies) and staging. As for any other tumour, a histopathological diagnosis will be preferred over a cytological diagnosis every time the knowledge of the precise type and grade of the tumour will significantly impact on the type and dose of surgery to be sought.

The feasibility of surgery is best determined from advanced imaging. Computed tomography, in particular, is particularly useful as it allows rapid scanning of the head and neck, as well as chest to screen for distant spreading in the lungs. It allows a good appreciation of the extension of the tumour in all planes, both of its soft tissue and bone components. Mutiplanar CT reconstructions are particularly valuable to determine whether ideal margins can be sought or not, and to position the resection lines to follow at surgery. Real-size 3D-printings of the skull can be obtained from the CT scan, which allows to simulate the bony sections to be performed. In addition, such models can be gas-sterilised and used during the procedure as real-scale anatomical guides. Lastly, if biocompatible materials are used for 3D-printing, they can be used to replace missing portions of bone. The replacement implant is often modelled as the mirror image of its contralateral equivalent as, obviously, the portion removed is anatomically abnormal. As it becomes increasingly available and affordable, 3D-printing will undoubtedly become commonplace in these indications.

In general, small animals tolerate wide resections of their face and head very well. Oral tumours are outside the scope of this text, but some facial resections involve portions of the maxillae or mandibles: for instance,

some tumours require en-bloc partial resections of the orbit and maxilla. Some subcutaneous or bony tumours of the nose require partial maxillectomy for complete resection. Extensive resections of the maxillae and mandibles are also well tolerated in most cases.

The potential difficulties associated with the resection phase of surgery pertain mostly to orientation, which requires a solid knowledge of anatomy but can be aided by 3D models as explained earlier, and haemostasis. Profuse bleeding can be encountered when extensive bone resections are required, especially of the caudal maxillae or orbits.

TUMOURS OF THE FACE: HOW TO RECONSTRUCT?

The reconstruction phase can be the most difficult part of a maxillofacial surgery, if the skin or oral mucosa cannot be spared, as both the face and the oral cavity lack loose tissues readily available for reconstruction. This is more or less a problem depending on species and breeds. Cats tend to have less facial soft tissues than dogs. Among dogs, some have looser facial soft tissues than others. Brachycephalic dogs, for instance, tend to have large amounts of facial soft tissues, which facilitates facial reconstructions. Dolicocephalic dogs, reversely, tend to have less soft tissues available for reconstruction. Their upper lips, for instance, are tighter and smaller than those of a mesocephalic dog (e.g., Labrador). Therefore, when considering reconstruction options, it is important not to strive to stick to a preconceived plan, but to adapt to each particular situation in each individual patient.

Although free skin grafts can be used on well-vascularised tissues or granulation tissues of the face as much as on many other areas of the body, they are uncommonly chosen for facial reconstruction because of the inherent technical difficulties related to having them tacked to underlying tissues and immobilised. In many areas of the head, only very little, if any, soft tissues remain in depth of the defect to be reconstructed. When bone resections are involved, the reconstruction frequently consists of covering a cavity (e.g., nasal cavities, sinuses). It is therefore valuable to use reconstruction techniques which are applicable on poorly vascularised or avascular wounds. In this indication, skin flaps are ideal as they come with their own vascularisation. The general principles of skin flap design and application apply for facial reconstruction as for the rest of the body and are outside the scope of this text. A panniculus muscle, the platysma, is present on the lateral aspect of the face. When elevating flaps from this portion of the face, it is critical to undermine the skin in depth of this muscle. Care should be then taken to preserve the branches of the facial nerve when undermining the skin. Where no panniculus muscle is present, the skin must be undermined flush with the underlying fascia or periosteum to preserve the subdermal plexus.

Subdermal skin flaps for facial reconstruction

As for any reconstruction, the essence of facial reconstruction is to locate reachable expandable soft tissues which could be transposed to the defect to cover. Except in brachycephalic dogs, little skin and subcutaneous tissues are available for reconstruction on the rostral portion of the face. The skin and subcutis are much looser on the neck and lateral aspect of the face, and larger subdermal skin flaps are most commonly harvested from these areas. In many dogs and cats, flaps can also be elevated from the top of the head. Depending on the respective positions of the excess skin-subcutis and wound, advancement, rotation, transposition or interpolation flaps can be used.

Axial pattern skin flaps for facial reconstruction

A limited number of axial pattern flaps are used for facial reconstruction. Two flaps elevated from the neck (omocervical, caudal auricular) and 2 flaps elevated from the head (superficial temporal and angularis oris) are most commonly used. The 2 former can be used to cover portions of the lateral and caudo-dorsal aspects of the head. The angularis oris skin flap can reach the tip of the nose [1].

The base superficial temporal axial pattern flap is along the zygomatic arch and its width is roughly that of the zygomatic arch. It is partly determined by the laxity of the skin on the head and the possibility to close the donor site primarily after transposition of the flap. The flap can be extended in length to the contralateral mid-point of the orbital rim in a ventrodorsal axis (Figure 1).

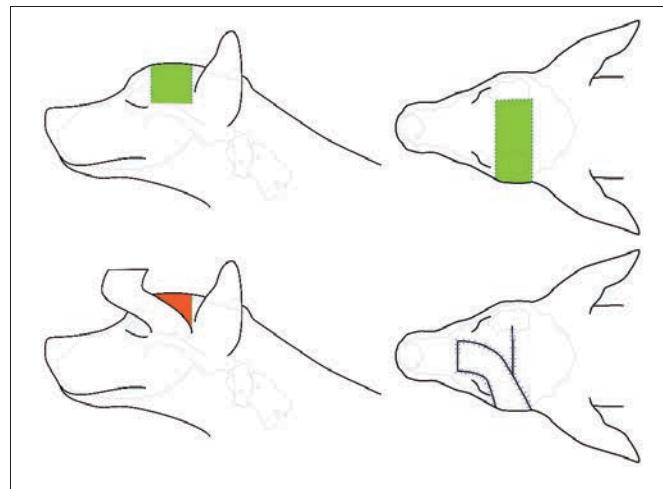


Figure 1: Superficial temporal axial pattern flap

The base of the angularis oris axial pattern flap (AOAPF) is centred on the commissure of the lips. The dorsal border of the flap follows the ventral border of the zygomatic arch in a slightly dorsal and caudal direction. The ventral border of the flap is parallel to its dorsal border and follows the ventral border of the mandible. The flap can be extended safely to the base of the ear canal or with a greater risk of apical necrosis, to the wing of the atlas (Figure 2). It can be rotated dorsally to reconstruct the lateral side of the nose, or ventrally to reconstruct intermandibular defects [1].

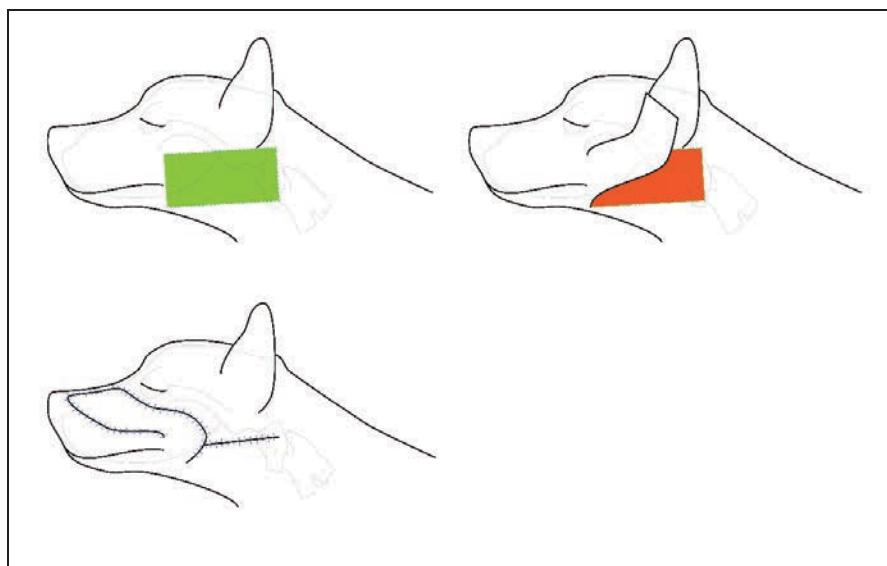


Figure 2: Angularis oris axial pattern flap

Reconstruction of eyelids

Eyelids serve as essential protective structures for the eyes. They shield the delicate ocular surface from debris, foreign objects, and excessive light. Blinking facilitated by eyelids helps distribute tears, maintaining proper moisture and lubrication on the cornea. Their rapid closure during potential dangers safeguards the eyes from harm. Their inner surface is made of mucosa and, ideally, a mucosal inner layer should be provided when large full-thickness defects of the eyelids are reconstructed. Numerous techniques exist, only a couple are listed here.

Simple Two-Layer Closure:

When the removed margin length is small, a simple two-layer closure can be performed. This involves suturing the tarsus and orbicularis muscle layer horizontally with a mattress suture. Then, the eyelid margin

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is sutured with a figure-of-eight suture. Care should be taken to prevent exposure of sutures through the palpebral conjunctiva. For very small resections, the first step can be skipped.

Wedge and Pentagonal Resection:

When reconstructing the eyelid margin using simple closure or a semicircular flap, a wedge or pentagonal mass resection is recommended. This involves excising tissue in a way that forms a wedge or pentagon shape with the eyelid margin as its base. The sides of the wedge or pentagon should be of equal length to ensure proper apposition.

Sliding Pedicle Advancement Flap:

For larger defects involving more than one-third of the eyelid margin, the sliding pedicle advancement flap is used. A rectangular excision is performed, and skin incisions are made to create a flap that can be shifted to cover the defect. The flap's leading edge extends slightly beyond the eyelid margin. The flap can be lined with conjunctiva from nearby areas to prevent corneal irritation.

Third Eyelid–Skin Flap Reconstruction of the Lower Eyelid

This technique can be used when the third eyelid can be preserved. Its external mucosal surface is stripped of the underlying cartilage, leaving a raw surface against which any subdermal flap can be applied. The internal mucosal lining of the third eyelid therefore provides protection for the corneal.

Lip-to-Lid Flap:

The lip-to-lid flap technique uses a segment of the upper lip to reconstruct large eyelid defects. A full-thickness labial flap is created at a specific angle, including the buccal mucosa. The flap is elevated, and the buccal mucosa defect is sutured. The flap is turned into the eyelid defect, and mucosal surfaces are sutured. The transposed lip margin is sutured to the recipient eyelid margins for a two-layer closure. This technique can be used to reconstruct either lower or upper eyelid defects.

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CAN YOU LEARN FROM MY COMPLICATIONS?

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All surgeons have complications, often more than they would care to admit. It is however said that it is in the face of complications that surgeons show their value. I would agree and believe this is because managing complications requires not only the know the "how", but also to understand the "why" and have a deep understanding of the biologic grounds of surgical principles. In other words, it requires to be an achieved surgical biologist, not a mere technician.

It is obviously impossible to discuss in detail how to all possible complications. The list of potential complications is long and unfortunate or inadequate surgeons add to it every day. It is however possible to take some perspective to try to figure out the common features of their management and help defining how surgeons should react when facing them.

ANTICIPATE THEM

An ounce of prevention is worth a pound of cure, or more appropriately, a stitch in time saves nine. This applies to complications as to anything else. Learning to recognise which patients, which diseases and which surgeries are associated with higher risks of complications helps limiting this risk. First of all, by staying away from lost battles. Good surgeons know *how to operate*, better surgeons know *when to operate* and the best surgeons know *when not to operate*. A fair number of postoperative complications results from

bad surgical indications. It is obviously always to find so with hindsight, but it remains true objectively. Fighting lost battles is in nobody's interest: not the patient's, not its owners', not the veterinary team's.

Knowing the risk factors for each disease and each procedure helps anticipating how likely and how severe potential complications are. This allows steering away from surgery if the risks outweigh the expected benefits, or at the very least helps advising clients clearly about the risks carried.

Generally, a good number of complications are associated with the poor general condition of the patient or with concurrent diseases and their treatments (e.g., haemostatic disorders, endocrine diseases). Hypoalbuminaemia, for instance, is a universal risk factor for surgical patients. When the plasmatic level of albumin is below 20 g/L, the risk of postoperative complication increases as wound healing deteriorates. It is particularly critical in gastrointestinal surgery in which wound dehiscence translate in septic peritonitis.

Fundamentally, avoiding complications lies in respecting the patient's biology and physiology. This is the object of respecting Halsted's very few and simple rules of atraumatic surgery. In addition, good knowledge of the pathophysiology of the treated disease and of the effects the surgical procedure has on it is essential to understand the roots of complications and how to prevent them.

DETECT THEM. ACKNOWLEDGE THEM

Detecting complications early is essential to limiting their severity and successfully treating them. Here again, the best way to detect complications early is to anticipate them, know which complications are most likely and actively monitor specific parameters associated with them. Unfortunately, some complications are uncommon or never experienced before and are therefore harder, if possible, to anticipate. It is therefore important to have a baseline surveillance protocol for all surgical patients. The heavier the surgery and the more important the organs operated on, the greater the risk of severe complications and the closer the monitoring should be.

The systematic postoperative monitoring of surgical patients should have 3 axes: assessment of the general condition of the patient (vital functions and systemic parameters), of the function of the operated system (digestive signs after gastrointestinal surgery, neurologic function after neurosurgery, locomotion after orthopaedic surgery, etc.) and of the surgical wound.

The last obstacles to the early detection of complications sadly are surgeons themselves. Mark M. Ravitch, paediatric surgeon observed that "the last man to see the necessity for reoperation is the one having performed the operation". Surgeons tend to be ostriches and be the last to admit that a patient they operated on may be complicating. If this only was deleterious for their ego, this would be of little consequence, but patients unfortunately are the ones ultimately paying the price for this delayed acknowledgement of complications. Detecting severe complications, such as septic peritonitis, early is a major determinant of patient survival. Being self-aware and realising this natural tendency helps fight it. Being humble and realising that all surgeons have complications help accepting them. In fact, it takes a good surgeon to be relaxed and remain reasonably self-confident in the face of complications, provided their number of complications remain within normal limits, obviously. In fact, to the educated observer (junior colleagues, support staff, etc.), a well-managed complication is probably the best benchmark of the value of a surgeon.

TREAT THEM

When a complication has developed, it is important to try to determine its cause. In the vast majority of cases, it lies in the disrespect of biology. Going against biology will invariably fail. Yet it is easy to ignore and persist. If a wound has broken down a couple of times already, it is futile to try and close it again grossly the same way with larger-gauge, tighter sutures. The successful approach will involve understanding the reasons for the repeated dehiscence and chose a closure method aiming at addressing them.

From experience, regardless of a surgeon's skills and expertise, when facing a complication seemingly difficult to treat, it is essential to get back to the fundamentals of surgery, especially if the complication is rare and unexpected. Complications are unfortunately unfrequently and only superficially covered in most textbooks and finding guidance as to how to address very specific ones is not easy. Surgeons must therefore often find the solution themselves and this requires going back to basics. Thinking again in terms of biology and find where the surgical treatment has gone against it. Reviewing essential points including the patient's general condition and concurrent diseases, the pathophysiology of the treated disease, the consequences of the surgery performed, the vascularisation of the operated organs and surgical approach, etc. The practical implications of the conclusions of this review are sometimes hard to accept or implement, as they can be heavy, disappointing or hardly possible, but they must be objectively determined. At the

very least, it allows discussing options and expectable results with owners and setting clear and acceptable objectives and limits in treatment. Nothing is costlier than a complication, both in terms of finances and morale, especially if it drags on. It is therefore essential, here again, not to embark on fighting lost battles. It is however more difficult to recognise these lost battles when it comes to complications. Any guilt the surgeon may feel, consciously or not, makes this even harder and in surgeons will more easily embark on treatments they may otherwise deem unreasonable when it comes to treating their complications.

Lastly, knowing our limits help both preventing and treating complications, by not embarking on surgeries beyond our abilities, in terms of biology, technique or facilities, and by knowing when to seek help. It is difficult to pass over or refer a complication, but it should be done if it is in the patient's best interest. The issue is almost invariably only in our minds: good surgeons will know that complications happen, having their own, and will not wrongly estimate colleagues seeking help or referring them.

REFLECT AND LEARN

Complications will happen. Our duty as surgeon is that no complication is ever "in vain". We must learn from each single of our complications. There is always something to be learnt from them. I would push to say that we only learn from our complications. If we never have complications, we can be perfectible or even wrong without ever knowing it. Only by approaching complications as described earlier, with humility and as much objectivity as possible, can we grow from them and make sure we limit the risks of encountering the same complication again. We owe it to our patients who suffer them.

GETTING STARTED IN THORACIC SURGERY

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Anaesthetic considerations

Depending on the underlying condition requiring thoracic surgery, the patients may suffer from respiratory compromise before surgery and require preoperative optimisation.

Patients undergoing thoracic surgery require mechanical ventilation from the moment the thorax is entered until it is closed, and negative pressure is re-established in the pleural cavity. However, it is important to monitor the ventilation pressure and to keep it moderate (i.e., around 15 to 20 cmH₂O) to prevent any barotrauma to the lungs. In addition, it is important to avoid restoring a void in the pleural cavity or re-expanding the lungs too aggressively, especially in patients whose lungs have been compressed for prolonged periods (e.g. large chest masses, chronic diaphragmatic hernias). Too rapid lung re-expansion exposes the patient to postoperative pulmonary oedema, which can be fatal. It is better to restore a moderate negative pressure in the pleural cavity at the end of surgery, allowing effective spontaneous ventilation of the patient in the immediate postoperative period, and progressively recreate the pleural void over 12 to 48 hours using the thoracic drain in place.

In addition to systemic analgesics, local anaesthetics are used during thoracotomy procedures. Most often, intercostal injections of local anaesthetics are performed before closure of the thoracic cavity (e.g. total dose of 2 mg/kg of bupivacaine in dogs). In addition, in the postoperative period, similar doses of bupivacaine can be instilled in the chest drain every 3 to 6 hours. The bupivacaine instilled intrapleurally is best diluted with 0.9% sodium chloride and administered slowly, as it can be quite painful in itself if administered rapidly and in a concentrated form.

Surgical approach

Lateral (intercostal) thoracotomy

This approach is rapid and technically easy. It provides good exposure of dorsal thoracic structures (heart base, thoracic trachea, carina, lymph nodes). However, it only provides access to a limited portion of one side of the chest. It is therefore only indicated for unilateral affections and requires that the affected side be known prior to surgery. When entering the thorax for lung lobectomy, it is preferable to have precisely determined the affected lobe in order to select the most adapted intercostal space for the approach. When in doubt as to the most appropriate of 2 intercostal spaces, it is usually preferable to choose the most caudal one as ribs can be more effectively retracted cranially than caudally.

	Right lung	Left lung
Cranial lobe	4, 5	4, 5
Middle lobe	5	-
Caudal lobe	5, 6	5, 6
Accessory lobe	5, 6	-

Table 1: Intercostal spaces of choice for lung lobectomies

	Right	Left
Heart, pericardium	4, 5	4, 5
PDA, PRAA		4, 5
Oesophagus (cranial)		3, 4
Oesophagus (caudal)	7 – 9	7 – 9
Thoracic duct (dog)	8-10	
Thoracic duct (cat)		8-10

Table 2: Intercostal spaces of choice to reach intrathoracic structures

For a lateral intercostal thoracotomy, the animal is placed in lateral recumbency opposite to the side to be operated (Figure 1). The affected hemithorax is entirely clipped and prepared for aseptic surgery. The ipsilateral thoracic limb is maintained mildly protracted to expose the cranial portion of the thoracic wall. The chosen intercostal space is identified by palpation. The skin and cutaneous trunci muscle are incised along this intercostal space. The latissimus dorsi muscle is then visible. It can either be retracted dorsally, which has my preference, or incised in its ventral portion, parallel to the underlying intercostal space. The bellies of the serratus ventralis muscle surrounding the chosen intercostal space are separated and ventrally, depending on the location, the scalenus or external oblique muscle is incised. The thoracic cavity is entered by blunt dissection through the intercostal muscles and the parietal pleura. This creates a pneumothorax, allowing the ipsilateral lung to fall away from the thoracic wall and thereby decreasing the risk of iatrogenic injury during the rest of the approach. The intercostal muscles of the selected intercostal space are then sectioned along a line parallel to this space, in its caudal half (closer from the cranial border of the caudal rib than from the caudal border of the cranial rib), to avoid iatrogenic injury to the intercostal nerve, artery and vein. Ventrally, attention is paid not to damage the internal thoracic artery and vein, which course inside the thoracic cavity. The borders of the rib next to the open intercostal space are protected with moist swabs and an autostatic retractor (e.g. Finochietto) is used to spread this space open.

When the intrathoracic step of the procedure is completed, a chest tube is placed under direct visual control (Figure 2). The retractor is removed and the thoracic cavity is closed by placing sutures around the ribs defining the open intercostal space. Monofilament absorbable sutures can be used to this purpose. Attention is paid not to damage the intercostal nerve, artery and vein caudal to the caudal rib when passing the suture caudal to it. In addition, care is taken not to overtighten the sutures to avoid maintaining the ribs around the open intercostal space too close, which is likely to cause unnecessary pain in the postoperative period. The latissimus dorsi muscle is either released or sutured, and the more superficial layers are closed routinely.

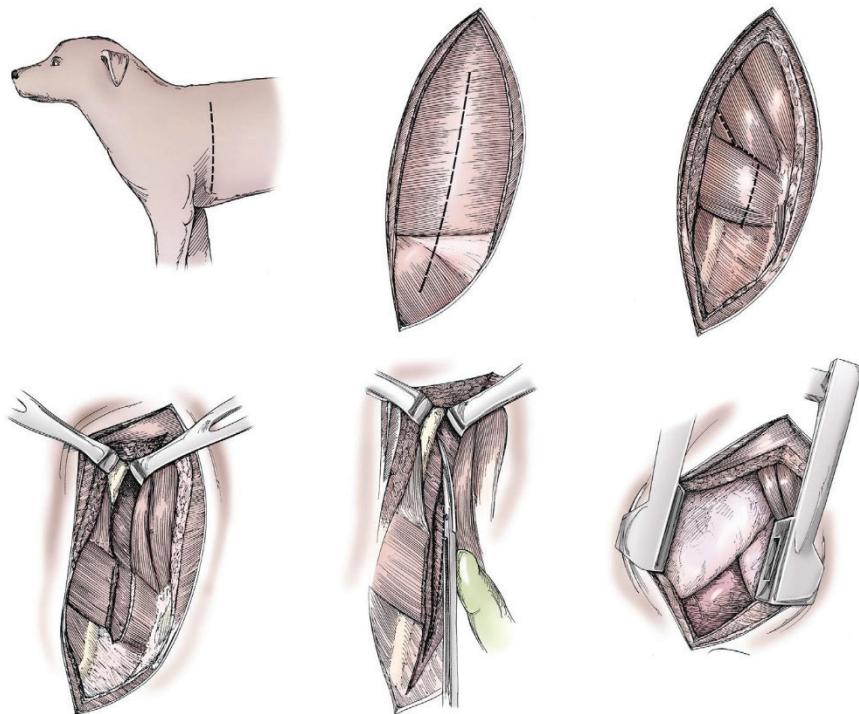


Figure 1: Lateral thoracotomy¹

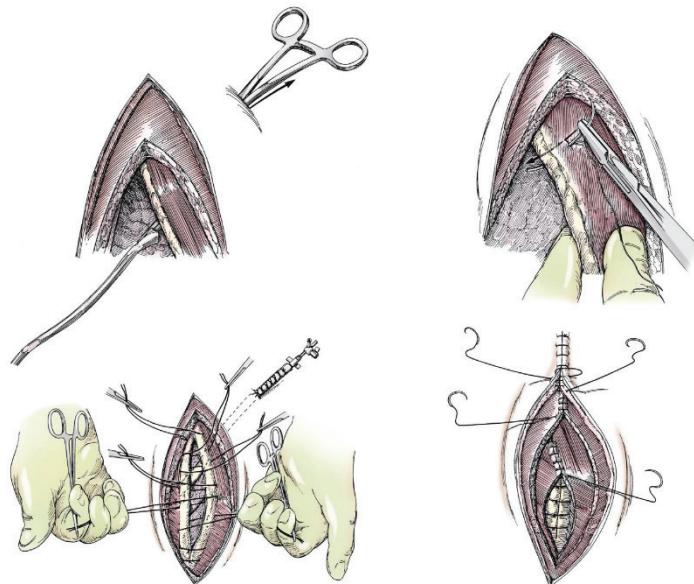


Figure 2: Lateral thoracotomy closure¹

Median sternotomy

For a median sternotomy, the animal is placed in dorsal recumbency (Figure 3). The ventral to $\frac{3}{4}$ of the thorax, caudal portion of the neck and cranial portion of the abdomen are clipped and prepared for aseptic surgery. The skin is incised on the ventral midline above the sternum. The sternum is exposed on the midline by minimal elevation of the insertions of the pectoral muscles on it. The sternum is then divided longitudinally using an oscillating saw, under constant irrigation to prevent thermic osteonecrosis. As rapidly as possible, a small retractor (e.g. Gelpi) is placed in the wound and the interior of the thoracic cavity is palpated to ascertain the absence of adhesions and minimise the risks of iatrogenic injury to intrathoracic organs during the rest of the approach. Whenever possible, efforts are made to avoid dividing the entire length of the sternum. Leaving the cranial-most or caudal-most portion of the sternum intact, and possibly both, will markedly increase the stability of the sternal closure in the postoperative period and reduce the risks of wound healing complications. When the sternum is divided, the borders of the wound are protected with moist swabs and an autostatic retractor is placed to maintain the wound open.

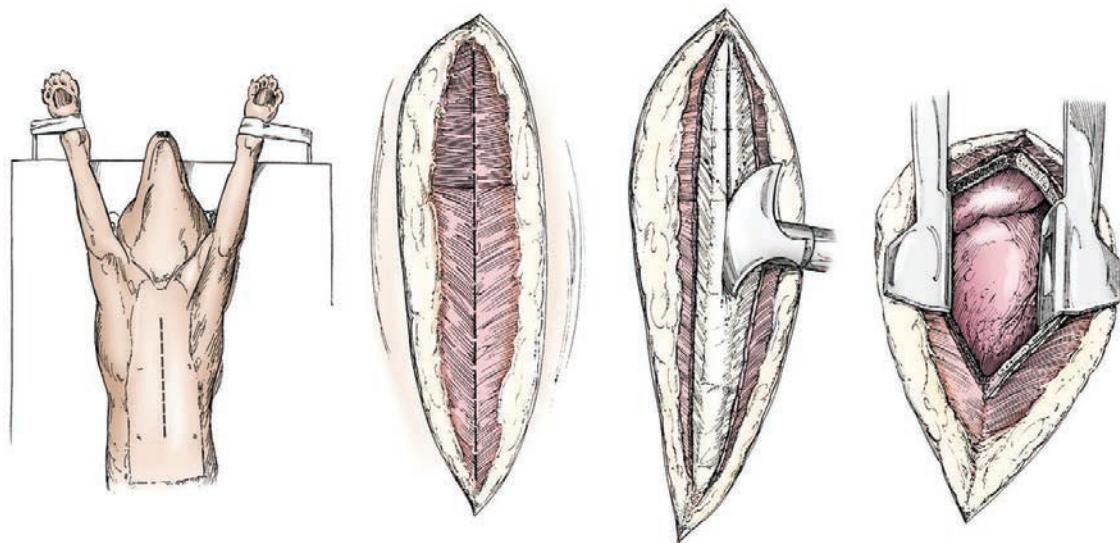


Figure 3: Median sternotomy¹

When the intrathoracic step of the surgery is completed, a chest drain is placed under direct visual control and the sternum is repaired by placement of sutures or wire in a figure of eight around the sternebrae (Figure 4). Closing the sternum with metallic wires is preferred by some authors on the account that it provides a more rigid and stable repair, especially in large dogs². I tend to use large non-absorbable monofilament sutures (e.g. polypropylene), irrespective of the dog's size, provided at least one of the sternum extremities (manubrium or xiphoid sternum) could be left intact. The more superficial layers are closed in a routine fashion.

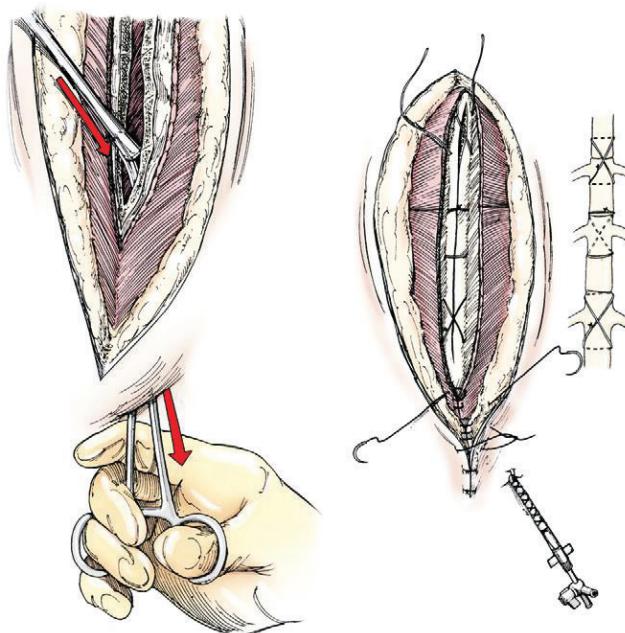


Figure 4: Median sternotomy closure¹

Thoracoscopy

Thoracoscopy is used in small animal surgery for an increasing range of diagnostic and therapeutic purposes³⁻⁶. Diagnostic procedures include visual inspection of the thoracic cavity and thoracoscopic biopsies (e.g. masses, pleura, lymph nodes, lungs). An increasing number of therapeutic procedures have also been reported, including partial and total lung lobectomies, pericardectomy, thoracic duct ligation, tumour resections (e.g. thymoma, atrial tumour) and correction of vascular abnormalities (patent ductus arteriosus, persistent right aortic arch). The description of these techniques is beyond the scope of this text and the reader is referred elsewhere for further information³⁻⁸.

Lung lobectomies

Partial lung lobectomy

Partial lung lobectomies are performed to preserve a portion of a lung lobe whilst excising its apical portion to resect a focal lesion or obtain a large biopsy.

Such biopsies can be performed by placement of overlapping sutures, both haemostatic and pneumostatic (Figure 5a). Alternatively, several rows of staggered mattress sutures can be used for the same effect (Figure 5b). Partial lobectomies can also be performed using automatic sutures, which is quicker and technically easier. Thoracoabdominal (TA) and gastrointestinal (GIA) staplers are most appropriate for this purpose. After resection of a portion of a lung lobe, the airtightness of the sutures are tested by pouring a warm sterile isotonic solution in the thoracic cavity until the pulmonary section line is immersed. Whilst the section is immersed, the patient is mechanically ventilated and the fluid in the chest is inspected for signs of air bubbles. If such bubbles are detected, the section is inspected to localise the source of the air leakage and additional mattress sutures are placed to seal the wound. Before closure of the thoracic cavity, the accessible lymph nodes are inspected and preferably biopsied.

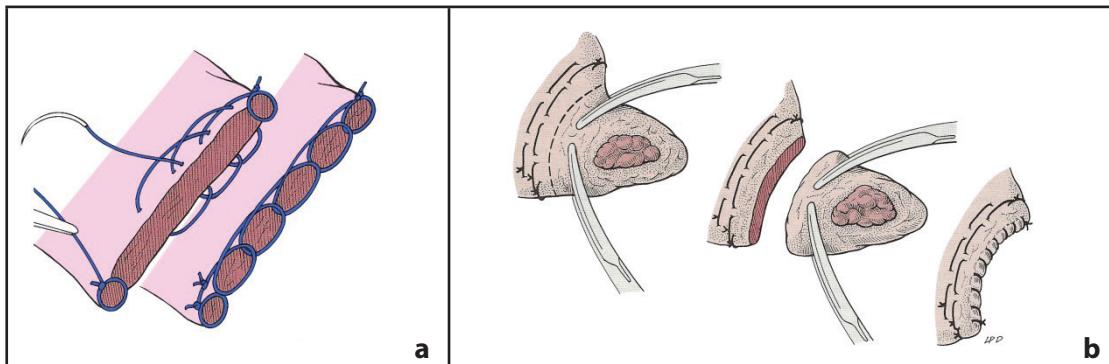


Figure 5: Partial lobectomy (a: overlapping suture; b: staggered mattress sutures)¹.

Experimentally, the use of sealing devices for partial lung lobectomies has been reported on healthy lungs⁹. This technique needs to be validated on diseased lungs before it can be used in clinical cases but may provide another means of performing partial lung lobectomies quickly and easily in the future.

Total lobectomy

Total lung lobectomies are performed by mobilisation and vascular and bronchial isolation of the lung lobe to resect.

In a first step, the lung lobe to excise is mobilised to isolate its hilus. For caudal lung lobes, it requires cutting the pulmonary ligament, which is a reflexion of the pleura connecting the mediastinum and the medial aspect of caudal lung lobes. The accessory lobe of the right lung can be more challenging to mobilise as its ventral portion is masked by the plica venaee cavae, which is a reflexion of the pleura joining the caudal vena cava, the pericardium and the diaphragm. Once the lung lobe is mobilised, its bronchial and vascular isolation must be carried out before its resection. Several techniques can be used. The hilus can be dissected and its artery, vein and bronchus are isolated and ligated separately. It is recommended to place 2 ligatures, including a transfixing one, on the portion of artery and vein remaining in the animal. Care is taken to place ligatures where it will cause no inference to the vascularisation of other lung lobes. Ligation of large bronchi may require their plication or placement of mattress sutures to achieve air-tightness (Figure 6). Alternatively, automatic sutures can be used to ligate the hilus without the need to dissect its components. Once the lung lobe is mobilised, a TA or GIA stapler is used to occlude the lobar artery, vein and bronchus.

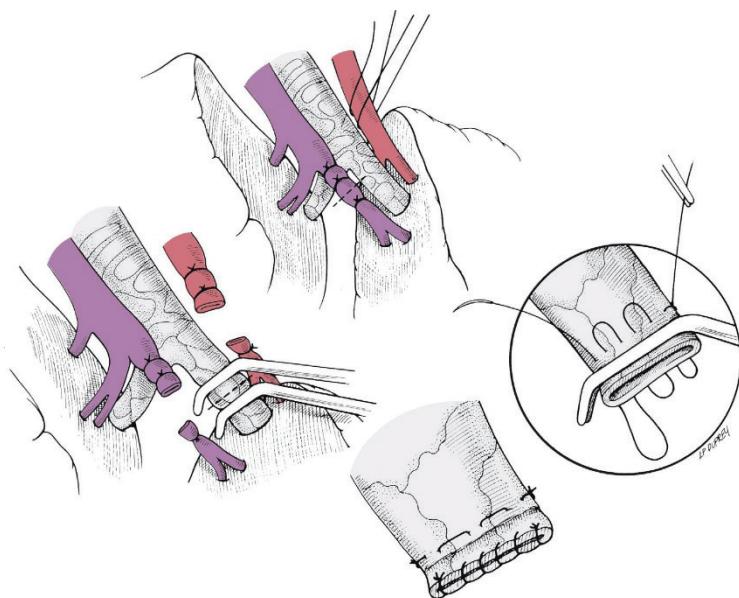


Figure 6: Total lung lobectomy¹

Similarly to what is done after partial lobectomy, the airtightness of the sutures is tested by their immersion in warm isotonic solution poured in the thoracic cavity. Again, the accessible lymph nodes are inspected and biopsied as required.

Thoracic drains

The chest is invariably closed over one or several chest tube(s). Different types of tubes can be used as chest tubes. Commercially available tubes are usually made of polyvinyl or silicone and contain a metal trocar-tipped stylet to facilitate placement. Many other types of tubes (e.g. feeding tubes) can be used as thoracostomy tubes and be placed with Carmalt-type forceps. When using non-specific tubes, additional holes need to be made, depending on the number of holes already present at the extremity of the tube. In this case, the length of the portion of the tube which will be inside the thoracic cavity must be anticipated so that no holes are located outside the thoracic cavity. Dedicated chest tubes usually are stiffer than other tubes, which eases their correct placement and make them more resistant to kinking and collapse. These stiffer tubes may however cause more discomfort than more supple ones. The widespread recommendations are to approximate the tube size to that of one of the mainstem bronchi, as seen on a lateral thoracic x-ray. This leads to using tubes ranging from 14 to 36 French, depending on the size of the patient. However, this rule should not be applied too rigidly and the tube size should also be adapted to the viscosity of the thoracic contents to be drained: smaller for air, intermediate for transudate or blood, larger for chyle or pus. Using tube which is larger than necessary will indeed induce more inflammation, which will itself be responsible for the production of thoracic effusion and discomfort. Recently, the use of small-bore chest tubes has gained interest in human medicine¹⁰⁻¹² and has been found to be associated with lower complication rates than large-bore tubes placed under pressure. It has been studied in small animals and appeared to be efficacious and associated with minimal complications, even when used to drain viscous fluids such as pus^{13,14}. Such an approach is therefore worthy of consideration or, in the least, should engage to use smaller chest tubes than classically recommended.

The chest tubes are then used for intermittent or continuous suction, depending on the type and amounts of air or fluids produced. When a tube is no longer needed, it is removed by simple traction after section of the finger-trap pattern suture. Its stoma is left to heal by second intention and need to be covered with a dressing for 24 to 48 hours. By then, it is usually sealed and airtight, and can be left uncovered. It is widely stated that the drain itself induces the production of 0.5 to 2 ml/kg/day of pleural fluid and that chest tubes should not be removed until less than 2 to 3 ml/kg/day of pleural effusion is drained. However, it has been showed that drains could be removed while the pleural effusion production was much greater than 3 ml/kg/day without incidence on the outcome, if data from clinical examination and clinical pathology supported their removal¹⁵. This shows that the amount of effusion produced is only one of many criteria to take into consideration in making the decision to remove chest drains.

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EASY TIPS AND TRICKS FOR WOUND CLOSURE

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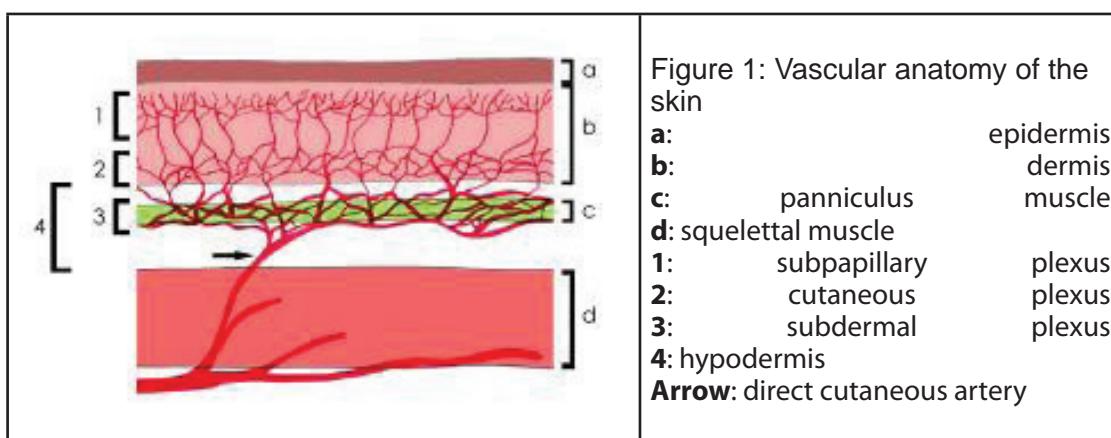
Mastering wound closure techniques is crucial for veterinary surgeons, as the complexity of these procedures can vary significantly, from simple sutures to advanced reconstructive surgery. Effective wound closure not only ensures proper healing but also minimises the risk of infection and promotes the best possible functional and cosmetic outcomes for the patient. While straightforward incisional wounds may only require basic suturing skills, more complex cases, such as those involving large tissue deficits, tension wounds, or areas with limited skin availability, demand a high level of expertise in reconstructive surgery. This expertise is essential for managing challenging cases, where improper technique can lead to complications like dehiscence, poor wound healing, or even loss of function. For a surgeon, investing in wound closure skills pays off enormously, as every single patient undergoing surgery will benefit from it. It will also optimise outcomes and minimise complications.

This is not a review of skin reconstruction in general, but rather a short list of tips and tricks which are thought to be helpful for most veterinary surgeons in practice.

- 1. Use of Towel Clamps:** Towel clamps are a versatile tool that can be used to manipulate and approximate skin during wound closure. They can help to minimize trauma to the skin and allow to try as many positions of the wound edges as needed to find the one resulting in the least wound tension. They facilitate placement of subcutaneous sutures by keeping the wound edges together in the desired position while they are placed.

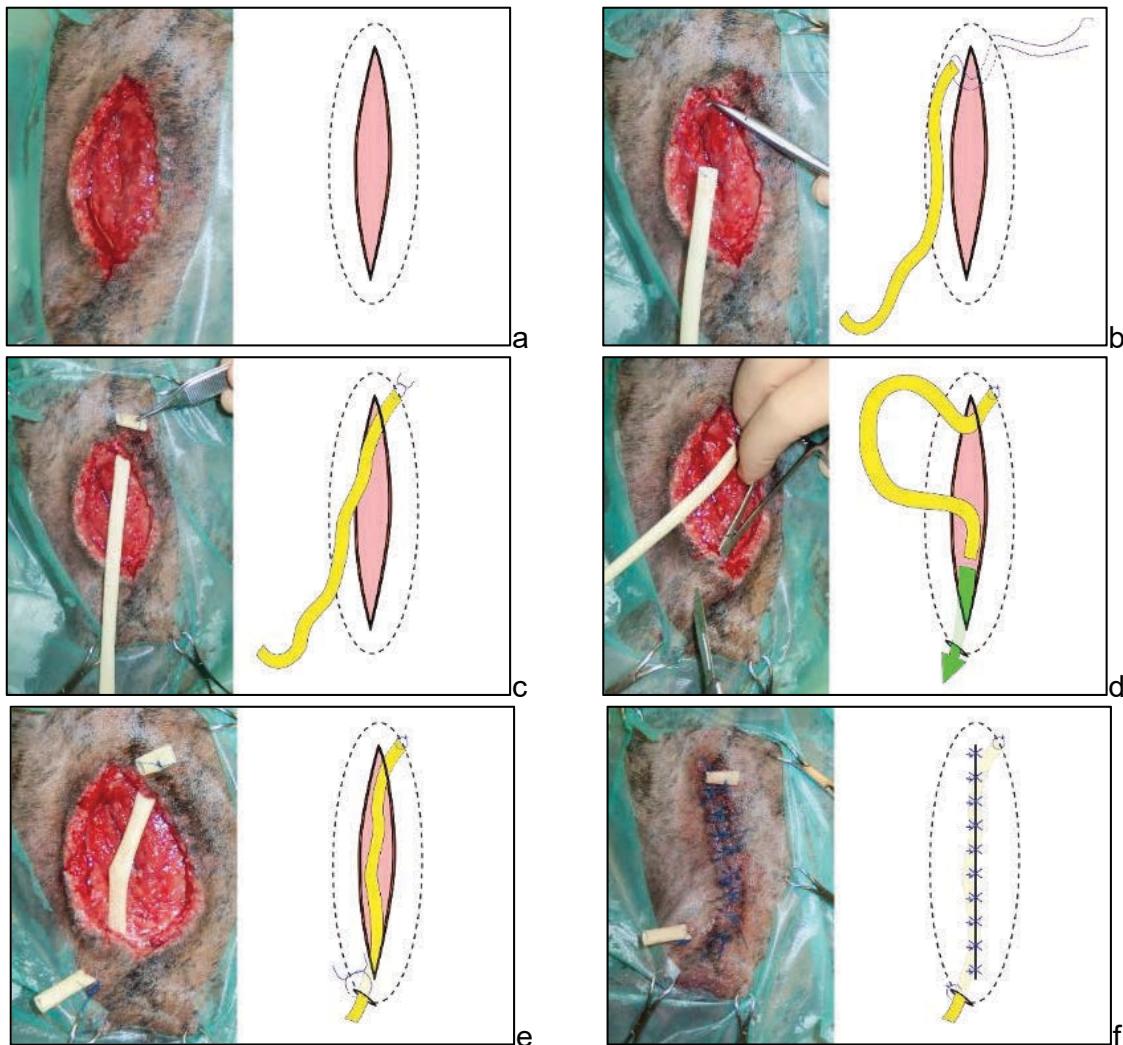
2. Undermining skin

In dogs and cats, the skin is vascularised by 3 plexi: the subpapillary, cutaneous and subdermal plexi. The two most superficial plexuses depend on the subdermal plexus, which is therefore the most important to preserve. This subdermal plexus lies in depth of the hypodermis. In regions of the body where a panniculus muscle is present (trunk, neck), the subdermal plexus runs immediately deeply and superficially to it. As a practical consequence, when the skin is undermined for primary closure or performance of a skin flap, it must be elevated in depth of the panniculus muscle. In areas where no such muscle is present, the skin must be elevated as close as possible from the underlying fascial or muscular plane.



- 3. Releasing Incisions:** In cases where primary wound closure is challenging due to tension, creating releasing incisions can help to reduce strain and promote healing. These incisions are typically left to heal by second intention.
- 4. Far-Near-Near-Far Suture Pattern:** This suture pattern is particularly useful for wounds with moderate tension. By placing sutures farther from the wound edges, the tension is distributed over a larger area, reducing the risk of tissue cutting.
- 5. Draw:** Using sterile pens to draw on the skin helps plan both resection and reconstruction phases. In complex cases, they help keep track of the original plan throughout the procedure, even at times when landmarks are harder to recognise because part of the tissues have already been resected or mobilised, and have changed position.

6. **Progressive Closure:** For wounds that are not ready for immediate primary closure, a progressive closure technique can be employed. This involves placing a loose continuous suture and gradually tightening it over time as the wound heals.
7. **Place passive drains correctly:** Drains can be used to manage dead space. Passive drains are cheap, easy to maintain and versatile, but they need to be placed correctly.

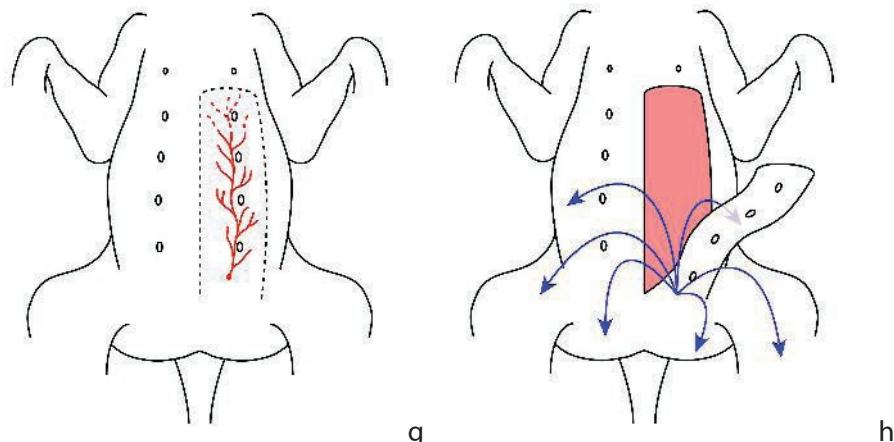


The wound is debrided and rinsed as necessary. Its subcutaneous/deep boundaries (dotted lines) are probed (a). A suture is placed through the skin opposite a point that the most non-dependant point of this cavity (b). This suture is passed through the drain before being taken out again through the skin. There is therefore no entry point for the drain in the upper region, the drain being fixed under the skin (c). The suture holding the drain can be identified by placing a piece of drain, so that it can be more easily located when the drain is removed. A drain exit wound is created in the most dependent region of the wound cavity, outside the skin wound. The free end of the drain is passed through this new orifice (d). The drain is fixed with a suture at its exit point from the skin (e). The wound is then closed in 1 or 2 layers, taking care not to incorporate the drain into any subcutaneous or cutaneous sutures (f).

Drain removal is typically performed 2 to 8 days later, depending on drain production, by cutting the suture holding the drain in the non-dependant, then the suture holding the drain at its exit point, and then pulling the drain out. This procedure does not normally require any sedation.

8. **Caudal Superficial Epigastric Flap:** This flap is based on the superficial caudal epigastric artery, which emerges out of the superficial inguinal ring, a paramedian breach in the aponeurosis of the external oblique abdominal muscle. The medial limit of the flap is the midline, and its lateral limit is parallel to the midline, both being on either side and equidistant from the line of the nipples (g). In the male dog, the medial incision must include the base of the prepuce to avoid the risk of injuring the superficial caudal epigastric artery.

The flap can safely incorporate the 3 most caudal mammary glands in the dog, and the 2 most caudal mammary glands in the cat. In both species, if necessary, it is possible to incorporate the second mammary gland, but the survival of the distal end of the flap is then more uncertain. The flap is elevated by advancing craniocaudally until the vascular pedicle is identified as it exits the superficial inguinal ring. If an insular flap is desired, the caudal incision line is then placed caudally to the insertion of the superficial caudal epigastric vessels on the flap. The flap can then be mobilised and sutured to the recipient site. Care is taken not to exert traction or excessive rotation ($>180^\circ$) on the vascular pedicle. Most often, the donor site can be closed simply as would a radical mastectomy wound. This axial patterned flap is a valuable option for reconstructing wounds in the flank, perineum, hindlimbs and inguinal area (h). It is easy to perform and can be adapted to various wound sizes and shapes by folding it on itself.



SURGERY OF PROSTATIC DISEASES

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Prostatic diseases

Prostatic diseases are common in dogs. In one study, 76% of dogs were found to have prostatic disease at necropsy¹. The most common prostatic diseases in dogs are:

Benign prostatic hyperplasia (BPH)

This is the most common prostatic disorder in intact male dogs. It affects approximately 50% of intact male dogs by 4 years of age, and nearly all intact males develop it at some point in their lives.

Prostatitis (bacterial infection of the prostate)

This is the second most common prostatic disease in dogs, with an incidence of 28-38.5%². It can be acute or chronic and is more common in intact males.

Prostatic cysts

These occur in 2.6-14% of cases of prostatic disease². They can include cystic hyperplasia secondary to BPH or paraprostatic cysts.

Prostatic tumours

This is relatively uncommon in dogs, with an incidence of only 0.2-0.35%². However, it tends to be aggressive and is more common in neutered males.

It is important to note that these conditions can often occur concurrently, particularly BPH and prostatitis. The prevalence of these diseases can vary depending on factors such as neutering status and age.

Anatomy and Surgical Considerations

The prostate gland is a bilobed organ located in the pelvic cavity, surrounding the proximal urethra caudal to the bladder neck. It is enclosed within a fibrous capsule and receives blood supply primarily from the prostatic artery, a branch of the internal pudendal artery.

When approaching prostatic surgery, the surgeon must consider the patient's overall health status, the specific prostatic pathology, and the potential impact on urinary and reproductive functions. Preoperative imaging, including ultrasonography and, in some cases, computed tomography or magnetic resonance imaging, is essential for accurate diagnosis and surgical planning. Additionally, a thorough understanding of the regional anatomy, including the course of the ureters, neurovascular bundles, and pelvic musculature, is paramount for successful surgical outcomes.

Surgical Approaches

The prostate is typically approached through a caudal median coeliotomy (e.g. umbilicopubic). It is important to open the linea alba as caudally as possible, until the pubis is reached, to maximise exposure. Stay sutures are placed on the apex of the bladder, which is retracted cranially. The fat covering the bladder is then bluntly dissected away starting from the midline and pushing the fat aside bilaterally. This exposes the ventral aspect of the prostate. The dissection should only be extended to the laterodorsal and dorsal aspects of the prostate if necessary, to limit the risks of damage to the nerves responsible for urinary continence travelling in the lateral ligaments of the bladder and within the dorsal aspect of the prostatic capsule.

Surgical Techniques for Specific Prostatic Diseases

Benign Prostatic Hyperplasia and Prostatitis

Benign prostatic hyperplasia (BPH) is a common condition in intact male dogs, particularly in older patients. When it is symptomatic, it is treated by castration, either chemical or surgical.

Prostatitis is often associated with BPH and caused by bacterial infections. They are treated by castration if BPH is present, and with systemic antibiotics. If abscesses are present and large enough, they may be drained by percutaneous aspiration³ or surgically (see below).

Prostatic Cysts and Abscesses

For small to moderate-sized cysts or abscesses, percutaneous drainage under ultrasonographic guidance may be attempted as a minimally invasive option. However, for larger or recurrent lesions, surgical drainage and debridement are often necessary.

The approach to prostatic cysts and abscesses is to resect as much of the wall of the cyst or abscess as possible, and omentalisising the remaining portions. For cysts and abscesses inside the prostate, debridement and omentalisation is the preferred approach^{4,5}.

Prostatic Neoplasia

Prostatic neoplasia in dogs is relatively uncommon but can present a significant therapeutic challenge when encountered. The most common types of prostatic tumours in dogs include adenocarcinoma, transitional cell carcinoma, and undifferentiated carcinoma.

Differentiating between carcinoma and transitional cell carcinoma (TCC) is challenging. Metastases are common (40% at the time of diagnosis and 80% at the time of death) and typically develop in lymph nodes, bones, liver or lungs. The diagnosis is made based on clinical signs, imaging, cytology and BRAF testing (mutation present in 80% of cases). Treatment modalities include surgery, chemotherapy, radiotherapy, NSAIDs, and interventional oncology techniques like intra-arterial chemotherapy⁶, prostatic arterial embolisation⁷, and urethral stenting. Prostatic arterial embolization has shown success in reducing prostatic volume and alleviating symptoms. Urethral stenting is a palliative option but may cause severe urinary incontinence in about 20% of cases.

Partial and total prostatectomy can be carried out for treatment of prostatic tumours. Unfortunately, the nature and the extent of the tumour often requires total prostatectomy. During this procedure, care is taken to minimise the trauma to nerves coursing along the prostate and bladder neck. In a study, permanent postoperative incontinence was observed in 8 of 23 dogs (35%)⁸.

Complications

Potential complications following prostatic surgery include urinary incontinence, urethral stricture, urine leakage, and recurrence of the primary condition. Urinary incontinence is a particularly concerning complication and may result from iatrogenic injury to the urethral sphincter mechanism or alterations in bladder neck function. Management of postoperative urinary incontinence may involve medical therapy, such as alpha-adrenergic agonists or anticholinergic medications, or additional surgical intervention in severe cases. Urethral stricture is another potential complication, particularly following total prostatectomy or extensive urethral manipulation. Regular monitoring of urinary function and early intervention with urethral dilation or stenting may be necessary to manage this complication. Prompt recognition and surgical repair are essential to prevent urine peritonitis and associated morbidity.

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**Jackie Demetriou (United Kingdom)**

BVetMed, CertSAS, DipECVS, MRCVS, European and RCVS-Recognised Specialist in Small Animal Surgery

EERVC 2024 Lectures

1. Nose surgery
2. Surgery of biliary mucoceles
3. Surgery for salivary gland disease
4. Ear surgery – dogs
5. Ear surgery – cats

Honorary Associate Professor, University of Nottingham. Jackie qualified from the Royal Veterinary College, London, in 1996 and spent one year in mixed practice before joining Edinburgh Veterinary School as an Intern. She completed a Residency in Small Animal Surgery in 2001 and, after a year in a private referral practice, was appointed Lecturer in Small Animal Surgery at the University of Cambridge. She joined DWR in 2012. Jackie passed her RCVS Certificate in Small Animal Surgery in 2000 and the ECVS Diploma in 2002. She has served on the European College of Veterinary Surgery examining board for 5 years. She enjoys all aspects of soft tissue surgery, from clinical work and teaching to participating in clinical research. She publishes widely in peer reviewed journals, has published a textbook on Small Animal Oncology with Rob Foale and is co-editor and contributor to the only dedicated book on feline soft tissue surgery. She contributes regularly to national and international conferences and surgery courses. She has undertaken advanced training in Interventional Radiology (IR) and laparoscopy.

Biliary Mucoceles : When is this a surgical disease?

Jackie Demetriou Dip ECVS, FRCVS

1

What are Gall Bladder Mucoceles?

Abnormal accumulation of semi-solid bile and mucous within the gall bladder lumen

Results in macroscopic distention of this organ with green - black gelatinous material causing various degrees of extrahepatic biliary obstruction

When obstruction occurs gallbladder distension often leads to necrosis and eventual rupture

2

Questions that need answers

- Those that may not need surgery
- Timing of surgery
- Those that have ruptured
- Co-morbidities

3

Do all GBM need surgery?

- What do we know?
- Dogs with signs relating to GBM or signs of obstruction are surgical candidates
- Surgery as an elective procedure results in better outcomes
- Cases with rupture are urgent surgical candidates



4

Do all GBM need surgery?

- What do we know?
- Significance of biliary sludge
 - Incidental finding?
 - 35-67% healthy dogs
- Humans – NOT considered incidental
 - Cholecystitis, biliary colic, pancreatitis
- Should this change our view?



5

Clinical characteristics and histology of cholecystectomised dogs with nongravity-dependent biliary sludge: 16 cases (2014–2019)

DOI: 10.1111/jvim.15607

80% had pathology in GB, liver, GIT

Resolution of clinical symptoms in 86%

13% developed GBM

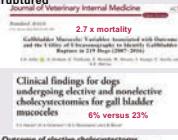
B. Terrier and Shetland Sheepdog

50% of GBM developed from NDBS

6

Identification of those that have ruptured

- Relationship with morbidity / mortality
- Prevalence with GBM 21-61%
- Non elective cases have worse prognosis
- Toxic effects of bile
- ...SIRS...sepsis...Multiple Organ Failure



7

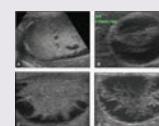
How to identify ruptures perioperatively?

- Ultrasound
- Ultrasound guided abdominocentesis
- CT

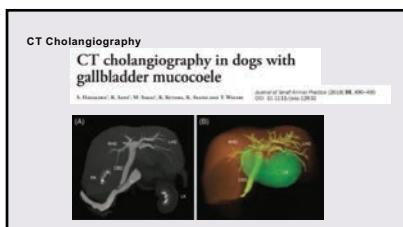
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How to identify ruptures perioperatively?

- Conventional ultrasound
- Sensitivity of 56% and specificity of 92% for cases with rupture
- So failing to identify nearly half of those that rupture



9



10

Perioperative morbidity – co-morbidities

- Association with endocrinopathies has been established

Presence of HAC associated with 2 x increase in mortality (Jaffey et al., 2019)

- Poor wound healing
- Immune dysregulation
- Hypercoagulability

Stabilising Cushings and normalising cortisol levels could reduce morbidity

11

Perioperative morbidity – co-morbidities

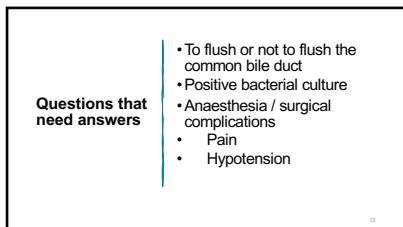
83% are hypercoagulable and some are hypocoagulable (Pavlik et al., 2020)

- HAC, liver disease, SIRS, pancreatitis etc
- Surgery may potentiate this
- 8% died due to thrombotic event

Coagulation profiles / TEG analysis would be relevant

- May dictate periop treatments
- Would lap cholecystectomy minimise further?

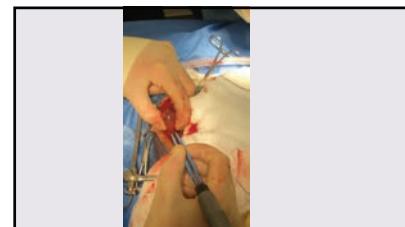
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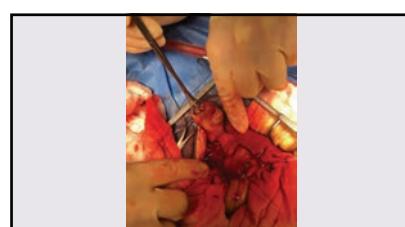
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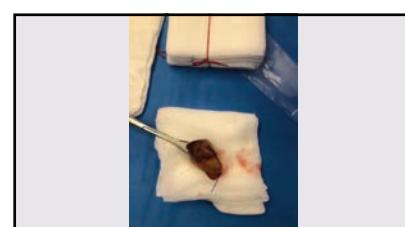
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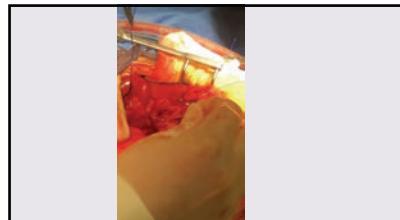
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18



19



20

The effect of flushing of the common bile duct on hepatobiliary markers and short-term outcomes in dogs undergoing cholecystectomy for the management of gall bladder mucocele: A randomized controlled prospective study

Tom L. Hermon BVSc CertAVPGSAS DipECVS MRCVS |
Ed J. Friend BVMSMed CertSAS DipECVS FRCVS |
Gordon D. McLean BVMS CertSAS DipECVIM-CA MRCVS FRCVW |
Vicki Black MA VetMB DipECVIM-CA MRCVS FHEA |
Lee R. Meakin MA MRCVS FRD VetMB DipECVS MRCVS |

21

32 Dogs
Flush or not flush group
Analysis by clinical signs, blood markers (ALP, ALP, GGT, bilirubin, cholesterol and triglycerides)

RESULTS:
 Mortal reduction following surgery in both groups
 No difference between "flush and not flush"
 Survival 90%

22

Positive bacterial culture

- Incidence and Impact ?
- Positive cultures in 10-22%
 - 43% having > 1 isolate
- > 20% mortality in people and use of ABX lowers morbidity

Factors affecting survival in 516 dogs that underwent cholecystectomy for the treatment of gallbladder mucocele
 Monty Galley, Jennifer Lang, Mark Mitchell, Jon Fletcher
 3 x increase mortality within 14 days

23

Hypotension and Pain

- Complex but significant cause of morbidity in the peri, intra and post op period
- Evidence to link hypotension to significantly reduced morbidity
- Why does this occur in mucocele patients?

24

How does hypotension affect outcomes?

Long-term survival and risk factors associated with biliary surgery in dogs: 34 cases (1994-2004)

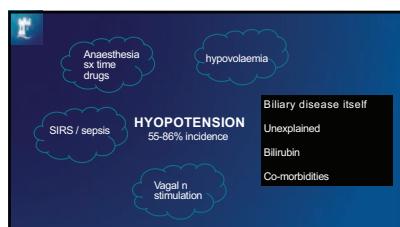
Variables Associated with Outcome in Dogs Undergoing Exploratory Biliary Surgery: 40 Cases (1998-2005)

Ultrasoundographic patterns, clinical findings, and prognostic variables in dogs from Asia with gallbladder mucoceles

13 x death in dogs with hypotension post op

Negative association with the cases that had lowest blood pressure

Association between intraoperative lowest blood pressure point and outcomes



26

So how can we influence this?

- Control the controllables!

Stabilise patients with SIRS / sepsis
Intraoperative handling – minimise additional procedures
Operative time
Fluid balance
Anaesthetic "sparing" (drugs, dose etc)

27

25

Introduction of extradural anaesthesia / analgesia and catheter placements

- Why?
- We were seeing a relatively high number of cases with intraoperative hypotension
- Unexplainable, difficult to
 - Predict
 - Prevent
 - Treat

28

Other benefits

- Pain management
- Reduction in postoperative opioids and other analgesic agents
- Effects on GI motility
- Effects on hypotension
- Effects on postoperative nutrition

29



30

What is the evidence?

Extradural anaesthesia-analgesia in dogs undergoing cholecystectomy: A single centre retrospective study
Felicity S. McIlroy and Garry Newbold

Frontiers in Veterinary Science | www.frontiersin.org
Volume 10 | Article 1000000 | March 2023 | DOI: 10.3389/fvets.2023.1000000

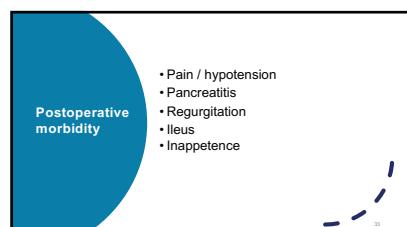
1. reduction in number of dogs requiring intraop interventions for pain control
2. post op use of a catheter would reduce need for opioid administration and additional analgesia during first 48 hours
3. use of an extradural catheter would increase likelihood and amount of voluntary food intake

31

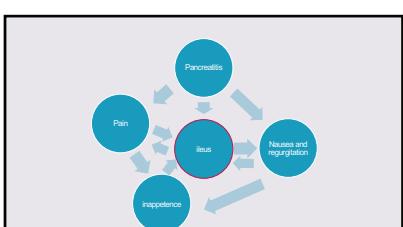
Any negatives?

- In this case series:
 - One catheter dislodgement
 - Two cases of pelvic limb weakness
- Reported
 - Dislodgement (16%)
 - Inflammation (2.4%)
 - Contamination (2.4%)
 - Abnormal urination patterns (39%)

32



33

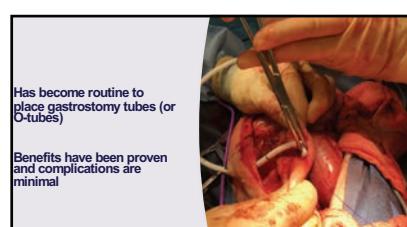


34

Nutrition cannot be an after thought

1. Stimulate peristalsis
2. Feed enterocytes
3. Reverse hypoproteinæmia
4. Cause release of cholecystokinin

35



36

In Summary

Biliary mucocles should be considered a surgical disease

Timing of surgery is important – catch these patients when they have EARLY clinical signs and before rupture

Outcomes are better in elective cases

Flushing is rarely required in these patients, possibly reducing morbidity

Consider anaesthesia methods to reduce hypotension

Nutrition is a vital addition and medical management of pancreatitis

Salivary Gland Surgery – Unleashing the Secrets!

Jackie Demetriou Dip ECVS, FRCVC

Jackie Demetriou 2023

1

Salivary gland disease

- Non surgical**
 - Sialadenosis
 - Non infectious sialadenitis
 - Infectious sialometaplasia
- Surgical**
 - Neoplastic
 - Non-neoplastic - idiopathic

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2

Salivary gland anatomy

- There are 4 major paired salivary glands in the dog and cat
 - Parotid
 - Mandibular
 - Sublingual
 - Zygomatic

Tobias and Johnson, 2nd Edition, 2016, Elsevier

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3

The parotid gland

- Triangular (U shaped) superficial to external ear canal
- Bordered by masseter (rostral), sternomastoid (caudal) and mandibular gland (ventral)
- Covered by fascia and platysma through which travels
 - Facial n. maxillary and temporal a. & v.
- Intimate association and non discrete nature of gland makes dissection challenging

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4

The Mandibular and sublingual glands

- Intimately associated so considered together
- Large and palpable
- Drained by lingual and mandibular vein junction (lateral), mandibular LN and larynx (ventral), MRPLN (medial)
- The sublingual gland is almost a continuation of the mandibular gland cranially, sharing the same capsule
 - Monostomotic and polystomotic portions

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5

Zygomatic gland

- Periorbital area ventral and rostral to the globe and medial to zygomatic arch
- Most tricky to locate due to location medial to zygomatic arch

Ramus of mandible
Zygomatic salivary gland
Transected mandible for medial exposure

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6

Salivary gland ducts

Parotid	• Ventro-rostral gland, over masseter, opening UAM
Zygomatic	• Major duct - caudo-lateral aspect of last LM
Mandibular and sublingual	• Medial to gland, continues rostrally, medial to SG exiting lateral to lingual frenulum

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7

Non-surgical salivary disease

- Sialadenosis**
 - Bilateral enlargement of (mostly) mandibular glands
 - Clinical signs include
 - Retching, hypersalivation, weight loss, gulping, lip smacking
 - Non painful generally
 - Does not respond to antibiotics or steroids
 - Treatment with phenobarbital may improve signs

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8

Non-surgical salivary disease

- Noninfectious sialadenitis and necrotizing sialometaplasia**
 - Progressive disease (inflammation to necrosis)
 - Similar signs to sialadenosis but usually painful
 - Dx of exclusion and histopath to eliminate neoplasia
 - Terrier breeds
 - Link with oesophageal disease and sometimes tx of this resolves salivary signs
 - Phenobarbital also may improve but px is guarded to poor

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9

Surgical diseases of the salivary glands

- Neoplasia
 - Rare location
 - Most are epithelial (carcinomas)
 - 300 cases of 1795 dogs had regional LN involvement
 - Staging indicated (local LN, thoracic imaging)
 - Treatment is targeted to surgical cytoreduction if rx or ct
 - One study MST cells and dogs 516 and 550 days (correlated with stage)

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Latest data

Outcomes and clinical features associated with surgically excised canine salivary gland carcinoma: A multi-institutional, retrospective, Veterinary Society of Surgical Oncology study

Kaleigh M. Bush DVM, Janice A. Grimes DVM, MS, DACVS-SA; Daniel S. Linden DVM, MS, DACVS-SA, ACVS Fellow, Surgical Oncology ... See all authors ✓
First published: 16 January 2023 | <https://doi.org/10.1111/jvso.13928>

MST was 1866
Local recurrence in 42% dogs with DF1 of 191 d
Metastasis in 325 with DF1 of 299
LN involvement DF1 of 98 d and MST 248

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Sialocele

- Also termed "salivary mucocoeles"
- Accumulation of saliva within s.c. tissues (lined by inflammatory tissue)
- Can affect any gland but predominantly the mandibular and St. Louis
- Causes often idiopathic but other causes include:
 - Trauma
 - Sialoliths
 - Neoplasia
 - IBD
- Poodles, Australian Silky Terriers, GSD, Dachs.

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Zygomatic sialoceles

- Presentation
 - Exophthalmos
 - strabismus
 - 3rd eyelid protrusion
 - Painless orbital swelling
- 1 case report of cervical presentation of a zygomatic sialocele

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Zygomatic sialocele - diagnosis

- FNA of swelling if visible
 - Stringy saliva
 - Can do cytology
 - Mucus and vacuolated macrophages

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Zygomatic sialocele - diagnosis

Advanced imaging

Computed tomographic appearance of sialoceles in 12 dogs. *Vet Radiol Ultrasound* 2022 Jan;63(1):20-31 doi: 10.1111/vru.13253. Epub 2021 Sep 28. PMID: 34561607

- Fluid attenuating
Non contrast enhancing contents
- Smooth external walls
- Irregular internal walls with protrusions
- Minimally foci in 7/13 (sialoliths or osseous metaplasia)

(a) (b)

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Zygomatic sialocele - surgery

- Traditionally this involves an osteotomy of the zygomatic arch to access the gland ventrally

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Veterinary Surgery. 2021;50:564-570 Wiley

16

Zygomatic sialocele - surgery

ORIGINAL ARTICLE • CLINICAL

Comparison of three surgical approaches for zygomatic sialoadection in canine cadavers

Judith Dörner DVM | Silvia Oberbacher DVM | Gilles Dupré DVM, DECVS

Traditional osteotomy / ecotomy of zygomatic arch | Dorsal approach to zygomatic arch | Ventral approach to zygomatic arch

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Zygomatic sialocele - surgery

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Veterinary Surgery. 2021;50:564-570 Wiley

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Zygomatic sialocele - surgery

Results: The osteotomy-based approach offered excellent surgical view and good exposure of the zygomatic gland but caused more tissue trauma. The dorsal nonosteotomy approach did not allow complete zygomatic gland extraction in nine of the 10 dogs, whereas the ventral nonosteotomy approach enabled complete extraction in all 10 dogs.

Conclusion: The ventral zygomatic approach allowed complete removal of the zygomatic gland, with good surgical overview, while reducing tissue trauma and preserving the zygomatic arch.

Clinical significance: The ventral nonosteotomy approach should be considered as an alternative to excise the zygomatic gland in dogs.

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Zygomatic sialocele - surgery

Intraoperative approach for zygomatic sialoadenectomy in dogs: An anatomical study and three clinical cases

Joni Viljanen ¹, Hilde de Rooster ², Adrienne Kishchuk ³, Boaz Arzi ³, Naukias Demetriou ³

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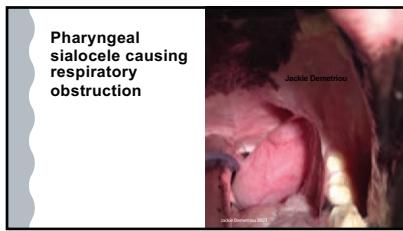
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Parotid and pharangeal sialoceles

Parotid <ul style="list-style-type: none"> Rarely reported as idiopathic cases 1 case report following parotid duct transposition in a dog 	Pharangeal <ul style="list-style-type: none"> Presentation often associated with dyspnoea Swelling visible intraorally Treatment is by incision into swelling to improve breathing and sialadenectomy of mandibular and SL duct
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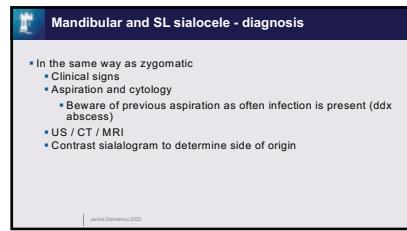
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Mandibular and SL sialocele - surgery

- Whether just a cervical swelling or just a ranula or both, treatment is the same
- Exirpation of the affected mandibular and sublingual salivary gland
- Incision and suction of the sialocele
- Both lateral and ventral approaches described
- Ventral approach will be described

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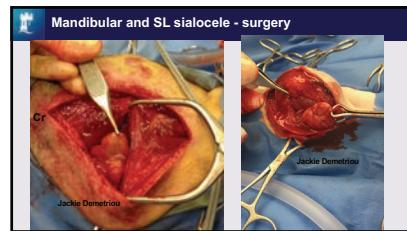
Mandibular and SL sialocele - surgery

- Often easier to see which side originates from once in dorsal recumbency
- Curvilinear incision from gland following medial aspect of mandible
- Dissection onto gland and through platysma muscle

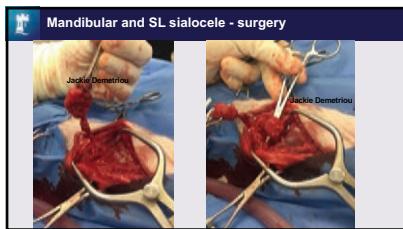
Jackie Demetriou 2023

Tabata and Johnston, 2nd Edition, 2018, Elsevier

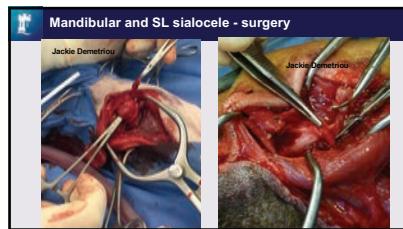
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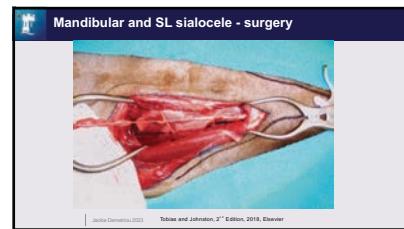
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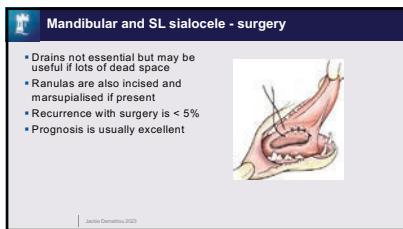
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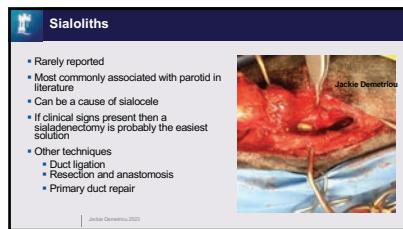
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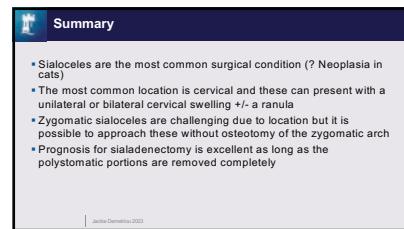
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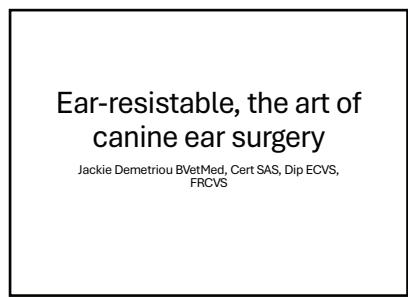
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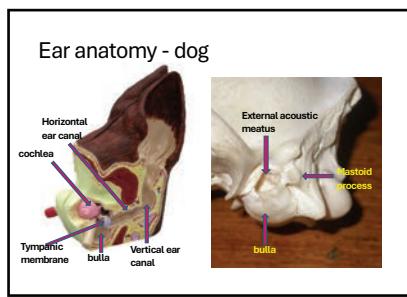
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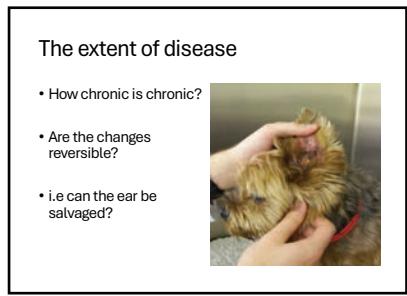
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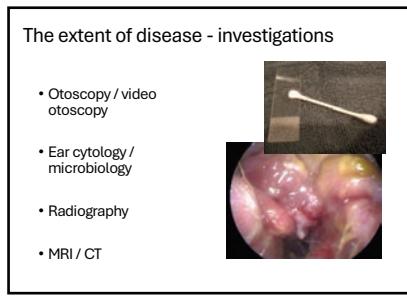
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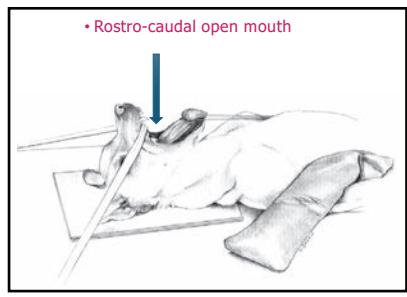
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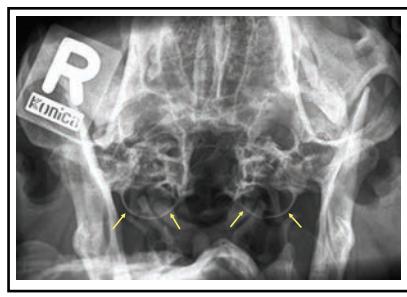
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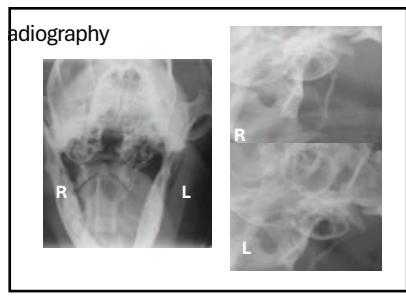
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MRI / CT

- Sensitive in evaluating the extent of infection
- Involvement of middle ear / inner ear
 - contrast
- Diagnosis of masses

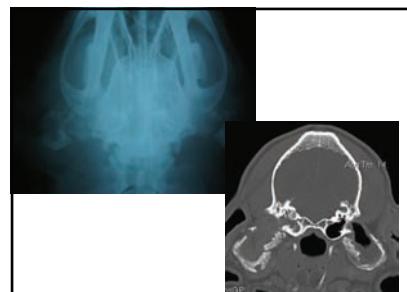
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Reversible or irreversible changes?

<ul style="list-style-type: none"> • Reversible • Ears that can be managed medically with a little bit of surgical help • Inflammation and infection have potential to resolve 	<ul style="list-style-type: none"> • Irreversible • Hyperplastic epithelium cannot resolve • Chronic fibrosis and calcification of ear canals • Stenosis not resolvable
---	---

14

The type of disease

<ul style="list-style-type: none"> • Inflammation versus neoplasia • Neoplasia <ul style="list-style-type: none"> • Benign adenomas / polyps • Malignant tumours 	<ul style="list-style-type: none"> • For malignancies only a total ear canal ablation will be adequate • Following staging (lymph nodes, thoracic radiographs) and biopsy
---	---

15

Surgery for chronic otitis

<ul style="list-style-type: none"> • Lateral wall resection • Indicated for ears that can be salvaged • for reversible changes 	<ul style="list-style-type: none"> • Total ear canal ablation • Indicated for ears that cannot be salvaged • for irreversible changes • OR owners cannot manage chronic otitis
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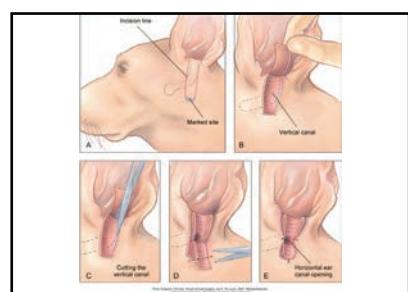
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Lateral wall resection

Aims

- Improve the local aural environment
 - Ventilation
- Improve access for medical treatment

17



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Lateral wall resection

- Good things:
- Ear is salvaged
- Improves efficacy of medical management
- Improved ventilation and drainage can resolve problems in some cases

- Bad things:
- Not a substitute for medical management
- Often case selection is poor
- Often ears are too chronic to obtain benefit

21

Complications

- Recurrence of infections
- Stenosis at the site
- Self – trauma / infection



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Total ear canal ablation

- For irreversible otitis
- For malignancies
- For chronic otitis with poor owner management
- For failed lateral wall resections



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Total ear canal ablation

Aims:

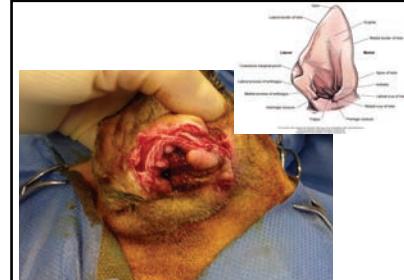
- To remove all infected tissue from the external and middle ear
- Most surgeons will perform concurrent lateral bulla osteotomy for better access to middle ear



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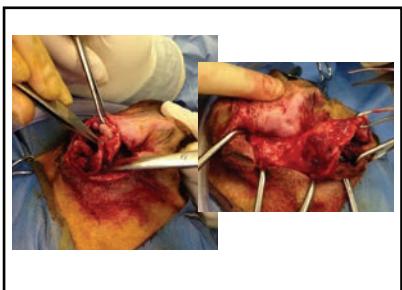
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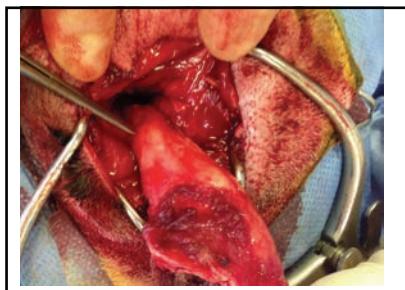
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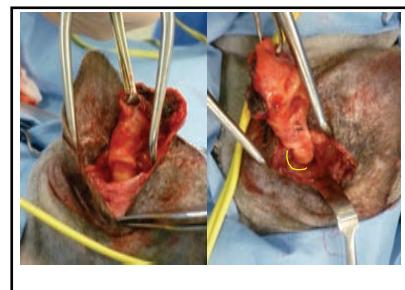
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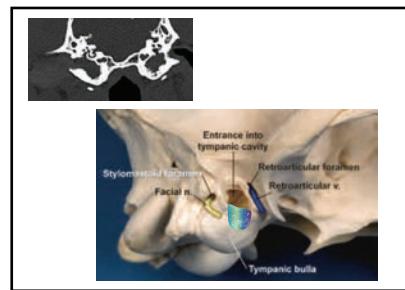


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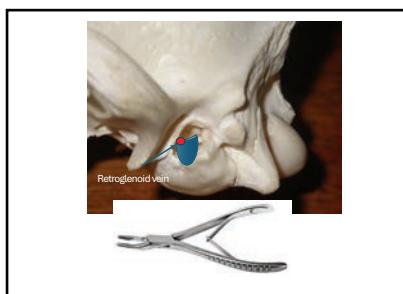
The purpose of the bulla osteotomy

- To enable visualization of the bulla
- To enable passage of instruments into the bulla
- Better access to bulla but may have increased risk for retrogenoid vein
- To do or not to do?
- Not necessary in all cases – BUT debridement and cleaning still necessary
- Brachycephalic breeds likely required

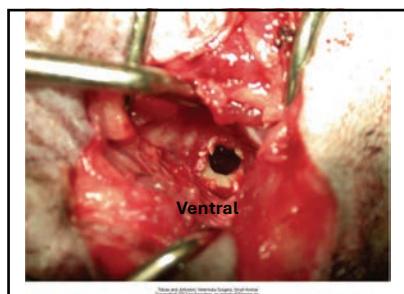
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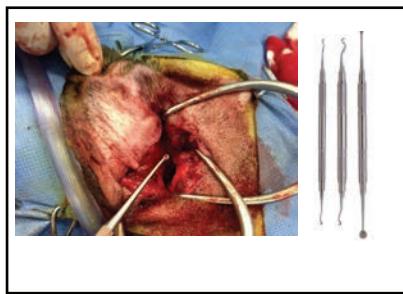
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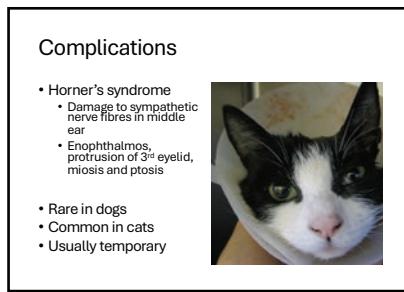
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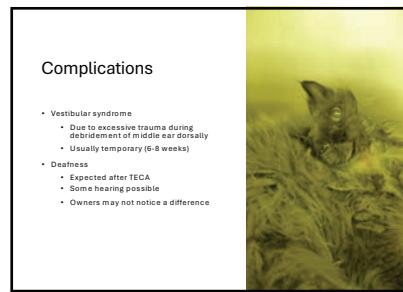
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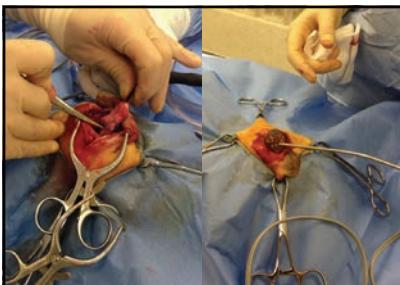
Complications

- Para-aural abscess
- Inadequate debridement
- Failure to remove infected tissue
- Usually from middle ear
- Presentation can be weeks to years
- Swelling and pain
- Revision surgery is necessary

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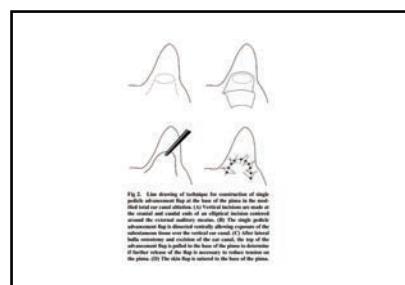
Advancement flap described in cats

> Vet Surg. 2004 Sep-Oct;33(5):435-6. doi: 10.1111/j.1532-950X.2004.04065.x.

Cosmetic results of a ventrally based advancement flap for closure of total ear canal ablations in 6 cats: 2002–2003

Amanda Hills McNabb ¹, James A. Hendren

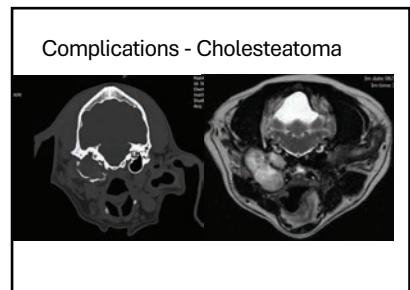
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Cholesteatoma

- Up to 50% recurrence rates
- some may be small masses but vast majority are massively expansile lesions
 - Distorted ear cavities
 - Poor quality bone
 - Severe bone proliferation
 - Removal of epithelium hard
 - Invaginations
 - Tight adherence
 - Therefore incomplete removal is difficult

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Summary

- TECA is the most common ear surgery for dogs with otitis externa and media, and likely the most successful
- Success relies on complete removal and debridement of external ear canal and bulla
- Bulla osteotomy is not required for most cases as long as you clean the bulla effectively
- On going atopy (allergy) management is required,
- Cholesteatomas are invasive and often a cause or effect of ear disease in dogs

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Purr-fecting ear surgery in the cat

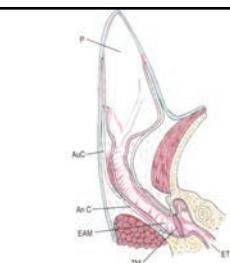
Jackie Demetriou BVetMed Cert SAS, Dip ECVS,
FRCVS

1

Summary

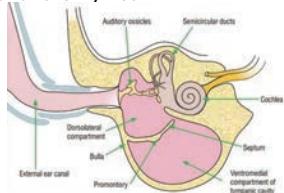
- Review of anatomy
- Diagnosis of ear conditions
- Surgery of the pinna
- Surgery of the external ear canal
- Surgery of the middle ear canal

2



3

Ear anatomy - cat



4

The external pinna

- Trauma
- Aural haematoma
- Pinna neoplasia



5

Pinna trauma

- Common after cat fights
- Can often leave to heal by secondary intention with wound management
- Better cosmetic effect with debridement and suturing



6

Aural haematoma

- Accumulation of blood between skin and cartilage
- Much rarer in cats than dogs
- In dogs related to chronic ear disease
- Cats often related to ear mites or trauma



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Aural Hematoma Secondary to Hemangiosarcoma in a Domestic Cat - Case Report

If suspicious consider biopsy as these will require a complete pinnectomy.
Prognosis seems to be better in cats than dogs (3 year survival)

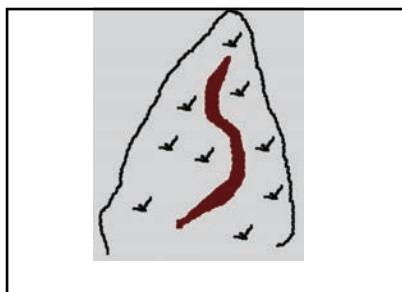
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Aural haematoma

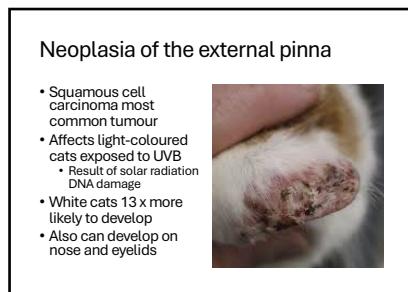
- Treat underlying cause of head shaking
- Needle drain initially, may need repeating
- Surgical drain sometimes required in these cases



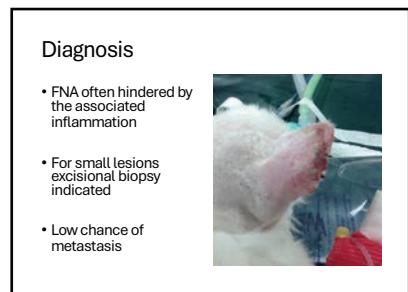
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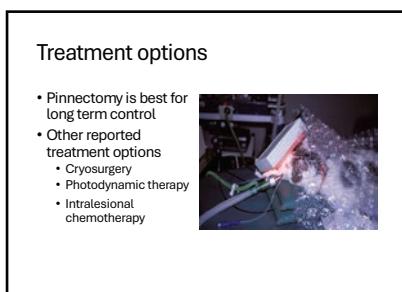
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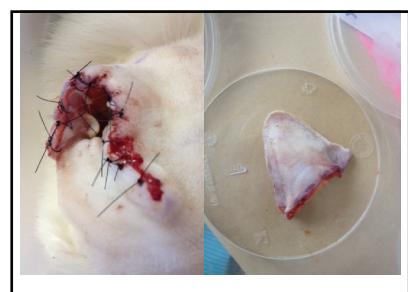
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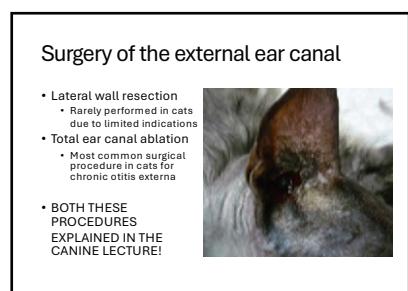
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Clinical examination findings

- Cats are more prone to developing Horner's syndrome than dogs
 - Sympathetic n
 - second or third order
 - Synapse is near tympanic bulla

19

Horner's syndrome

- Miosis
- Ptosis
- Enophthalmos
- Protrusion of nictitans glans (third eyelid)

20

Clinical signs

- Cats are more likely to develop peripheral vestibular signs compared with dogs with extension into middle and inner ear
- Seen in 25% of cats with external ear canal tumours

21

Surgery on the middle ear

- Most common ear surgery in cats
- Due to middle ear or nasopharyngeal polyps
- Benign inflammatory condition in cats

22

Pathophysiology of polyps

- Unknown
- May be result of congenital defect
- May be result of inflammation, infection of middle ear
- No virus has been consistently found
- Often a previous history of upper respiratory infection

23

polyps

- Often young adults
- Inspiratory dyspnoea
- Stertor
- Gagging
- Neurological signs
 - Vestibular
 - Horners
- Most common mass found in external ear canal in cats

24

Diagnosis

- Clinical otoscopy
- If extending into pharynx, elevate soft palate and can often visualise
- Treatment options
 - Traction
 - Surgical removal

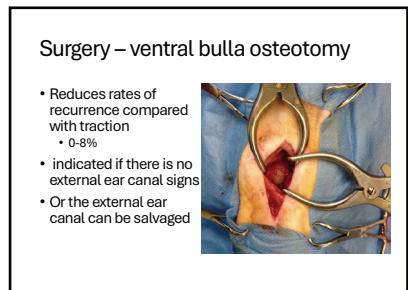
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Polyp traction

- Grasp and pull!
- Higher recurrence rate with those pulled from the ear (50%) compared with those pulled from the nasopharynx (11%)
- Overall 33-55% recurrence with traction

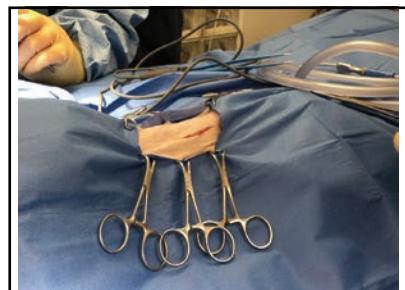
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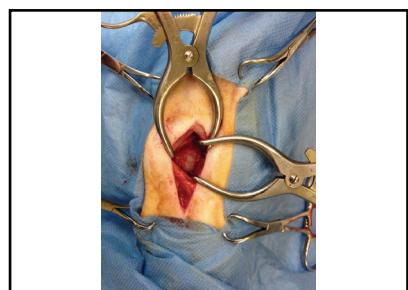
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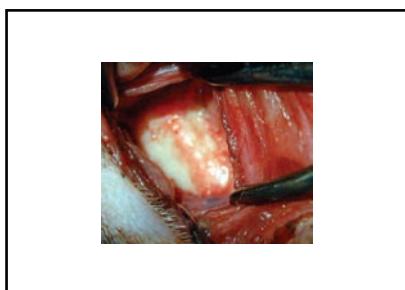
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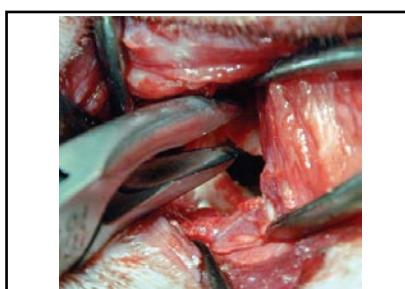
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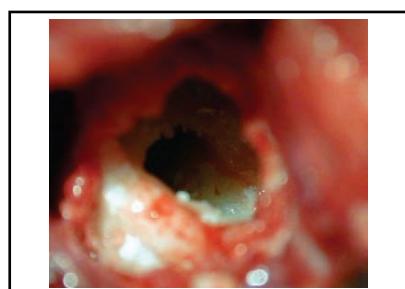
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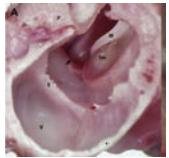
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Important surgical steps

- Breakdown septum between dorsolateral and ventromedial
- Care to avoid damage to sympathetic nerves on the dorsomedial wall
- Care to avoid curettage of the dorsal parts to prevent damage to the round window (inner ear)



41

Important surgical steps

- Submit polyp for histopathology
- Flush area
- Take culture swab from middle ear
- Close
 - Drain may be placed



42

Complications

- Horner's almost always a complication
 - Often resolves within 2-4 weeks but likely to be permanent if present before surgery
- Facial nerve paralysis - uncommon
- Sometimes worsening of head tilt
- Swelling and upper airway obstruction
 - Particularly if bilateral surgery



43

Should we do bilateral VBO at the same time?

Severe upper airway obstruction following bilateral ventral bulla osteotomy in a cat

Chiara De Gennaro ¹, Enzo Vettorato ¹, Federico Corletto ¹

Affiliations + expand

PMID: 25203943 PMID: PMC5680743

Abstract in English, French
A cat that underwent bilateral ventral bulla osteotomy (VBO) for treatment of otitis media and otitis interna secondary to bilateral inflammatory polyps, developed upper airway obstruction (UAO) soon after tracheal extubation. The cat was re-intubated but the UAO did not resolve at the next extubation. Eventually, tracheostomy was performed. Upper airway obstruction is a potential postoperative complication of bilateral VBO in cats.

44

Further research

Comparison of complications and outcome following unilateral, staged bilateral, and single-stage bilateral ventral bulla osteotomy in cats

282 cats had VBO either unilateral, staged bilateral and single-stage bilateral

Respiratory complications in 9% (unilateral), 29% (staged bilateral) and 47% (single stage bilateral)

Conclusion: staged bilateral is recommended over single stage bilateral

45

The Nose

Belgrade 2024
Jackie Demetriou BvetMed, Cert SAS, Dip ECVS, FRCVS

1

Conditions of the nose

- Tumours
 - External nares
- Foreign bodies
- Fungal infections



2

The external nares

- Tumours
 - Squamous cell carcinoma
 - CATS and dogs
- Unpigmented areas
 - + pinna, lips, eyelids
 - Ultraviolet light
 - Mediterranean regions



3

Clinical signs

- Progressive condition
- Premalignant forms
 - Carcinoma in situ
- Superficial lesions
 - Can confuse with trauma



4

Clinical signs

- Deep lesions
 - Crusting and erythema
 - Superficial erosions
 - Ulcers
 - Deeply invasive and erosive lesions
- Can confuse all forms with eosinophilic granulomas



5

Diagnosis

- Visual exam, rhinoscopy, MRI/CT
- Biopsy!
 - Incisional
 - Deep enough to avoid surface inflammation
- Assess and aspirate regional lymph nodes
 - Submandibular
- Radiograph thorax
 - Distant metastasis rare (especially in the cat)
 - MRI/CT to determine extent

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Treatment

- Superficial minimally invasive SCC
- Deep infiltrating disease



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Superficial minimally invasive

- Surgery
- Radiotherapy
- Photodynamic therapy
- Intraleisonal carboplatin

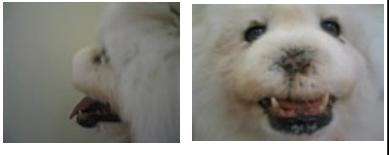
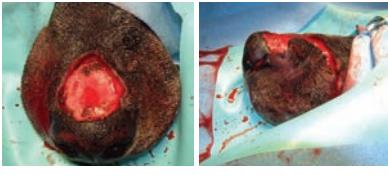
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Photodynamic therapy

- Light activation of photosensitiser
- Photosensitiser accumulates preferentially in neoplastic tissue
- Destruction of tumour tissue
- Spares normal tissue
- IV or topical application
- Excellent cosmetic



9

<p>Deeply invasive SCC</p> <ul style="list-style-type: none"> Surgery only real option Nosectomy <ul style="list-style-type: none"> 360° skin incision Transection of underlying turbinates Purse string suture to pull skin around airway Combined removal of premaxilla and nasal planum reported in dogs Stenosis a complication 	<p>Nosectomy - cat</p> 	<p>Nosectomy - cat</p> 
10	11	12
<p>Nosectomy/premaxillectomy</p> 	<p>Prognosis</p> <ul style="list-style-type: none"> Good for early noninvasive <ul style="list-style-type: none"> Later development in other areas common Wide excision for cats 80% free of disease at 1 year Median survival times of >22months Good results with photodynamic therapy for small superficial lesions 	<p>Rotation flaps</p> 
13	14	15
 <p>Rotation flaps</p>	<p>Rotation flaps</p> 	<p>Nasal foreign bodies</p> <div style="border: 1px solid blue; padding: 5px; margin-bottom: 5px;"> Most small foreign bodies are filtered and expelled by sneezing </div> <div style="border: 1px solid blue; padding: 5px; margin-bottom: 5px;"> Occasionally embed in the mucosa and cause severe inflammation </div> 
16	17	18

History and clinical signs

- Normally acute onset sneezing
- Followed by nasal discharge
- Epistaxis
- Purulent discharge



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Diagnosis

- Radiographs
 - Unless opaque will not usually see
- MRI/CT
 - Chronic active rhinitis
 - May see FB
- Rhinoscopy



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Removal

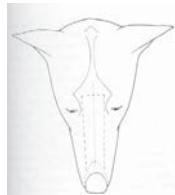
- Propulsive flushing
- Rhinoscopy
 - Old method
 - Using camera and forceps
 - Least invasive
 - Avoids major surgery
- Surgery- last option



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Dorsal approach

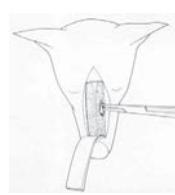
- Traditional approach
 - For pathology cranial to ethmoid turbinates including turbinates and sinuses
 - Midline incision
 - Ventral recumbancy
 - Skin, subcutaneous tissue and periosteum incised and reflected
 - Bone flap elevated



22

23

24



25



26



27

Ventral rhinotomy

- For ventral nasal passages and frontal sinuses
- More cosmetic
- Midline incision through hard palate mucoperiosteum and incision into bone
- Bone not saved
- Can extend caudally into soft palate

28



29

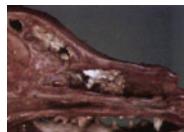
Postoperative care

- Morbidity less with ventral approach
- Clear oropharynx and nasopharynx of all fluid and debris
- Keep head lowered
- Keep external nares clean
- Feed soft food for 10 days with ventral approach, then canned
- Avoid hard food for 3 months

30

Fungal rhinitis

- Mainly dog disease!
- *Aspergillus fumigatus/ flavum*
- *Penicillium, Cryptosporascus neoformans*
- Infection first seen in caudal nasal conchae
- Spreads to frontal sinuses with infiltration of maxillae and nasal bones



31

Epidemiology

- 2 age groups
 - 1-4 years
 - 10-12 years
- Mainly male
- Lab/retrievers/ESS/ GSD, collies
- Throughout the year
- Spores commonplace
- ?immunodeficiency



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Clinical signs

- Depigmentation
- Pain around the head
- Sneezing
- Profound depression
- Epistaxis
- Purulent nasal discharge



33

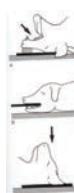
Diagnosis

- No single diagnostic test
- Two or more of:
 - Diagnostic imaging findings (nasal destruction)
 - Isolation of organisms (brush cytology or squash prep, histology or culture)
 - Positive serology of Ab

34

Radiography

- Define the extent
- Presumptive diagnosis
- Locate area for bx
 - Lateral
 - DV
 - Frontal sinus
 - Open mouth oblique
 - caudal cavity and cribriform plate



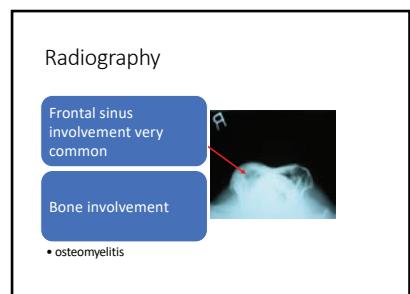
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Radiography

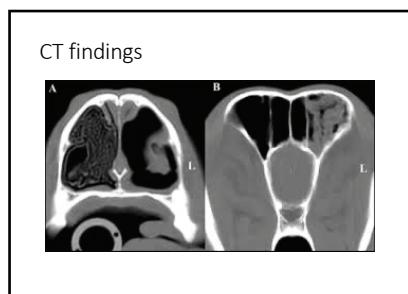
- Turbinate destruction is the main feature
- Localised or generalised
- Unilateral or bilateral



36



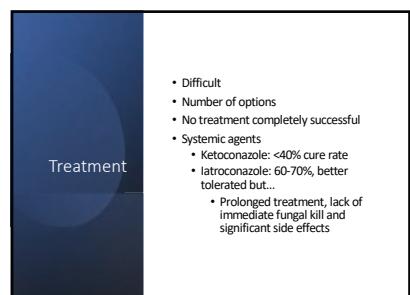
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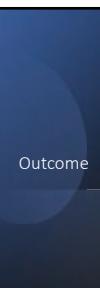
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Treatment

- Enilconazole (Imaverol)
 - Foley catheters remain in sinuses for 7-10 days for absorption
- Works well either as a primary treatment or for cases that don't respond to clotrimazole



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- Cure
 - Clotrimazole and enilconazole
 - About 2/3rds cured after one treatment
 - 85-90% cured after after second treatment
- Persistent discharge
 - + pain/depression/similar in nature
 - Recurrence or persistence
 - Change in nature/ no pain/ no depression
 - Rhinitis/bacterial infection

J Vet Intern Med. 2009 Mar; 23(2): 580-591.
Published online 2009 Jan 27. doi: 10.1111/j.1939-1681.2009.01509.x

PAOLO POGGIO ET ALIA

A retrospective multi-center study of treatment, outcome, and prognostic factors in 34 dogs with disseminated aspergillosis in Australia

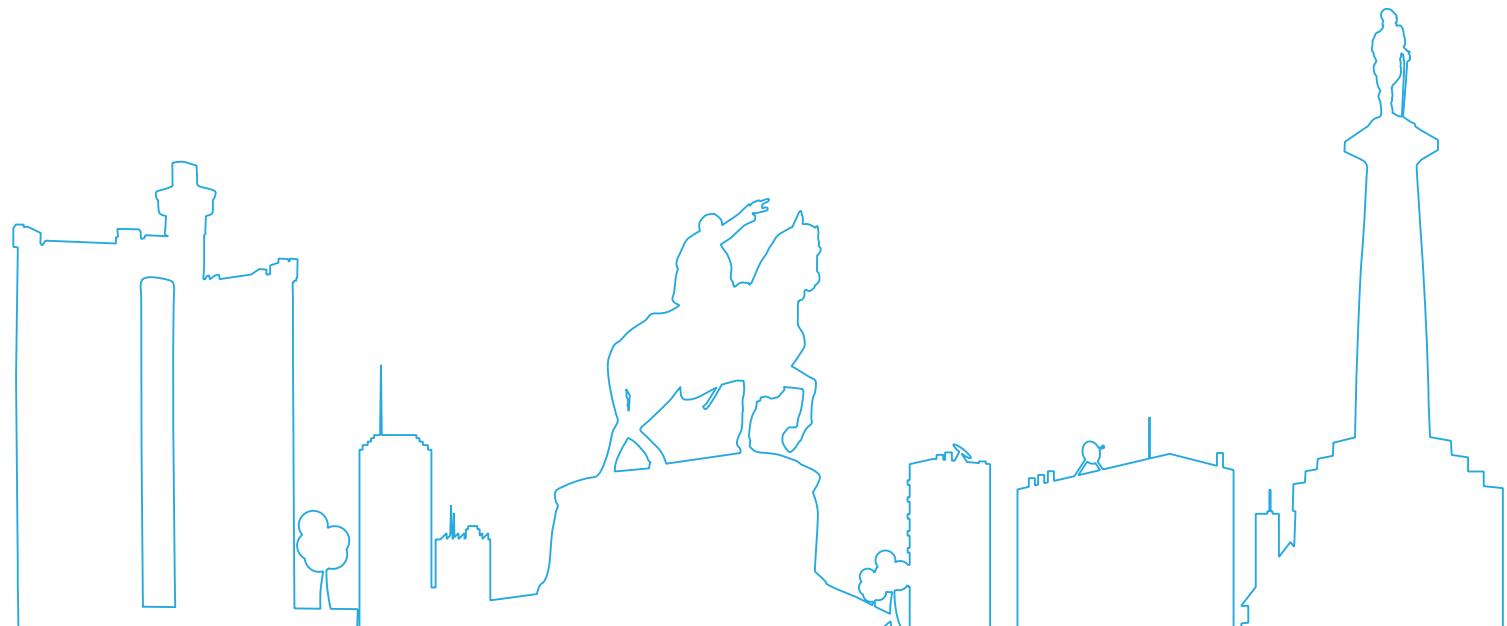
Yvonne Linfoot,¹ H. Caroline Mervielis,² Mark Stevenson,³ Mark Chinnomma,³ G. David Davies,⁴ ^{1,4} Joanne Williams,⁵ ^{1,5} Elise Jacobs,⁶ ^{1,6} Anna Bell,⁷ ^{1,7} Georgia Fox,⁸ ^{1,8} Shelly Booth,⁹ ^{1,9} Vickie Hockenhull,¹⁰ ^{1,10} Quade Dicks,¹¹ and ^{1,12} John H. S. Dennerlein¹²

» Author information » Article metrics » Copyright and License information » PMC Disclosure

- Treatment with itraconazole alone – MST 63 days
- Multimodal antifungal therapy – 830 days
- Serum creatinine was a negative prognostic indicator
- Survival times higher than expected

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VET NURSES / TECHNICIANS





Matthew Rendle (United Kingdom)

RVN

EERVC 2024 Lectures

1. Veterinary Nursing – team work makes the dream work!
2. Fluid Therapy for nurses – what to use and when?
3. First principles of anaesthesia in practice
4. Anaesthesia monitoring – keeping things safe!
5. Fundamental clinical nutrition for nurses
6. Welfare minded blood sampling of pet mammals

Matt started his veterinary nursing career at The Park Veterinary Centre, Watford in September 1989, a busy mixed and exotics 13-vet practice, where he completed his veterinary nursing qualification and was made Senior Theatre Nurse in 1994.

Matt has experience in the nursing care for many species from ants to elephants (and most things in-between) and has been fortunate to travel extensively across the world as part of his roles.

Matt is the Chair of AZEVN which he helped found in 2016 to provide CPD for nurses working with zoo, exotics, and wildlife species.

Matt also is a member of the board of ACOVENE, who help promote and support veterinary nursing education in Europe.

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1. Nicolas et al. "Movoflex® Soft Chews Can Improve Dogs' Mobility, According to Owners". EC Veterinary Science 7.10 (2022): 13-2;

*MOVOFLEX® Cat Soft Chews does not include *Boswellia Serrata*.

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