ESAVA
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KAHUN 1ST

ESAVA Kahun 1st Congress lecture notes

ENHANCING CLINICAL DECISION-MAKING SKILLS IN SMALL ANIMAL PRACTICE

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Veterinarians must make rapid decisions every day about diagnostic and treatment options for their patients. Clinical reasoning skills form the cornerstone of those decisions as well as a sound and appropriate (to the case) knowledge base. The latter also must include an understanding of important pathophysiological principles relevant to the patient's clinical problem. However, knowing the facts is not the same as knowing what to do. Knowledge is only useful if it can be accessed, formulated and applied to the problem at hand. For example, a good understanding the mechanics of an automobile engine are highly useful if the problem to be solved is a malfunctioning car but not as useful when learning to drive.

When a patient presents with one or more clinical problems, there are various methods we can use to solve the case and come up with a list of possible diagnoses to consider. One method involves pattern recognition – looking at the pattern of clinical signs and trying to match that pattern to known diagnoses. This is also referred to as developing an illness script. Another method can involve relying on blood tests to tell us what is wrong with the patient – also referred to as the minimum database. Or we can use problem-based clinical reasoning. Often we may use all three methods.

Pattern recognition?

Pattern recognition involves trying to remember all diseases that fit the "pattern" of clinical signs/pathological abnormalities that the animal presents with. This may be relatively simple (but can also lead to errors of omission) and works best:

- For common disorders with typical presentations.
- If a disorder has a unique pattern of clinical signs.
- When all clinical signs have been recognised and considered and the differential list is not just based on one cardinal clinical sign and the signalment of the patient presented.
- If there are only a few diagnostic possibilities which are:
    - easily remembered or
    - can easily be ruled in or out by routine tests.

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- If the vet has extensive experience, is well read and up to date, reviews all of the diagnoses made regularly and critically and has an excellent memory.

Pattern recognition works well for many common disorders and has the advantage of being quick and cost effective provided ……. the diagnosis is correct. The vet looks good to the client because they have acted decisively and confidently …… provided the diagnosis and treatment are correct.

However, pattern recognition can be flawed and unsatisfactory when the clinician is inexperienced (and therefore has seen very few patterns).

Or, even if the clinician is experienced, it can be flawed for:

- Uncommon diseases.
- Common diseases presenting atypically.
- When the patient is exhibiting multiple clinical signs that are not immediately recognisable as a specific disease.
- If the pattern of clinical signs is suggestive of certain disorders but not specific for them.

In addition, for the experienced clinician, the success of pattern recognition relies on a correct diagnosis for the pattern observed previously being reached and not assuming that similar patterns must equal the same diagnosis. Pattern recognition can lead to dangerous tunnel vision where the clinician pursues his/her initial diagnostic hunch based on pattern spotting to the exclusion of other diagnostic possibilities. Several types or indeed combinations of diagnostic bias can result – some examples are shown in Table 1

Table 1: Diagnostic biases in clinical medicine

Availability bias	A tendency to favour a diagnosis because of a case the clinician has been seen recently.
Anchoring bias	Where a prior diagnosis is favoured but is misleading. The clinician persists with the initial diagnosis and is unwilling to change his or her mind
Framing bias	When then context or way in which a case is presented influences clinical thinking rather than just the clinical facts

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Confirmation bias	When information is selectively chosen to confirm, not refute a hypothesis. The clinician only seeks or takes note of information that will confirm his or her diagnosis and does not seek or ignore information that will challenge it.
Premature closure	Failing to look for additional information after reaching a potential diagnosis and as a result, narrowing the choice of diagnostic hypotheses too early.

And finally, the disadvantage of relying entirely on pattern recognition to solve clinical problems means that should the clinician realise subsequently that their pattern recognition was incorrect they have no logical intellectual framework to help them reassess the patient. Thus pattern-based assessment of clinical cases can result at best in a speedy, correct, "good value" diagnosis but at worst in wasted time, money and sometimes endangers the life of the patient.

I'll do bloods!

Routine diagnostic tests such as haematology and biochemistry can be enormously useful in progressing the understanding of a patient's clinical condition. However, relying on blood tests to give us more information about the patient before we form any assessment of possible diagnoses can be useful for disorders of some body systems but totally unhelpful for others. Serious, even life-threatening disorders of the gut, brain, nerves, muscles, pancreas (especially in cats) and heart for example rarely cause significant changes in haematological and biochemical parameters that are measured on routine tests done in practice. Over-reliance on blood tests to steer us in the right clinical direction can also be problematical when the results do not clearly confirm a diagnosis. The vet can waste much time and the client's money searching without much direction for clues as to what is wrong with the patient. And of course the financial implications of non-discriminatory blood testing can be considerable and many clients are able or willing to pay for comprehensive testing. Using blood testing to "screen" for diagnoses can be misleading as the sensitivity and specificity of any test is very much influenced by the prevalence of a disorder in the population. Many tests do not have absolute "right/normal" or "wrong/abnormal" results. Some have considerable "shades of grey" zones and most importantly, interpretation of any result must be contextual for the individual patient including signalment, demographics and the reason for the testing in the first place.

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For experienced veterinarians, pattern recognition combined with 'fishing expeditions' (i.e." I have no idea what's going on so I'll just do bloods and hopefully something will come up!") can result in a successful diagnostic or therapeutic outcome in many medical cases in first opinion practice. But there are always cases do not yield their secrets so readily using these approaches and it is these cases that frustrate the veterinarians, prolong animal suffering, impair communication with clients and all in all make veterinary practice less pleasant than it should be. You also have to know about and remember lots of diagnoses for this approach to be effective. That is problematical if the vet does not recognise or remember potential diagnoses or if, as discussed above, the pattern of clinical signs doesn't suggest a relatively limited number of differentials. It is also less useful for inexperienced veterinarians or veterinarians returning to practice after a career break or changing their area of practice.

It is for all these reasons we hope this course will enhance your problem solving skills as well as build your knowledge base about key pathophysiological principles. We want to assist you develop a framework for a logical approach to clinical problems that is easy to remember, robust and can be applied in principle to a wide range of clinical problems. The formal term for this is problem-based inductive clinical reasoning.

Problem based inductive clinical reasoning

In problem based inductive clinical reasoning each significant clinico-pathological problem is assessed in a structured way before being related to the other problems that the patient may present with. Using this approach, the pathophysiological basis and leading questions (see below) for the most specific clinical signs the patient is exhibiting are considered before a pattern is sought. This ensures that one's mind remains more open to other diagnostic possibilities than what might seem to be initially the most obvious and helps prevent pattern-based tunnel vision.

If there are multiple clinical signs, e.g. vomiting, polydipsia and a pulse deficit, each problem is considered separately then in relation to the other problems to determine if there is a disorder (or disorders) that could explain all the clinical signs present. In this way, the clinician, if she/he has developed a logical approach to each clinical problem, will be able to easily assess the potential differentials for each problem and then relate them rather than trying to remember every disease process that could cause that pattern of particular signs.

Thus we do look for patterns but not until we have put in place an intellectual framework that helps prevent tunnel vision too early in the diagnostic process.

The initial step in logical clinical problem-solving is to clarify and articulate the clinical problems the patient has presented with. This is best achieved by

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constructing a problem list – either in your head or in more complex cases, on paper or the computer.

Constructing a problem list:

- Helps makes explicit the clinical signs.
- Transforms the vague to the more specific.
- Helps prevent overlooking less obvious but nevertheless crucial clinical signs.

Problem, System, Location, Lesion

Problem-based approach means different things to different people and you may already have read about or been to courses where it is discussed. Some regard the problem based approach as meaning "*Write a problem list then list every differential possible for every problem"*. Not a feasible task unless you have an amazing memory and endless time! Others view the problem based approach as meaning "*Write a problem list then list your differentials".* This is really just a form of pattern recognition but at least it makes a good start by formulating a problem list.

The cornerstone of problem-based approach that is the basis of this course is more accurately defined as problem based inductive clinical reasoning. It provides steps between developing a problem list and deciding on a differential diagnoses list that allows a logical format to be applied to most problems by rigorously pursuing as appropriate the following questions:

The cornerstone of this approach is to always approach each problem logically and (usually) to rigorously pursue, as appropriate, some or all of the following questions:

- What is the problem?
- What system is involved?
- Where within the system is the problem located?
- What is the lesion?

The answers to these questions or the pursuit of the answers will determine the appropriate questions to ask in the history, or alert you to pay particular attention to aspects of the physical examination, or will indicate the most appropriate and logical diagnostic test to use to answer the question as well as prepare you intellectually to assess the results of diagnostic procedures.

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Define the problem

Example:
The owner reports that the dog is vomiting. Is the animal really vomiting or regurgitating or experiencing reflux?

When considering the important clinical signs the patient is exhibiting, it is essential to try to define the problem as accurately as possible. "A problem well defined is a problem half solved" is a good maxim to work from. The first question to ask is "Is there another clinical sign that this problem could be confused with?" This is a vital step and failure to define the problem correctly has often derailed a clinical investigation that might otherwise have been relatively straight forward.

Other examples

- The owner says the dog is having fits - is it having seizures or episodes of syncope?
- The owner says the dog has red urine - is it blood, haemoglobin or myoglobin?

Refine the problem

Some problems require further refining to clarify the best diagnostic approach.

Examples

- Weight loss - because of inappetance or despite a normal appetite?
- Collapse - with or without loss of consciousness?

Why is it so important to define and refine the problem?
The range of diagnoses to consider, diagnostic tools used and potential treatment or management options for clinical problems that may be perceived by the owner to be the same and present similarly to the vet can be very different. Failure to appropriately define and/or refine the problem can often lead to wasted time and money as the wrong problem is investigated or treated. This can delay treatment, prolong the disease, prolong the patient's suffering, sometimes potentially endanger the life of the patient, may increase unnecessarily the costs to the client, frustrate the vet and client and potentially impair the relationship between vet and client.

Define the body system affected and how it is involved

Once the problem is defined the next step is usually to consider the system involved. For every clinical sign there is a system/s that must be involved i.e. it "creates" the clinical sign. But the really important question is – how is it involved? The key questions here are *"What systems could be involved in causing this clinical sign?"* and "*Do I have a primary i.e. structural, problem of a body*

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system or a secondary problem i.e. functional problem where the system involved is affected by other factors?"

Examples

- The body system always involved when a patient vomits is the gastrointestinal (GI) system. But it may be directly involved due to primary pathology of the gut such as parasites, inflammation, neoplasia, obstruction. This is defined as primary (structural) GI disease. Or vomiting may be occurring due to dysfunction of non-gastrointestinal organs such as the liver, kidney, adrenal glands, pancreas. This is defined as secondary (functional) GI disease.
- The body system that is always involved when a patient has generalised weakness is the neuromuscular system. But it may be directly involved due to primary neuromuscular pathology (e.g. inflammation, toxins, neoplasia, infection, degeneration). Or the neuromuscular system may be malfunctioning due to the effect of pathology in other organs causing metabolic derangements that impair neurological function such as hypoglycaemia, anaemia, hypoxia and electrolyte disturbances. This is defined as secondary neuromuscular disease.

Other examples include:

- Chronic cough - cardiac or respiratory system?
- Jaundice - due to prehepatic or hepatic/post hepatic disorder?
- Cardiac arrhythmia - is it due to primary (structural) cardiac disease or extra-cardiac disease, e.g. GDV, splenic pathology?

An alternative, though closely related, question for some problems is ***'Is the problem local or systemic?***

- Epistaxis - due to local nasal disease or systemic disease, e.g. coagulopathy, hyperviscosity, hypertension?
- Melaena - GI bleeding due to local disease (ulceration – which in turn may be due to primary or secondary GI disease) or systemic disease e.g. coagulopathy?

Why is it so important to define and refine the system?

The range of diagnoses to consider, diagnostic tools used and potential treatment or management options for primary, structural problems of a body system are often very different to those relevant to secondary, functional problems of that system. Investigation of primary, structural problems often involves imaging the system in some manner (radiology, ultrasound, endoscopy, surgery) and/or biopsy. Routine haematology and biochemistry are often of little diagnostic value. For secondary, functional disorders on the other hand, haematology and biochemistry are often critically important in progressing towards a diagnosis.

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Failure to consider what body system is involved and how can often lead to wasted time and money. This can delay treatment, prolong the disease, prolong the patient's suffering, sometimes potentially endanger the life of the patient, may increase unnecessarily the costs to the client, frustrate the vet and client and potentially impair the relationship between vet and client. (Notice a recurring theme here?). In fact, if you do nothing else when assessing a case before seeking the diagnostic 'pattern', ask yourself for each of the specific problems - 'what system could be involved and how – primarily or secondarily?'. This simple question will immediately open up your mind to diagnostic possibilities you may never have contemplated if you were just focusing on the 'pattern'.

Define the location

Example:
Having determined that vomiting is due to primary gastrointestinal disease, where in the gastrointestinal tract is the lesion located?

In this example, by asking this question you will select the most appropriate method to either answer the question or to move on to the next step. For example, if you believe that your history and physical examination and other ancillary data indicate a lower small intestinal lesion, endoscopy is not going to be an appropriate method of visualising the area or obtain biopsies. On the other hand, if all information you have suggests a gastric lesion, endoscopy would be appropriate.

Other examples

- Hind limb weakness due to neurological dysfunction - is the lesion in the spinal cord (and where), peripheral nerves or brain?
- Diarrhoea due to primary GI disease - due to small bowel disease? Large bowel disease? Both?
- Haematuria - from urethra, prostate, bladder or kidneys?

Define the lesion

Once the location of a problem within a body system is determined the next key question is "What is it?" i.e. you need to define the pathology. It can be helpful to remember the types of pathology that can occur on broad terms – e.g. degeneration, infection, inflammation, trauma, nutritional, toxic, neoplasia. Which type of pathology is most likely going to depend on the body system or organ involved, the signalment of the patient (species, breed, age, sex etc), the geographic location of the patient and what's common in that population. This can be influenced by whether the patient is in a general clinic or a referral hospital. Common things occur commonly - this doesn't mean that uncommon diagnoses should not be considered (and they will of course be more common in referral

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hospitals). It's just that common disorders usually receive diagnostic priority at the beginning of a clinical investigation.

Example:
The patient has a gastric lesion - ***Is it a tumour, foreign body, or ulcer?***

- This question will require visualisation and/or biopsy to answer but it would have been a waste of time asking the question until you had arrived at the right location.

Other examples

- Spinal cord lesion visible on a myelogram - is it a tumour or granuloma or abscess?
- Haematuria due to lower urinary tract disease - infection, calculi or neoplasia?
- Large bowel diarrhoea - parasites, infection, ulceration, stricture, neoplasia, diet-related?

The final result

Instead of thinking, when faced with a vomiting patient, "I wonder if it has a gastric foreign body or renal failure or a liver tumour?", your initial energies are directed at defining the problem, then the system etc. In this way, the diagnosis is made logically and thoughtfully, and, during the process, all diagnostic options can be considered as the need arises.

Does pattern recognition have a place?
Having said all of this, it is important to reiterate that pattern recognition for many cases is appropriate and justified - *depending on your level of experience*. For example, if a potbellied terrier with bilaterally symmetrical alopecia, seborrhoea, hyperpigmentation and comedomes walked into your consulting room and the owner reported that the dog was drinking lots of water, was ravenously hungry and seemed to be panting excessively, obviously hyperadrenocorticism is the most obvious diagnosis and going through the motions of assessing each specific problem would be ridiculous (but not if you had never seen a dog with hyperadrenocorticism before!).

However, it is important to be aware that pattern recognition is only fool proof if the pattern is virtually unique to the disease or there are a very limited number of diagnostic options and its value is very dependent on the clinician's experience, depth of knowledge, ability to sort data quickly and efficiently etc.

Of course, once you have considered each individual problem you do in fact look for a pattern in the clinical signs. But the insertion of that initial step of considering each specific problem individually and then relating it to the other problems

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present should ensure that you don't miss the less obvious possible diagnoses that you may not have initially been aware of.

There are some combinations/patterns of clinical signs that make the diagnostic options very limited. For example, a patient with PU/PD who is also polyphagic. If the PU/PD and polyphagia have been present for the same length of time then they are almost certainly due to the same disorder and it is quite appropriate to assess them together. There are very few conditions that will cause this pattern of clinical signs so it is quite appropriate to concentrate on these first. Even if the polyphagia is not due to the same pathology there are a number of causes of PU/PD that simply aren't feasible in a dog that is also easting well (because they would decrease not increase appetite).

In addition, the process of developing a sound problem-orientated approach can enhance your ability to recognise patterns, because you have a greater understanding of the reasons why you believe a certain pattern suggests some disorders more than others.

In conclusion

Problem-based inductive clinical reasoning …..

- Is much more than just listing problems, then listing differentials for each problem (a common misconception about problem-based medicine).
- Has 'rules' that are easy to remember and can be applied to most clinical problems animals present with.
- Has a structured approach to clinical problems centred on four main steps as follows:
    - Define +/- refine the problem
    - Define and refine the system
    - Define the location (where appropriate)
    - Define the lesion
- Provides an intellectual and visual framework to hang your knowledge on allowing you to recognise and retrieve more easily the information you need.
- Reduces the need to remember long list of differentials (see the first point).
- Helps prevent getting trapped by a perceived 'obvious' diagnosis – it helps avoid diagnostic bias.
- Provides memory triggers to ensure an appropriate history is taken and a thorough clinical examination performed.
- Provides a clear rationale for choosing diagnostic tests or treatments that can be communicated to the owner.
- Helps turn a terrifying case into a manageable one!

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INTERPRETING THE NUMBERS: WHAT DO INCREASED LIVER ENZYMES VALUES REALLY MEAN?

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ASSESSING THE LIVER

Alanine Aminotransferase (ALT, Formerly SGPT)

This is a liver-specific enzyme in the dog and cat. Highest cellular concentrations occur in the cytosol therefore the enzyme is released following acute and diffuse hepatocellular necrosis. However, the insult does not need to be severe –being central to many body functions, the liver is easily affected by disorders of other organs and has been described as a sympathy organ. Thus, in general, serum levels are not regarded as definitively indicating hepatocellular pathology unless they are at least two to three times above the upper reference limit. Even at this level, non-hepatic disorders can cause moderate increases and the level probably needs to be approaching 5-10 times above the upper reference limit to conclude that the increase is due to primary hepatocellular disease.

As a rule of thumb, levels of ALT that are elevated more than 5 times should be investigated. Levels less than this may not need to be if the animal appears otherwise well. However, if the level is increasing or is persistently increased over a prolonged period of time (even if only 2-3 fold) further investigation may be warranted. See comment re cats below.

Non-hepatic disorders that will cause ALT to be moderately (3-5 times reference range) increased include:

- Treatment with anticonvulsants or glucocorticoids
- Biliary stasis for any reason
- Hypoxia
- GI disease - especially if inflammatory or infectious
- Haemolysis (in the cat but not the dog)

The serum half-life of ALT is approximately 30 hours in dogs. Levels peak two to three days after hepatic insult and return to normal in one to three weeks if hepatic insult resolves. Persistent increase indicates continuing hepatocellular insult.

The half life of ALT is shorter in cats than dogs (3-5 hours). This tends to mean that levels that occur in hepatic disorders in cats may be lower than those achieved by a similar degree of pathology in dogs. As such I suspect clinically significant

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hepatocellular disease even with mild ALT increases (2-3 times reference range) in cats.

AST

AST levels are also increased in dogs and cats with hepatic necrosis. The enzyme is not specific for liver disease as it may increase with muscle necrosis. However, it has been suggested (but not to my knowledge conclusively demonstrated in dogs or cats) that because AST represents liberation of mitochondrial isoenzymes, if AST is higher than ALT (and the CPK is normal, ruling out muscle disease) this may reflect the presence of severe hepatocellular injury associated with organ devitalisation. AST does not appear to be increased by phenobarbitone treatment (in contrast to ALT).

Alkaline Phosphatase (ALP)

ALP is bound to membranes of bile canaliculi and bile ducts. Values are increased by any condition causing cholestasis, either intra- or extra-hepatic. Cholestasis results in increased synthesis and regurgitation of the enzyme from the biliary system into the serum.

Other isoenzymes of ALP are also found in bone, intestine, kidney tubules and the placenta. However, the half-life of the intestinal, renal and placental isoenzymes are so short (two to six minutes) that serum elevations of ALP would rarely occur from these organs.

Usually an elevation in ALP is due to hepatic or bone isoenzymes. However, exogenous and endogenous glucocorticoids can induce a specific isoenzyme and thus result in elevated serum levels in the dog (but not in the cat). The value in measuring the ALP isoenzyme in the diagnosis of hyperadrenocorticism is questionable (see reference in Module 4).

ALP levels will be increased in young growing animals (bone isoenzyme) and in destructive bone disease. ALP is also increased in certain carcinomas and mammary gland tumours, and with anticonvulsant therapy in dogs but not cats. ALP is commonly increased in cats with hyperthyroidism.

The half-life of ALP varies between dogs and cats. In the dog, the half-life is 72 hours, in the cat only six hours. As well, feline bile canaliculi do not excrete as much ALP in cholestasis as dogs. Therefore, any elevation in cats is very significant and cholestasis and jaundice will occur often without an elevation in ALP. In contrast to the dog, systemic conditions, other than hyperthyroidism and bone growth, do not cause induction of ALP synthesis without concurrent cholestatic disease.

The pretenders

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Non-hepatic disorders that can result in a substantial increase in ALP include:

- Glucocorticoid administration or hyperadrenocorticism – in dogs but not cats
- Some tumours e.g. mammary tumours
- Pancreatitis
- Old dogs (nodular hyperplasia, asymptomatic vacuolar hepatopathy)

Asymptomatic vacuolar hepatopathy

These patients have a high ALP but no clinical signs. They are often detected because of a routine pre-anaesthetic screen. If a biopsy is done the histopathological changes seen are similar to glucocorticoid hepatopathy. It appears to be breed associated in Scottish terriers. Some patients can have hypertension and proteinuria - monitor and treat as necessary

Gamma Glutamyl Transpeptidase (GGT)

GGT levels are increased in most conditions that cause elevation in ALP, i.e. cholestasis, glucocorticoid therapy, hyperadrenocorticism. However, unlike ALP, GGT is not elevated with increased osteoblastic activity (e.g. growing dogs) and may not be elevated in dogs on anticonvulsant medication. It is not increased by corticosteroids or anticonvulsants in cats.

ALP is slightly more sensitive than GGT for detection of cholestatic disease in dogs but GGT is significantly more sensitive than ALP in cats – therefore it is a useful enzyme to include in the biochemical profile of the cat (but not much use in the dog). GGT increases may be greater than ALP in feline extrahepatic bile duct obstructions, cholangio-hepatitis and cirrhosis. Disproportionately low GGT with high ALP can be a useful pointer to hepatic lipidosis. In both dogs and cats, acute hepatic necrosis is unlikely to cause an increase in GGT whereas ALP may be elevated.

Liver Enzymes - General comments

In acute hepatic injury, enzyme (ALT, AST) release is proportional to the amount of affected tissue. However, note that liver enzymes are not liver function tests and in sub-acute and chronic hepatic disorders there is no correlation between the magnitude of the enzyme increase and severity or reversibility of the condition though they might be used to monitor the progress of an individual patient. Several serious and potentially irreversible liver diseases such as cirrhosis and portocaval shunts may be associated with only mild-moderate enzyme changes.

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Bilirubin

Bilirubin is primarily formed from the breakdown of haemoglobin from aged red blood cells in the mononuclear phagocytic system (MPS) of the spleen, liver, bone marrow and lymph nodes. A small amount is derived from myoglobin and heme-containing liver enzymes. Haemoglobin is split to release a protein molecule and heme (iron containing porphyrin). The iron is released and stored in RE cells or reutilised for heme synthesis. The remainder of heme is converted to biliverdin and then to bilirubin.

Bilirubin is transported to the liver in blood tightly bound to albumin = **unconjugated bilirubin**. Unconjugated bilirubin is not water-soluble and is **not** filtered by the renal glomeruli or excreted by renal tubules.

Dogs (males more than females) have a low resorptive threshold for bilirubin. They also have renal enzyme systems that produce and conjugate bilirubin to a limited extent. Therefore, mild bilirubinuria (up to 2+) can occur in normal dog urine of specific gravity greater than 1.025.

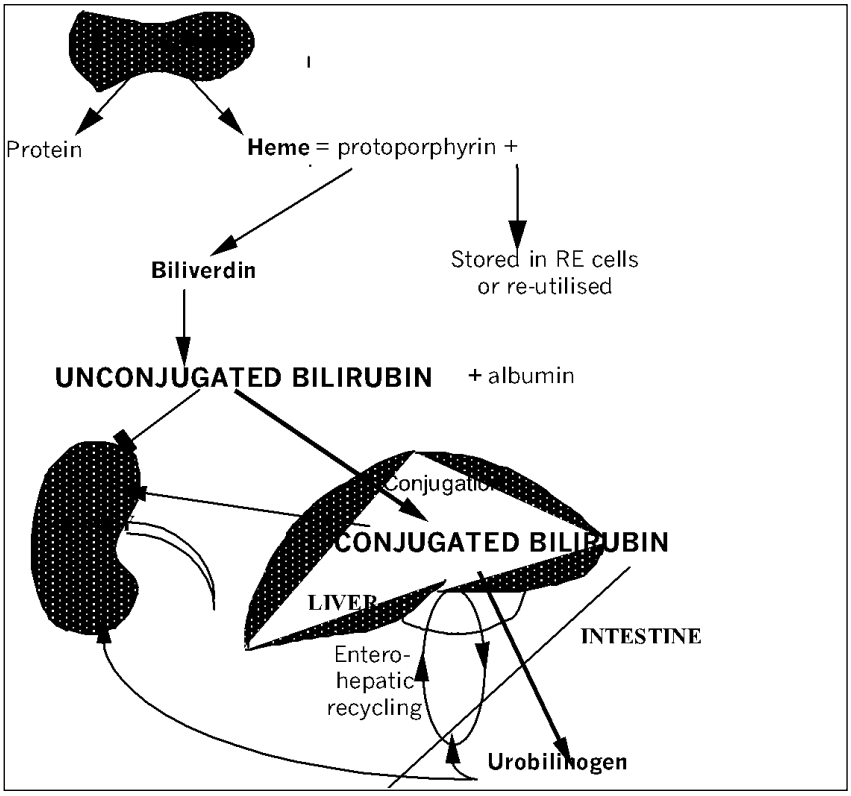
In contrast, cat kidneys cannot conjugate bilirubin and their renal threshold is nine times higher, therefore **bilirubinuria in a cat is always pathological**.

In the liver bilirubin is conjugated in hepatocytes and excreted into bile canaliculi and thence transported to the bile ducts and intestine. This conjugation and excretion maintains the concentration gradient across the blood-hepatocyte membrane for continued uptake. The canalicular excretion phase of bilirubin metabolism is the rate limiting step in this process and will be overloaded first if there is excessive demand for bilirubin excretion.

The metabolic pathway for bilirubin is illustrated in Figure 1.

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Figure 1: Bilirubin Metabolism



Unconjugated bilirubinaemia is detected by the indirect van den Bergh test. Conjugated bilirubinaemia: by the direct van den Bergh test. However, differentiation of conjugated and unconjugated bilirubin is rarely diagnostically useful.

Conjugated bilirubin is water-soluble, dialysable and filtered by the kidneys; therefore, it can appear in urine. Trace to two plus bilirubinuria is normal in dogs as they have a low renal threshold for conjugated bilirubin and a low rate of renal conjugation can occur.

Slight bilirubinuria will also occur in *starvation* and *febrile states* and, as previously mentioned, mild bilirubinaemia and bilirubinuria can also occurs with *sepsis*. Bilirubinuria will develop well before overt jaundice in dogs due to the low renal threshold.

In cats, the renal threshold for bilirubin is nine times that of dogs, therefore any bilirubinuria in the cat indicates altered bilirubin metabolism and should be investigated. Note that any cat with bilirubinuria will also be jaundiced.

Conjugated bilirubin excreted through the bile duct into the small intestine is converted by colonic bacteria to **urobilinogen** (colourless), which is oxidised to **urobilin** (orange).

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Some urobilinogen is reabsorbed by the intestine, enters the enterohepatic circulation and is re-excreted into bile. A small amount is also excreted into urine (about 5%). Urobilinogen remaining in the intestinal tract is converted to stercobilinogen which is oxidized to stercobilin, which imparts normal faecal colour.

Urobilinogen is a useful test in people but is not useful in cats or dogs.

Complete bile duct obstruction or traumatic bile duct transection results in acholic faeces as it is the bile pigments that impart faecal colour.

Obstruction must be complete as sufficient stercobilin can be formed from only a small amount of bile pigment. The faeces of animals with bile duct obstruction are also pale because of impaired fat assimilation resulting in steatorrhoea

Serum Albumin

Albumin is synthesised only in the liver. A loss of greater than 70 per cent of liver function is required before hypoalbuminaemia occurs. Hypoalbuminaemia most commonly occurs in cirrhosis and portosystemic encephalopathy but will also occur in severe diffuse necrosis. Albumin concentrations may also be decreased in renal and gut disease, severe cutaneous burns, protein malnutrition, in the presence of acute phase reactants and in patients with exudative effusions (which causes sequestration of albumin).

Serum Globulins

Increased serum immunoglobulin levels may occur in inflammatory hepatic disease or when the hepatic mononuclear phagocytic system is compromised. Decreased levels will often occur in portosystemic encephalopathy as a large proportion of globulins are synthesised in the liver.

Cholesterol

Very low serum cholesterol concentrations may occur in patients with congenital or acquired portosystemic shunts and fulminant hepatic failure. Increased serum cholesterol in a jaundiced patient usually indicates widespread bile duct occlusion, particularly in cats. However, cholesterol concentrations are also increased in non-hepatic diseases such as pancreatitis, diabetes mellitus, hyperadrenocorticism and hypothyroidism which, if present concurrently, can confuse interpretation.

Ammonia

Ammonia is normally detoxified in the liver via the urea cycle. There is a large reserve capacity for this process. Hyperammonaemia most commonly occurs with portosystemic shunts, either congenital or secondary to cirrhosis. Blood

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ammonia is also increased in severe, diffuse, acute necrosis or severe chronic necrosis (more than 70 per cent loss of function).

Coagulation Tests

In severe hepatic disease, coagulation abnormalities may occur as a number of clotting factors are synthesised in the liver. In addition, activation of vitamin K-dependent factors II, VII, IX and X also occurs in the liver.

Bile Acids

'Primary' bile acids (cholic, chenodeoxycholic) are synthesised only in the liver and are secreted into bile after being conjugated with various amino acids (primarily taurine). Once released into the small intestine when the gall bladder contracts (where they facilitate fat absorption) 'primary' bile acids are efficiently reabsorbed into the portal vein and returned to the liver for reuptake and resecretion into bile. A small percentage of primary bile acids escape reabsorption and are converted by intestinal bacteria to secondary bile acids (deoxycholic, lithocholic), some of which are also reabsorbed into the portal circulation.

Absorption of bile acids by the intestine is extremely efficient, but hepatic extraction from the portal vein is not, which results in a small proportion of the bile acids being present in the blood of normal cats and dogs.

Abnormally high levels occur if there is disturbance of hepatic excretion into bile or disruption at any point along the path of portal venous return to the liver and hepatocellular uptake.

Bile acid levels will increase in blood when there is:

- Decreased clearance of bile acids from portal blood
    - Diffuse hepatocellular disease
    - Portacaval shunting

Or

- Decreased bile acid excretion in bile
    - Obstructive cholestasis – intrahepatic
    - Post-hepatic cholestasis

Bile acids can be increased well above normal by GI bacterial metabolism so levels should be interpreted with care in patients with diarrhoea where the bacterial flora balance may be disturbed.

Serum bile acids are a sensitive test for the presence of liver, biliary or hepatic vasculature pathology in the cat and dog. They should be considered when other routine clinical pathology results do not permit an unequivocal diagnosis of liver

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disease to be made. It is not necessary to do the test if the patient is jaundiced and not anaemic or if liver enzyme changes permit an unequivocal diagnosis of liver disease to be made.

Bile acids cannot be used to estimate the severity or extent of liver injury nor determine the type or distribution of pathology. Quantification of bile acids only indicates the presence of 'significant' liver injury (if there are few hepatocytes available to recycle bile acids, increased amounts spill into blood), altered bile flow or altered hepatic vasculature usually due to portal vein abnormalities or development of collateral shunts as a result of portal hypertension. Relative changes to abnormal values cannot be used to quantify the improvement or worsening of a condition. A value is either abnormal or normal and has no linear relationship to the extent of circulatory or tissue compromise due to the many variables involved in increasing or decreasing their levels in the blood.

They are useful as a screening test for hepatic encephalopathy (except in Maltese dogs - see reference). Their major advantage in this context is the lack of stringent requirements for sample collection and processing in contrast to blood ammonia determination. The sensitivity of a post-prandial bile acid test for detecting portacaval shunting is close to 100%, though there are the occasional patients with shunts who will have normal bile acids.

Bile acids can be normal in patients with hepatic disease depending on the reference range used. Reviewing the results of various studies it would appear that roughly 20-25% of cases of confirmed hepatobiliary disease can have fasting bile acids less than 20μM and postprandial bile acids less than 25μM.

In a recent study at the RVC of 217 dogs (99 with normal livers, 218 with confirmed hepatic disease – parenchymal, vascular or biliary), 40 dogs with confirmed level disease had pre-prandial BAs < 25 μg/L. 16 dogs had post-prandial levels less than 25 μg/L. The bile acid level both pre- and post-prandially was less than 25 μg/L in 9 dogs. These were the "false negatives".

With reference to the "false positives", 9 dogs without liver disease had a pre-prandial bile acid level > 25 μg/L and 19 has a post-prandial level >25 μg/L. It was only when results were greater than 34 μmol/L that the false positive rate was less than 10% and the cut off for 100% specificity (i.e. no false positives) was 58 μmol/L.There were smaller number of cats in the study (18 with normal livers, 21 with hepatic disease).

- 11 cats with confirmed liver disease (52%) had pre-prandial BAs < 25 μg/L
- 7 cats with confirmed liver disease (33%) had post-prandial BAs < 25 μg/L

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- 5 (24%) cats with confirmed liver disease had both pre and post BAs < 25 μg/L

The take home message is to interpret bile acid results with great care as they neither rule hepatic pathology in or out. It is usually recommended that values greater than 40μM warrant biopsy if possible to determine the aetiology (unless due to portocaval shunting).

Serum bile acid concentrations are usually not affected by corticosteroid administration but occasionally they can be markedly altered due to alteration of hepatic architecture as a result of hepatic glycogen accumulation. Serum bile acids are therefore useful but not infallible for differentiating elevated ALP values due to steroids (endogenous or exogenous) or hepatobiliary disease. About 20% of patients with hyperadrenocorticism will have increased bile acids.

Portal vein hypoplasia

Dogs with portal vein hypoplasia (PVH) have no clinical signs but their bile acids are increased. Breeds affected similar to portacaval shunts (PSS) e.g. Yorkshire terriers, Maltese, Cairn terriers. You may become aware of them because of a slight increase in ALT on a routine blood profile e.g. pre-anaesthetic which is further investigated by doing bile acids. Dogs with PVH can have a low Protein C wheras dogs with PSS do not. They require no treatment and 95% live normal lives although very occasionally a dog may develop ascites.

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Integrating quality of life assessment into the management of patients with chronic disease (case discussion)

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Introduction

In recent times, quite rightly, a heavy focus has been applied on the value of practising contextualised care. At times though, communications surrounding this topic, imply tension to exist between evidence-based medicine approaches suggesting a gold standard to exist and such contextualised care approach. This tension does not need to exist when we apply evidence-based medicine to contextualised care. Since: how can we practise contextualised care without knowing for certain which target to hit and whether we are, for certain, hitting that target with our treatment? How can we, as vets, and thus veterinary scientists, be truly scientific about contextualised care? Because if we are not scientific about contextualised care, we risk that we are vulnerable to not aligning to the context of contextualised care because of subjectivity, bias and lack of evidence.

This lecture will explain that for us to practice contextualised care it is absolutely essential that we as a profession start to systematically, patient-by-patient, integrate a missing link into our exam room behaviour. What is this missing link? Applying an evidence-based approach to documenting and monitoring quality of life of pets and their owners using psychometrically validated and individualisable quality of life measurement tools. Why? Well, these scientific tools will tell us whether our chosen diagnostic or treatment trajectory gets the outcome that the context, the pet and the owner are most suited by.

Examples of these tools will be provided to implement this approach and delegates will be introduced to the impartial website ***www.qol-pet.com***, which

hosts an ever-expanding collection of these tools, free for use by colleagues and caregivers. After this lecture, no one should ever practice without quality of life measurement ever again.

Why do we need to measure quality of life in our animals?

When I teach colleagues on this subject, I regularly hear: "With my experience, I can now assess quality of life very well myself – I am much better at it than those mathematical calculations of yours!". And most of the time I answer that I believe that will be true most of the time (and I believe it is). But I also point out that it is important that practitioner and owner look at quality of life in the same way. As soon as there is a difference of opinion about quality of life, this leads to tension in the relationship between client and practitioner, as well as to a suboptimal climate for making the right decisions. The need for euthanasia may then be clear to such a colleague, but cannot be carried out because of an owner who sees it all differently. It is also essential to qualify and quantify the context, in order to be able to truly practice contextualised care.

A second point I would like to make clear to colleagues why it is worth the time investment to use quality of life assessment tools is the following: even the most experienced colleague is not always right. And if we are wrong, it comes at a high price, namely the suffering of the animal.

To illustrate: the lack of structured quality of life assessment tools has already led to a small revolution in human medicine years ago. This is in response to a number of remarkable studies. Sugarbaker et al. (1982) looked at quality of life in people under treatment for sarcoma of the legs. The researchers hypothesized that a new surgery and radiation treatment, in which the leg was not amputated, led to better quality of life than the old treatment, leg amputation. You would say a reasonable assumption, because who wouldn't want to keep both his or her legs?

But then came the surprise: when they looked in more detail, using validated quality of life assessment tools, they saw that patients who did go for amputation reported a better quality of life. They scored better on questions about psuchoscoial impact on their lives, overall function, economic impact, mobility, sex-life and pain. If this had not been specifically looked at, the conclusion might have been very different.

A second interesting conclusion of this study was that just asking about all aspects of their quality of life had a positive effect on the quality-of-life scores of the patients. In other words, it made the relationship between caregiver and care recipient more positive and powerful.

These are powerful examples that make it clear that we in veterinary medicine also need to take quality of life assessment more seriously. We must assume that, as in human medicine, this greatly benefits the quality of our care. For example, if an anxious cat needs daily oral medication, we may see the negative impact of the disease decrease, but this can be largely overshadowed if there is also a daily fight between cat and owner because of that same medication.

And if we regularly talk about quality of life throughout the animal's life, together with the owner, in a structured way, using tools developed for and by pet owners themselves, and validated in a scientific way (using so-called psychometric statistics), this will certainly lead to a greater chance that owner and caregiver will choose the same point to proceed with euthanasia.

But you can't measure quality of life, can you?

The tendency is to believe that this is a subjective subject; You can't argue about taste either, right? We can't measure quality of life objectively, can we? Nothing could be further from the truth. How do we measure blood sugar? The glucose is metabolised by an enzyme in the sensor, which releases electrons, which is translated into an electrical signal, and which is translated into a number. That number is just a number – in every intermediate step of this measurement process, an inaccuracy creeps in. Nevertheless, if you do enough tests with those blood sugar levels, you will see that there is a pattern of truth in it. We can also do this when measuring seemingly immeasurable entities, such as quality of life. If you then think that you have created something that measures quality of life, you use it with hundreds of owners and animals. You analyse the results of this with so-called psychometric statistics to look at the patterns of shifts. If the tool doesn't work properly, those patterns are missing; If it does work well, the statistics show this.

What kind of tools can we use in practice?

If we look at human medicine, especially in studies of people who cannot communicate well (think of babies, children, elderly people with dementia), we suddenly learn that these techniques have been used successfully for years and

are a regular part of studies, as well as daily practice. Veterinary medicine is therefore lagging far behind; But we quickly make up for that deficit. Whereas in the past the focus was usually only on blood values and survival times, and not on objective quality of life parameters, more and more studies are appearing with examples of tools that we could all use in our practices tomorrow.

In general, there are two types of tools: tools that assess overall quality of life (see e.g. the study "Psychometric Validation of a General Health Quality of Life Tool for Cats Used to Compare Healthy Cats and Cats with Chronic Kidney Disease.") and tools that are disease-specific (see e.g. the study Development and evaluation of a health-related quality-of-life tool for dogs with Cushing's syndrome.). The advantages of overall quality of life assessment tools are that they can be applied to any animal with any disease; They are often short and therefore quick to use. The main disadvantage is that they do not address the impact of specific aspects of specific diseases and their treatment. For conversations about the aging animal and also euthanasia conversations, the overall quality of life tools can be very useful.

In an ideal world, but especially in difficult situations (for example, when the care team and owner do not agree on the time point of euthanasia), it can be useful to use two or more tools (one general tool and one disease-specific tool). This can then make both parties think in the same way about what quality of life is and what exactly it means for the animal in question. Just as we look at metabolism with a blood biochemistry profile of the laboratory, it is wise to see quality of life in the same light: blood biochemistry measures different metabolic factors (e.g. related to liver, kidneys, muscles, etc.) and we can choose a minimal blood profile or a complete profile. A single quality of life assessment tool looks at quality of life (or a part of it) in one way; Two tools see more than one, three even more.

Nevertheless, it would be nice if we clinicians and paraveterinarians started using at least one tool regularly throughout an animal's life. This will probably also prevent us from ending up in a situation where the care team considers euthanasia to be correct because of suboptimal quality of life, while the owners do not agree with this. Ideally, therefore, the use of one of the available tools should be introduced before the quality of life has deteriorated or before illness or aging processes occur.

Many of these studies are in the English language, but nevertheless often easy to consult on the internet or to translate by us in practice.

Where can I find these tools?

Many examples of quality-of-life assessment tools are assembled on the independent, free to use website: www.qol-pet.com.

Now there is truly no excuse anymore to not use quality of life assessment in your clinic as from tomorrow.

Finally

If we want to truly practice contextualised care, quality of life measurement through several tools, ensures that we are still practising evidence-based medicine. This way contextualised care effects can be established, compared and we avoid it becoming a term used to justify choosing treatment modalities for the wrong reasons or due to subjective misconceptions (which we all have!). Let's start using quality of life science as well as possible to continue to live up to our veterinary oath in a robust, scientific manner.

What does that result really mean? Navigating the labyrinth of laboratory testing

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Introduction

Diagnostic testing is a cornerstone of veterinary medicine, yet its application often falls short of providing the clarity clinicians expect. This presentation examines why diagnostic tests can mislead, particularly when applied to **clinically healthy animals** or in **low-prevalence contexts**. It also debates the necessity of health screening programmes which integrates blood and urine tests, especially relevant given the various pros and cons of such programmes, and the increased availability of in-house testing to veterinary teams and patient populations.

The lecture, through a range of examples, will emphasise that tests are not absolute arbiters of disease. Their usefulness depends on four main factors:

1. **Prevalence of disease in the population tested**
2. **Accuracy of the diagnostic test**
3. **Impact of detecting the disease**—whether treatment exists and improves outcomes
4. **Clinical context**—patient characteristics, including signalment, history, physical examination

The key theme is that **testing is only truly beneficial when prevalence is high**, meaning the prior probability of disease is already elevated due to signalment, risk, or clinical suspicion.

The lecture also aims to show that testing is not a harmless affair.

Screening Blood Tests in Healthy Animals or The Temptation of Universal Screening

It might seem a good idea to perform routine **screening bloodwork** in healthy cats and dogs. Many clinicians feel reassured by testing, owners may demand it, and early disease detection is often used as justification. However, the reality is more nuanced. For screening to make sense, four conditions should be met:

1. **Prevalence is high** – Disease is common in the population tested.
2. **Disease may be occult** – It can exist without obvious clinical signs.
3. **Tests are accurate** – Sensitivity and specificity are high enough to minimize errors.
4. **Treatment is effective** – Early intervention truly leads to better outcomes.

While these criteria may apply in selected contexts (e.g., chronic kidney disease in older cats), they often do **not** apply to the general population.

The Role of Prevalence in Predictive Values - Understanding Predictive Values

The presentation stresses the difference between **sensitivity/specificity** and **predictive values**:

- **Sensitivity & specificity** are inherent to the test.
- **Positive predictive value (PPV)** and **negative predictive value (NPV)** depend on **disease prevalence**.

Formulas (TP = true positive; FP = false positive; TN = true negative; FN = false negative):

- PPV = TP / (TP + FP) × 100
- NPV = TN / (TN + FN) × 100

When prevalence is low, even a highly specific test yields more **false positives** than true positives.

Example: using liver enzyme determination to screen for chronic hepatitis in dogs

- **General crossbreed dogs, without clinical signs**: some papers suggest a prevalence of 0.07% in the crossbreed population.

    - Calculated PPV = **0.12%** (12 correct diagnoses in 10,000 positives)
    - Almost all positives are false.
- **Labradors, higher-risk group**: Prevalence 10%
    - PPV improves to **16%** (16 true diagnoses in 100 positives)
    - Still, 84% of positives are **not** true disease.

The conclusion: in low-prevalence groups, screening produces **false reassurance or unnecessary alarm**. Testing only makes sense in breeds or populations with **substantially higher prevalence** and if we are prepared to also subsequently do additional testing, which, for liver disease, includes taking a large enough biopsy of the liver to attain sufficient certainty to include/exclude disease. However, do we truly want that? The answer to this question will likely come best from a discussion with an individual client on the basis of the individual pet, though is not easy to follow for the average pet owner. For us to truly practice contextualised and appropriate medicine, an educated, balanced veterinary surgeon is needed to ensure the discussion is led on the basis of the above figures.

The Illusion of Security from Normal Results

Owners often value normal test results because they provide **peace of mind**. Yet, this reassurance can be misleading:

- Very often, a normal test does not rule out disease (limited sensitivity). This is true for most parameters that we screen for in the general health screening profiles.
- This false sense of security may foster **false security**, delaying recognition of emerging clinical signs.

Thus, clinicians must weigh whether testing provides genuine clinical value or just **psychological comfort (to owner and clinician), as well as whether this psychological comfort is justified**. Studies investigating the sensitivity of liver enzyme screening for chronic hepatitis ranged from 13-71% at best. In the end, anyone wanting to re-assure oneself there is no significant liver disease, would need to do more testing than only blood tests and very often would need to be prepared to do a liver biopsy. The latter entails significant risk and costs, thus should be equally questioned.

Example: Screening for urinary tract infections in non-clinical patients

Definition and Problem: Subclinical bacteriuria is the presence of bacteria in urine cultures without clinical signs of urinary tract infection (UTI). Routine screening in populations such as pets with diabetes mellitus, renal disease, Cushing's patients, or those on corticosteroids is common—but often misguided.

Evidence

- **Dogs & cats with subclinical bacteriuria**: No proven link with later cystitis or systemic disease.
- **Healthy female dogs**: 9% bacteriuria detected, but no progression to UTI during 3-month follow-up.
- **Older cats**: 10–13% had subclinical bacteriuria, but it did not shorten survival even without antibiotics.

Implications

- Treating subclinical bacteriuria has risks: inducing antibiotic resistance, adverse effects, owners getting scratched or bitten, microbiome derangements, diarrhoea, costs.
- Not treating poses minimal risk.

Therefore, screening for UTIs in non-clinical animals is currently discouraged. Instead we see subclinical bacteriuria as a physiological phenomenon that represents a shift in the local microbiome, not necessarily pathology.

Example: Blood Pressure Measurement in Cats

Hypertension screening in older cats (>9 years) provides another instructive case:

- **Prevalence**: Around 1 in 6 cats (likely higher in some populations).
- **Tests**: Doppler or oscillometric measurement; variability and operator technique matter.
- **Clinical consequences**: Hypertension is silent but causes blindness, brain and renal damage, as well as potential cardiac disease.
- **Treatment**: Effective and improves outcomes.

Here, the conditions for **worthwhile screening** are satisfied because prevalence is substantial, disease can be occult until it is too late and intervention alters prognosis.

Example: C-Reactive Protein (CRP) and Inflammatory Markers

CRP and similar acute-phase proteins are often used to detect or monitor inflammation.

However, the lecture highlights that:

- CRP is **non-specific**: it rises with many inflammatory or infectious conditions.
- It cannot pinpoint the disease cause.
- Utility lies more in **monitoring progression or response to therapy** than in initial screening.

Once again, testing indiscriminately in **low-prevalence, asymptomatic animals** generates more confusion than clarity.

Example: Hormonal Testing and Cushing's syndrome

Cushing's syndrome (hyperadrenocorticism) illustrates the problem of testing in low-prevalence disease with non-specific signs.

- **Prevalence**: Low in general dog populations.
- **Clinical signs**: Often vague (polyuria, potbelly, lethargy).
- **Tests**: ACTH stimulation test and low-dose dexamethasone suppression test—neither highly accurate.
- **PPV**: Low in low-prevalence groups, leading to many false positives.

The presentation shows how artificial intelligence-based prediction models using **multiple risk factors** (signalment, bloodwork, clinical signs) can improve pre-test probability, thus enhancing PPV before formal testing.

This reinforces the central rule: **raise prevalence first (by careful case selection), then test.**

Example: screening for pancreatitis

Pancreatitis is notoriously difficult to diagnose with confidence, even on postmortem examination. There was therefore a sense of relief when specPLI was introduced to the veterinary landscape promising a single blood sample to help us out. The easy to use SNAP-variant has since become very popular too.

More recent use has highlighted to danger of indiscriminate use of blood-based parameters to diagnose pancreatitis. These parameters have also been shown to elevate under a range of other conditions than mere (primary) inflammation of

the pancreatic tissue, thus leading to a significant risk of misdiagnosis. Clinicians are therefore urged to not blame “pancreatitis” too quickly and remain openminded, especially if the clinical signs do not fit (completely) and other options have not been excluded. In addition, although a normal specPLI / SNAP makes pancreatitis much less likely, the disease can still be present.

The Owner’s Role and Demand for Testing

Nonetheless, we need to acknowledge a practical challenge:

- Motivated owners often **demand extensive testing**, believing it guarantees best care or even blame us clinicians when we happen to have a case that encounters difficulties prior to us having done routine testing (e.g. pre-GA)
- Normal results sometimes provide reassurance, even if clinically unhelpful.

These thoughts might lead to us vets over-testing to satisfy expectations. The recommendation therefore probably should be that we shift our focus from “testing everything” or “client-driven testing” to **individualized, risk-based screening including the client characteristics**. In this, we emphasize importance of **signalment, history, and clinical examination** as the primary filters before testing.

Artificial Intelligence (AI) in Diagnostics

AI and machine learning models can integrate large datasets (clinical signs, lab results, imaging) to estimate disease likelihood and thus pre-test probability – that is a good thing overall.

Advantages:

- Improve **pre-test probability** by stratifying patients into high or low risk.
- Potentially outperform single diagnostic tests in predictive power.

Challenges:

- Require validation and careful oversight. AI can show significant bias and when developed in a different population than where it is applied (e.g. American pets vs UK/European pets, different laboratory machines being used with different reference intervals to develop the AI-tool) the results can be unreliable/ inaccurate.

Therefore, AI-tools should never replace clinician judgment and rather complement it.

Clinical Philosophy: Back to Basics

In the end the **best care** is not necessarily "more testing." Instead, it comes back to good old pillars of being a veterinary scientist and clinician:

1. **Take time for thorough history and examination.**
2. **Build a structured problem list.**
3. Recognize that the **problem list is often more valuable than test results** in guiding management.
4. Identify the body system(s) at play either in a primary capacity or secondarily
5. Use tests to **confirm or exclude** suspicions in high-prevalence contexts—not as blind screens.

Practical Guidelines for Testing Strategy

All in all, it serves us to following the several guiding principles:

1. **Always consider prevalence.**
    - If prevalence is very low, testing is likely to mislead.
    - Only screen in high-risk groups or patients with clinical signs.
2. **Use predictive values, not just sensitivity/specificity.**
    - A test's PPV and NPV change dramatically with prevalence.
3. **Choose individualized screening.**
    - Focus on breeds, ages, or conditions with known higher risk.
4. **Avoid overtreatment of incidental findings.**
    - Subclinical bacteriuria, enzyme elevations, or equivocal hormone tests may not require intervention.
5. **Communicate with owners.**
    - Explain why testing indiscriminately may do more harm than good.
    - Consider owner's needs, desires and circumstances, including economics, realising that more testing because of a large budget being available is not always (or very often?) not the right way forward.
    - Emphasize monitoring and vigilance for clinical signs instead. Owners can be trained better to be better observers. This does take time and effort but is the wisest of investments into the welfare of your patient population.

Diagnostic tests are not inherently good or bad—their value depends on us clinicians.

We should always seek to integrate the approach with what is right by the owner and the pet as part of practising contextualised (and thus individualised) care.

Overall, using these principles in a holistic manner therefore also likely requires a veterinary surgeon to be a human being – in that sense, we are not being replaced by algorithms and diagnostics test facilities just yet!

Dealing with respiratory diseases – an interactive discussion

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When the Waiting Room Holds Its Breath….

Few sights in practice quicken the pulse quite like the cat presenting with open-mouth breathing, or the dog who arrives at the door tinged an unsettling shade of blue. Such moments test even the most seasoned clinician. Stress, as we know, is no friend to clear thinking. What we require—calmly and methodically—is a structured approach.

In this session, delegates are guided through a practical and systematic framework for approaching respiratory emergencies. The emphasis will be on accurate localisation of the problem, employing both fundamental clinical skills and more advanced diagnostic techniques. Do bring your eyes and ears—both shall be put to proper use in what promises to be an interactive and thoroughly practical discussion.

From Wheeze to Wisdom — Building Clinical Confidence

Delegates will leave better equipped to manage both acute and chronic respiratory conditions in dogs and cats. Through a series of carefully selected clinical cases, we shall explore everyday challenges that often conceal subtle complexities.

We will discuss, for instance, the less favourable consequences of chronic salbutamol use in certain feline patients, as well as surprisingly effective ways of employing household devices to improve the quality of life of the asthmatic cat. Nebulisation can be tremendously beneficial—provided it is done correctly. There are, as always, do’s and don’ts. And as for placing a patient in a steamy

bathroom? I am afraid the answer is usually disappointing. The particles generated are simply too large to penetrate to the bronchioles and alveoli, where they are truly needed.

Of Antibiotics and Alveoli

We shall also examine antibiotic selection in cases of pneumonia. Many clinicians reach instinctively for amoxycillin-clavulanic acid. A fair choice in some instances, certainly—but how effectively does it penetrate the alveolar space? Are superior alternatives available? In many situations, yes.

Whilst fluoroquinolones may offer admirable penetration, we must balance efficacy with stewardship. Agents less likely to drive antimicrobial resistance and safer in cats are often preferable. Clindamycin, for example, may serve us well in appropriate cases. Thoughtful prescribing remains a cornerstone of responsible practice.

The Stethoscope — Not Just for the Chest

We are all well accustomed to auscultating the thorax. But how often do we pause to place the stethoscope over the upper airway? A simple shift to the transtracheal region can reveal airflow disturbances that might otherwise be misattributed to the lower airway. One ignores this at one’s peril.

The Curious Case of the Shower Curtain

What, you might ask, has a shower curtain to do with respiratory medicine? Quite a great deal, in fact.

The so-called “shower curtain effect” is most commonly explained by Bernoulli's principle. In essence, as the velocity of airflow increases, pressure decreases. Just as a shower curtain is drawn inward when the shower is switched on (to the mild annoyance of the bather), so too may an airway collapse inward when air moves rapidly past a fixed or dynamic obstruction.

In upper airway disease, forced inspiration tends to exacerbate collapse, leading to dyspnoea and characteristic respiratory noise. The effect is most pronounced when the airway lumen is already narrow—whether through pathology or

inherent conformation. An understanding of this principle assists greatly in localising disease.

Mastering the Broncho-Alveolar Lavage

The lecture will maintain a decidedly practical flavour. Delegates should leave feeling confident in performing broncho-alveolar lavage (BAL) safely and effectively.

We shall discuss ideal fluid volumes and how to recognise a satisfactory sample. In human medicine, hundreds of millilitres may be instilled without consequence. In our patients, considerably smaller volumes suffice. A useful guideline is approximately 2 mL/kg of sterile saline. A sample that appears frothy upon agitation is reassuring—it suggests surfactant is present and that material has indeed been obtained from the lower airway.

Cytology remains the single most informative test for BAL fluid. Routine culture may yield misleading positives. PCR testing for Mycoplasma and Bordetella may be indicated, and geographical considerations should guide further testing. In feline patients, Mycobacterium may be both underdiagnosed and increasingly prevalent.

The Art of Observation

A series of videos will hone delegates' observational skills. Respiratory medicine begins, as ever, with careful looking and listening.

Is the difficulty predominantly inspiratory? One should then suspect the upper airway. Is it expiratory? The lower airway is the more likely culprit. Such distinctions, while seemingly simple, are profoundly informative.

Oxygen, Air, and the Importance of Calm

In the acute patient, oxygen supplementation is paramount. In chronic cases, clean, well-filtered room air can make a significant difference.

When an oxygen cage is unavailable, nasal prongs or nasotracheal oxygen delivery can be highly effective. Above all, we must minimise stress in the dyspnoeic patient; undue handling may tip a precarious balance.

Modern air-quality monitoring and filtration systems may further support our chronic respiratory cases. Small refinements in management often yield substantial benefits.

In respiratory medicine, it is rarely one dramatic intervention that transforms a case, but rather the careful accumulation of small, thoughtful steps. Master these, and one will serve one’s breathless patients exceedingly well.

Feline Infectious Peritonitis: Diagnostic Challenges and Modern Antiviral Therapy

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Feline infectious peritonitis (FIP) is a severe systemic disease of cats caused by a mutated form of feline coronavirus (FCoV). FCoV infection is extremely common in domestic cats, particularly in multicat environments such as shelters, catteries and rescue centres. In most animals the virus remains confined to the gastrointestinal tract and is shed in faeces with minimal clinical impact. However, in a small proportion of infected cats the virus mutates and acquires the ability to infect monocytes and macrophages, leading to the systemic inflammatory disease recognised as FIP.

Pathogenesis

The development of FIP involves both viral mutation and host immune responses. The mutated virus develops tropism for macrophages, enabling efficient intracellular replication and dissemination throughout the body. This triggers an intense immune-mediated inflammatory response characterised by vasculitis and immune complex deposition. These processes lead to vascular leakage, granulomatous lesions and effusion formation in body cavities. Although several viral genes have been implicated in this transformation, no single mutation consistently explains the conversion from feline enteric coronavirus (FECV) to the FIP-causing variant.

Host immune responses are a critical determinant of disease expression. A strong cell-mediated immune response may prevent clinical disease, whereas ineffective cellular immunity combined with a strong humoral response contributes to immune complex formation and systemic inflammation.

Epidemiology and Risk Factors

Several epidemiological risk factors have been identified. FIP most commonly affects young cats, particularly those under two years of age. Intact males appear to be overrepresented in clinical cases. Certain breeds, including Bengals and Birmans, may have increased susceptibility, suggesting a genetic predisposition.

Environmental factors are also important. Cats originating from breeding establishments, rescue centres or multicat households are exposed to higher levels of FCoV transmission. Stressful events such as surgery, gastrointestinal disease, vaccination, travel, or the introduction of new animals may precipitate clinical disease. Concurrent infections such as feline immunodeficiency virus (FIV) or feline leukaemia virus (FeLV) may increase susceptibility through immunosuppression.

Clinical Presentation

Two classical forms of FIP are recognised: effusive ("wet") and non-effusive ("dry").

The effusive form accounts for approximately 75–80% of cases and is characterised by accumulation of protein-rich effusions within the abdomen or thorax. Typical clinical signs include pyrexia, lethargy, anorexia, weight loss and progressive abdominal distension or respiratory distress.

The non-effusive form tends to follow a more chronic course and is associated with granulomatous inflammatory lesions in multiple organs. Clinical manifestations vary depending on the organs affected but frequently include neurological signs, ocular disease (particularly uveitis), lymphadenopathy and organ dysfunction. Many cats initially presenting with the dry form later develop effusions.

Diagnostic Challenges

Diagnosing FIP remains challenging because no single non-invasive test can definitively confirm or exclude the disease. Instead, diagnosis typically requires integration of clinical findings, laboratory results and specialised diagnostic tests.

Routine haematology and biochemistry frequently reveal non-specific abnormalities. Common findings include mild to moderate anaemia, lymphopenia, neutrophilia and monocytosis. Hyperglobulinaemia is frequently observed and may lead to hyperproteinaemia. Hypoalbuminaemia may occur, particularly in cats with effusions.

The albumin-to-globulin ratio (A:G ratio) can help assess the likelihood of FIP. Ratios below approximately 0.4 increase suspicion, whereas ratios

above 0.6 make FIP less likely, although they do not completely exclude the disease.

Diagnostic imaging is also useful. Abdominal or thoracic ultrasonography may reveal effusions, mesenteric lymphadenomegaly, or organ abnormalities consistent with granulomatous inflammation.

Effusion Analysis

When present, effusion analysis provides valuable diagnostic information. FIP effusions are typically:

- Yellow to straw-coloured
- Viscous and protein-rich
- Low in cellularity

The Rivalta test is a simple in-house test that may assist with interpretation of effusions. A negative Rivalta test strongly argues against FIP, whereas a positive result increases suspicion but requires confirmation through additional testing.

Specific Diagnostic Tests

More specific tests aim to detect the virus or viral antigens.

Immunohistochemistry and Immunocytochemistry

Detection of coronavirus antigen within macrophages in tissues or effusions using immunohistochemistry (IHC) or immunocytochemistry (ICC) provides strong evidence supporting a diagnosis of FIP. Tissue biopsy with IHC remains the diagnostic gold standard, although this approach is invasive and not always feasible in unstable patients.

Molecular Diagnostics

Reverse transcription polymerase chain reaction (RT-PCR) assays are widely used to detect viral RNA in blood, effusions, tissues or cerebrospinal fluid. However, interpretation can be challenging because non-mutated enteric coronavirus may also be detected in some samples.

Quantitative PCR approaches are increasingly used because viral loads tend to be substantially higher in cats with FIP compared with those with benign enteric coronavirus infection. Nevertheless, PCR results should always be interpreted in conjunction with clinical findings and other diagnostic tests.

Advances in Treatment: Antiviral Therapy

For decades FIP was considered uniformly fatal, and treatment options were largely palliative. Corticosteroids such as prednisolone were used to reduce inflammation and improve quality of life but had minimal impact on survival.

In recent years, however, the development of antiviral drugs has dramatically changed the prognosis of FIP.

Nucleoside Analogues

The nucleoside analogue GS-441524 and its prodrug remdesivir inhibit viral RNA-dependent RNA polymerase and prevent viral replication. These drugs represent the cornerstone of modern FIP therapy.

Clinical studies have demonstrated high survival rates with antiviral treatment. In retrospective and prospective clinical studies, treatment with GS-441524 or remdesivir has been associated with survival rates exceeding 80%, with many cats achieving long-term remission.

A systematic review including more than 650 treated cats reported an overall treatment success rate of approximately 84.6%, with higher success rates observed in uncomplicated cases.

Large retrospective studies have also demonstrated that legally sourced remdesivir and GS-441524 protocols are highly effective in treating naturally occurring FIP in clinical practice.

Current Treatment Protocols

Guidelines from the International Society of Feline Medicine (ISFM) and International Cat Care recommend antiviral treatment courses of approximately 12 weeks (84 days).

Table 1. Antiviral treatment protocols for FIP (adapted from ISFM / International Cat Care guidance)

Clinical presentation	GS-441524 (oral)	Remdesivir (IV or SC)
Effusive FIP without ocular or neurological signs	15 mg/kg q24h (or 7.5 mg/kg q12h)	10–15 mg/kg q24h
Non-effusive FIP without ocular or neurological signs	15 mg/kg q24h (or divided q12h)	12–15 mg/kg q24h
Ocular FIP	15–20 mg/kg q24h (or divided q12h)	15 mg/kg q24h
Neurological FIP	10 mg/kg q12h	20 mg/kg q24h

Remdesivir may be administered intravenously or subcutaneously during the initial stages of treatment, particularly in severely ill cats that are unable to tolerate oral medication. Once clinical improvement occurs, therapy is commonly transitioned to oral GS-441524 for the remainder of the treatment course.

Alternative and Emerging Antivirals

Additional antivirals are being explored for FIP treatment.

Molnupiravir (EIDD-1931), another nucleoside analogue, has demonstrated comparable efficacy to GS-441524 in some studies and may represent an alternative treatment option in certain cases.

Protease inhibitors such as GC376 were among the earliest antivirals studied for FIP and showed promising results in experimental studies, although nucleoside analogues are currently the preferred treatment in most clinical protocols.

Monitoring During Treatment

Monitoring is an essential component of successful therapy. Clinical improvement is typically observed within the first week of treatment, including improved appetite, increased activity and reduction in fever.

Laboratory parameters such as globulin concentrations, bilirubin levels and haematological abnormalities generally improve within several weeks of initiating treatment.

ISFM guidelines recommend regular monitoring with haematology and biochemistry every two to four weeks during treatment. Therapy should generally continue for at least 84 days and should not be discontinued until both clinical signs and laboratory abnormalities have resolved.

Table 2. Monitoring and treatment considerations during antiviral therapy

Aspect	Recommendations
Treatment duration	Minimum 84 days (12 weeks) in most protocols
Monitoring	Clinical exam, CBC and biochemistry every 2–4 weeks
Response to treatment	Improved appetite, reduced fever and activity increase typically within days
Dose adjustment	Adjust dose according to weight gain and clinical response
Relapse management	Restart antiviral therapy at higher dosage if relapse occurs

Conclusion

Feline infectious peritonitis remains one of the most complex infectious diseases in feline medicine due to its multifactorial pathogenesis and diagnostic challenges. Historically considered uniformly fatal, FIP is now a treatable disease thanks to the development of antiviral drugs such as remdesivir and GS-441524.

Accurate diagnosis requires careful integration of clinical findings, laboratory data and specialised diagnostic tests. Advances in molecular diagnostics and antiviral therapy have significantly improved outcomes, with survival rates exceeding 85–90% when appropriate treatment protocols are used.

Continued research into viral pathogenesis, improved diagnostic tools and optimised antiviral protocols will further enhance our ability to diagnose, treat and ultimately prevent this devastating disease.

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Managing diabetes in cats without insulin injections

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The introduction of SGLT2-inhibitors has been nothing short of a revolution in the management of feline diabetes mellitus. Treatment of diabetes mellitus is now possible without the constant threat of hypoglycaemia and thus the need of lifelong glucose monitoring. Treatment is also possible for those caregivers who cannot deal with the daily necessity of insulin injection therapy.

Two years after the official introduction of the SGLT2-inhibitor velagliflozin (Senvelgo) on the UK and European market and more than 10 years after the initial clinical trials with SGLT2 inhibitors plenty of new lessons have been learned that go beyond the official guidelines that were designed to introduce this category of drugs for the first time into the veterinary landscape. As is often the case (and should be the case), at the moment of introduction, safety was the primary focus in setting up guidelines for usage. Clinicians had only been used to using insulin to treat feline diabetes mellitus and were now confronted with an entirely new modality of treatment without insulin. These guidelines have served their purpose well, resulting in minimal side effects in the many thousands of cats with diabetes mellitus treated thus far.

Nonetheless, lessons have been learned, and this lecture will focus on those lessons, which therefore go beyond the initial guidelines provided with the introduction of this drug category. Some of the lessons described during this lecture therefore also go beyond the official label that is associated with market authorisation obtained earlier. It is important though that we learn from the experiences thus far and follow the science to ensure we make full use of the SGLT2i when they can be used and avoid them when they should not be used.

In short, this lecture will deal with all the issues surrounding SGLT2is that the drug companies will not tell you. Example issues (more will be discussed during the lecture) are detailed below.

Diabetes mellitus is expensive already - how far should I go to establish that a diabetic cat is a good candidate for SGLT2-inhibitors?

Short answer: not far! A good history and physical examination will help establish a low likelihood of relevant disease in most cases. We need to ensure there are no additional signs than those of diabetes mellitus and the signalment fits that of a diabetic cat. If that's the case, it has to be an option that for a cash-strapped caregiver a diagnosis of diabetes mellitus using international ALIVE criteria (www.esve.org) is sufficient to allow treatment to start. When more budget is available routine biochemistry and haematology can screen for other diseases that could be associated with the same clinical signs as diabetes mellitus does, including weight loss.

When other clinical signs than pu/pd, polyphagia and weight loss are mentioned during the history taking, that, however, should trigger a search for explanations, especially if it involves inappetence.

This author explicitly avoids using screening tests for pancreatitis in cats (and dogs) without clinical signs, since their positive predictive value (is there actual disease or is this a false positive) is actually very low. SpecPLI and other pancreatitis indicators are routinely elevated in many diabetic patients without clinical relevance. The same is true for abdominal ultrasounds in the clinically happy diabetic cat.

My patient on SGLT2i is happy but ketones have appeared in the urine – do I stop treatment immediately?

Short, but firm answer: no!

Although the initial guidelines and the label still state this (and the author has chaired the writing of those guidelines, guilty as charged!), most, and including this author, are content with continuing SGLT2i treatment regardless of ketonuria (of any magnitude) or hyperketonaemia (of any magnitude). Experience and currently ongoing studies tell us that this is seemingly a normal physiological phenomenon in cats in general, diabetic cats and diabetic cats on insulin or SGLT2i treatment. Presence of ketones is not predictive of impending DKA and does not constitute a diagnosis of DKA in a otherwise clinically happy cat. The international criteria for diagnosis of DKA in fact dictate that pH of blood is low, or, if pH measurement is not available, that the clinical picture indicates a low pH. This means that any cat with DKA will be ill. A happy cat

with just diabetic signs but NO appetite issues, lethargy, acute vomiting, etc., never has DKA. The process of monitoring for ketones during SGLT2i treatment could therefore be questioned. This author still does it since it is a tool to keep talking to the caregivers and when ketonuria or hyperketonaemia appears, it forms almost an excuse to get the owner to call the clinic, confirm that the clinical signs are fine, and confirm which clinical signs we would be concerned about in this context and would be a reason to seek immediate help. Once again though, the SGLT2i would not be stopped in the clinically happy ketonuric/hyperketonaemic cat. It will still be useful to discuss this with the client, specifically mentioning that the official label till dictated the stopping of the drug in these situations and switching to insulin. Unfortunately, here, there is currently therefore a mismatch between the regulatory systems way of operating and difficulties to easily update labels, and the scientific reality of medicine, which has changed.

SGLT2-inhibitors are only labelled for type 2 diabetes mellitus in cats – what about a cat with hypersomatotropism/acromegaly, Cushing's, iatrogenic steroid induced diabetes mellitus and other forms of diabetes?

Short answer: for some other types of diabetes, we can use these drugs.

There is now sufficient evidence to say that SGLT2i work well to control the glycaemia in cats with diabetes mellitus associated with hypersomatotropism/acromegaly (a condition affecting 1 in 4 cats with diabetes mellitus without necessarily any phenotypic clues that this condition is present). It should be remembered that it would not lower excess growth hormone and IGF-1 levels, nor reduce pituitary hyperplasia or adenoma growth. For this, we would need to consider medical (cabergoline, pasireotide), surgical (hypophysectomy) or, though less effective, radiotherapy.

Case studies have also shown the effective use in Cushing's syndrome and cats with diabetes mellitus induced by high dose steroids (e.g. used for treating IMHA). For this reason, this drug type can most certainly be used in a range of scenarios where insulin resistance predominates the aetiology of the diabetes mellitus.

The latter also means that, likely, the one type of feline DM that is not suitable to monotherapy with SGLT2i, is a type akin to type 1 DM, as seen in dogs or due to destruction of the pancreas through other means (e.g. tumour, surgery).

What have we learned about the DKA dangers of SGLT2-inhibitors – should I remain scared about not giving insulin?

Short answer: no.

The DKA rates remain similar to the field studies used to acquire market authorisation with now more than 100,000 animals having been treated with one of the authorised SGLT2i types worldwide. Simply put: 1 in 15 diabetic cats with a new diagnosis of DM will suffer from DKA after start of SGLT2i and 1 in 10 of diabetic cats which were previously on insulin treatment. Those are reassuringly low rates, maybe even comparable to the rates we see under insulin treatment worldwide.

Nonetheless, DKA remains the one complication we are most focussed on once a SGLT2i has been started and especially when a cat on treatment becomes clinically unwell. Although we cannot predict DKA to happen, we do have increased chances when we start SGLT2i in an unwell cat. This is why we prefer that these cats are started on insulin treatment. Once cats have cleared whatever co-morbodity there was and are well again, switch to SGLT2i could be contemplated (contrary to the label recommendations, therefore).

We have also learned that when DKA does happen we need to be aware of two key differences compared to the DKA patient which has been on no treatment or on insulin treatment. Firstly, the DKA patient on SGLT2i might present with a normal glucose, so called euglycaemic DKA. Treatment with fluids and insulin are indicated, but, since these cats can have a blood glucose already lower than 14 mmol/L, concurrent dextrose or glucose should be provided from the get-go. Secondly, if a patient has previously been on SGLT2i, we have noticed that a subpopulation will need a longer hospitalisation time, presumably due to the ongoing and longer-lasting effects of the SGLT2i, which can result in hypoglycaemic episodes for days after stopping the SGLT2i, when insulin has been started concurrently. More erratic electrolyte abnormalities have also been reported with DKA after SGLT2i treatment. Ideally therefore (though similar to the recommendation with classic DKA under insulin treatment), these cases are dealt with in an ICU setting with appropriate ongoing monitoring for a sufficiently long time to assure ourselves that the metabolism has truly stabilised.

Can we use SGLT2i in dogs?

Short answer: yes, but with insulin if it concerns the most common type of diabetes mellitus in dogs.

Although this would also mean using these drugs off-label, there is increasing experience with SGLT2i use in canine diabetes mellitus, both to prevent diabetes mellitus due to insulin resistance (e.g. due to Cushing's syndrome or iatrogenic steroids, due to dioestrus induced diabetes mellitus) and to treat diabetes mellitus alongside insulin. Dogs seem more sensitive to SGLT2i and therefore dosing needs to be adjusted to avoid excessive stool abnormalities. Research is still fresh but likely coming forward with more concrete recommendations soon.

Final remarks

More issues will be discussed during the lecture and questions can be asked during and after the session. Bottomline remains, however, that these drugs have shown to be very safe, with good patient selection (the happy cat), DKA scenarios are rare and do not justify excluding clients from the benefit of starting SGLT2i instead of insulin in the cat.

If our task as veterinary teams is to seek to optimise the human-animal bond despite illness, then this author cannot think of a better reason to consider SGLT2i to be used as a preferential treatment over insulin whenever possible in the diabetic cat.

Hyperthyroidism in cats: not always skinny and anxious

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INTRODUCTION

Hyperthyroidism is one of the most common, if not, THE most common feline endocrinopathy. By now, we all know what the main presenting signs are, namely: weight loss despite polyphagia, tachycardia, anxiousness, palpable thyroid nodule, etc. Nevertheless, if we are prepared to screen for the disease earlier, we will see more variety in case presentations. This lecture will discuss these cases, as well as how best to deal with them.

THYROTOXICOSIS

Thyrotoxicosis represents the toxic effect of having overall increased exposure of body tissue to thyroid hormone activity.

Thyrotoxicosis affects multiple organ systems and the associated clinical signs range in severity from mild to severe and are quite variable from cat to cat. The fact that a cat is not (yet) displaying the above mentioned classical signs of hyperthyroidism, does therefore not mean that the cat's body is healthy; the opposite is true, cells and tissue are suffering.

MYOCARDIAL EFFECTS

Each cat displaying an image of hypertrophy of the myocardium should be subjected to thyroid testing. When these cats present with congestive heart failure, the diagnosis of hyperthyroidism could be hampered by sick euthyroid syndrome-like phenomena. This could lead to the documentation of a normal Total T4 value. The lecture will discuss the value of alternative testing methods in such scenarios, including free T4, T3 and TSH.

RENAL EFFECTS

The effects of excess thyroid hormone on the kidneys has been described reasonably well in the veterinary literature where it comes to increasing GFR; in fact, this line of thought has caused clinicians to be fearful of treating hyperthyroidism when there is suspicion of renal disease. Nevertheless, this knowledge should be balanced with the knowledge from human medicine that shows that excess T4 also negatively impact on renal health. Therefore, especially in the hyperthyroid cat with no or mild renal disease, early intervention and resolution of the hyperthyroid state is indicated. Even with renal disease, treatment of hyperthyroidism will only usually impact on longevity if we are dealing with advanced renal disease (IRIS stage 3 and more).

THYROID STORM

Thyroid storm is a syndrome described in human medicine to define a multi systemic disorder resulting from acute onset organ exposure to excessive levels of thyroid hormone. It can be life-threatening and is a significant cause of mortality in human medicine. With feline cases of hyperthyroidism being diagnosed earlier and earlier, clinicians could be caught out by cats presenting in such acute state, despite not fulfilling the classical criteria of the hyperthyroid cat in the first instance (skinny cat).

Activation of the sympathetic nervous system has been implicated in the onset of thyroid storm. Many of the clinical signs and physiological symptoms seen in thyroid storm are similar to those seen during catecholamine excess. Beta-blocking forms part of the treatment protocols.

Whether this disease entity truly exists in veterinary medicine is actually still debated; in the author's clinical a handful of cases would have qualified to fall within the definition of the syndrome, some occurring after treatment of the hyperthyroid state through surgery or radioactive iodine.

HYPERTENSION

Hypertension is frequently encountered in the hyperthyroid cat; vice versa, finding hypertension should elicit consideration of testing for presence of hyperthyroidism in the middle aged to older cat. Whether there is truly a causal relationship is still up for debate.

JUVENILE HYPERTHYROIDISM

Another atypical presentation involved exceptionally young cats with hyperthyroidism. Anecdotal, as well as recorded cases exist in veterinary medicine. Among them an 8-month-old, male domestic shorthaired cat presented for chronic weight loss, intermittent dyspnoea, chronic diarrhoea, hyperactivity, and weakness. The cat had a palpable thyroid nodule and increased serum total thyroxine and 3,5,3' triiodothyronine levels. The cat had a unilateral thyroidectomy performed followed by radioactive iodine at a later date. The clinical signs resolved following radioactive iodine, but the cat subsequently developed clinical hypothyroidism.

Small Animal Pulmonary Patterns: Parts I and II

Michael Q. Bailey DVM, Dip ACVR, Fellow AAAS

Radiographs are not very specific because most pathologies involve fluid and soft tissue, making it difficult to distinguish among possible pathologies. Remember that the radiograph's specificity limits the differential diagnosis.

Principles of Pulmonary Changes

* Biology
    * Age
    * Species
    * Size
* Opacity of the Lung
    * Decreased density.
    * Normal density
    * Increased density
* Pulmonary radiographic Patterns
    * Interstitial
    * Bronchial
    * Alveolar
    * Vascular
* Macroscopic distribution of Altered Opacity
    * Craniovental
    * Dorsocaudal
    * Diffuse
    * Lobar
    * focal

Anesthetic Lung

Volume of gas, Atelectasis, pressure Incomplete Expansion, Inflammation, etc. Unilateral hyperinflation may be secondary to bronchial obstruction, atelectasis, and bronchial dilation in the right lung.

Pulmonary hyperinflation.

Usually defined as **an abnormal increase in functional residual capacity**, i.e., lung volume at the end of tidal expiration. As such, it is virtually universal in patients with symptomatic diffuse airway obstruction.

Small Animal Pulmonary Patterns: Parts I and II

Michael Q. Bailey DVM, Dip ACVR, Fellow AAAS

Hypovolemia, also known as **volume depletion** or **volume contraction**, is a state of abnormally low extracellular fluid in the body.

Pneumothorax is an abnormal collection of air in the pleural space between the lung and the chest wall.

Reasons for Decreased Thoracic Density

Over-Exposure: This has changed with digital radiographs.
Over-Inflated Lung
Hypo-Volemic Lung
Free Gas Pleural Space

Focal Loss of lobar volume

Signs of incomplete lung expansion

- Decreased lung size.
- Increased opacity
- Lobar sign
- Crowding of ribs
- Air bronchogram sign
- Positive silhouette sign
- Poorly defined margins of vessels
- Mediastinal shift (toward collapse)
- Crowding and reorientation of pulmonary blood vessels
- Compensatory hyperinflation
- Bronchial rearrangement
- Cardiac rotation
- Displacement of diaphragm
- Rounded pulmonary margins
- Displacement of pleural fissures
- Changed the location of abnormal structures.

Pulmonary patterns

- Alveolar

- Bronchial
- Interstitial
- Vascular
- **Mixed Pattern:** Pulmonary patterns are usually not solitary and are therefore often combined.

*Alveolar Pattern 3 Reason [1]

- Fluid
- Cells
- Lack of air
    - Air Bronchograms are a type of Alveolar pattern, not bronchial.

*Bronchial Pattern [2]

- Fibrosis
    a) Age
    b) Chronic disease
- Infiltrates
    c) Any Fluid
    d) Cells
    e) May originate from another site.

*Interstitial Pattern, Increased [3]

- Fibrosis
    f) Age [4]
    g) Chronic disease
- Infiltrates
    h) Any Fluid
    i) Cells

Interstitial Pattern. Decreased

- Radiographic Technique
- Hyperinflation
- Decreased Fluid Volume

*Vascular Pattern, Increased [5]

- Pre-capillary Hypertension
- Post-Capillary Hypertension
- Vasculitis
- Heartworm

Small Animal Pulmonary Patterns: Parts I and II

Michael Q. Bailey DVM, Dip ACVR, Fellow AAAS

The diameter of the CVC

Vascular Pattern, Decreased

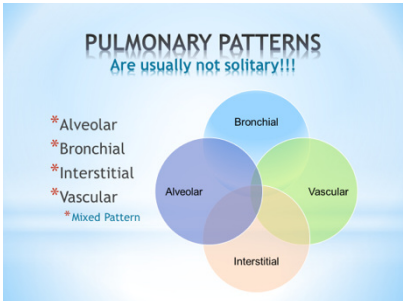
- Decreased Fluid Volume

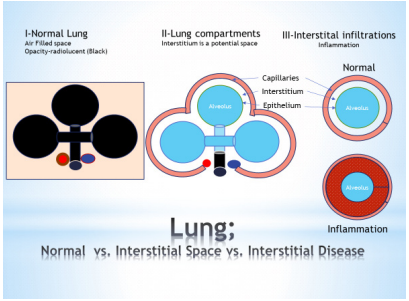
Consolidation

Consolidation results from replacing air in the alveoli with transudate, pus, blood, cells, or other substances. Pneumonia is by far the most common cause of consolidation.

Air space opacification [6, 7]

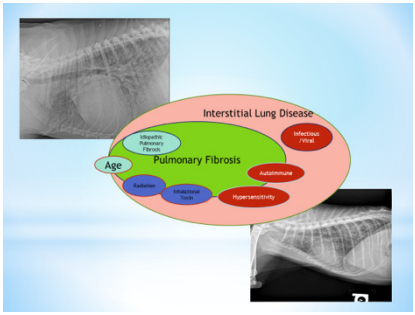
- This descriptive term refers to filling the pulmonary tree with material that attenuates X-rays more than the surrounding lung parenchyma. Air space opacification is equivalent to the pathological diagnosis of pulmonary consolidation.
- In radiological studies, air space opacification presents as increased attenuation of the lung parenchyma causing obscuration of pulmonary vessels, without significant loss of volume, in the segment(s) affected.





Small Animal Pulmonary Patterns: Parts I and II

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Pulmonary fibrosis is a descriptive term used when there is an excess of fibrotic tissue in the lung. It can occur in a wide range of clinical settings and can be precipitated by a multitude of causes.

Pneumonia

- Pneumonia is an accumulation of pus in the lungs, leading to increased lung density.
- It is possibly confused with blood, water, or even neoplasia; therefore, clinical history is necessary to help differentiate pneumonia from other possible pulmonary pathologies.
- The radiographic appearance depends on the type and location of pulmonary infiltrates:
  - Aspiration Pneumonia
  - Lobar Pneumonia
  - BronchioPneumonia
  - Interstitial/viral Pneumonia
  - Parasitic Pneumonia

Pulmonary Nodules

Cavitary

- The infection-causing pneumonia may lead to cavitation or destruction of the lung tissue, forming abscesses. The three most common organisms that cause pulmonary abscesses are staph aureus, strep pyogenes, and klebsiella. Gram-negatives are also prevalent causes of abscesses.

Pulmonary Nodules

- Focal Pulmonary Nodules can be benign, malignant primary, or metastatic nodules. Primary lung tumors are usually focal and locally invasive.

- Metastatic disease spreads hematogenously from another primary source, such as the thyroid or the abdomen, which, more than likely, presents as multiple nodules.
- Remember that plain film specificity is low; sarcoma can resemble other pathologies, such as Fungal pneumonia.
- Criteria for malignancy: the presence of calcification, size, margins not defined, growth, patient age/sex, and history.
- If the nodule is highly calcified, it is usually a benign process. They are typically a granuloma. However, osteosarcoma will ossify
- What should you do if you discover a nodule?

Bronchial Disease

- Increased Pattern
- Bronchial wall thickening
- Bronchial wall dilation Reversible vs Irreversible dilation
    - Tram or Railroad Tracks
    - Varicose
    - Sacculation or cystic

ASTHMA:

- The radiographic features of asthma are not specific. In the absence of other concurrent illnesses, the chest radiograph is almost always normal in patients with asthma.
- Possible findings are bronchial wall thickening and hyperinflation.
- Thoracic radiography may be normal, or abnormalities may be present, including peribronchial cuffing; alveolar or interstitial infiltrates; flattening of the diaphragm; air trapping or hyperlucency; and atelectasis of the right middle lung lobe.

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Cushing's syndrome – what do we need to prioritise to allow us to successfully manage these patients ?

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While Cushing's syndrome has been a well-recognised clinical problem in both dogs and cats for over a century, there continues to be debate regarding the best methods of diagnosing this condition and the optimum protocols for managing both the pituitary dependent and adrenal dependent forms of Cushing's syndrome. These notes will concentrate on some of the more recent issues pertaining to the two broad issues of confirming a diagnosis of Cushing's syndrome and the primary criteria that need to be considered in optimising management.

Confirming a diagnosis

It is important to remember that, as the name implies, this is a *clinical* syndrome created by a sub-acute exposure to additional glucocorticoid activity. The clinical syndrome can be quite variable because this can be a slowly developing problem. As a result, often the owners have not noticed how much their dog or cat has changed. Effectively the abnormalities evident at presentation have become the "new normal".

In 2026 the most common presenting findings are different between the dog and cat. In the dog the most common findings are polydipsia and polyuria, panting, epaxial muscle wasting and a tendency towards abdominal distention, subtle but palpable hepatomegaly and glabrous alopecia. In the cat the clinical picture is usually a poorly controlled and difficult to manage animal with diabetes mellitus.

While various abnormalities on routine clinical pathology can be expected in Cushingoid patients, confirmation of a diagnosis requires some form of dynamic testing of the pituitary adrenal axis. It is important to remember in attempting to confirm a diagnosis of spontaneous Cushing's syndrome, the urinary corticoid-creatinine ratio has a sensitivity of around 75% and the ACTH stimulation test a sensitivity of at best ~80%. If these tests are being used to try and confirm a diagnosis of Cushing's syndrome in a group of dogs reasonably likely to have the disease, a result within the reference range can have poor discriminatory capacity. As an example, when applied to a sample of dogs where the prevalence of Cushing's syndrome is around 60% an ACTH stimulation test is likely to be within the reference range in 25% of dogs who have the disease.

The higher sensitivity of the low dose dexamethasone suppression test (LDDST) makes it the test of choice in attempting to confirm a diagnosis of Cushing's

syndrome. The LDDST can also help differentiate pituitary-dependent Cushing's syndrome (PDHCS) from adrenal-dependent Cushing's syndrome (ADCS) as the latter will almost always be characterised by a lack of any meaningful suppression of endogenous serum cortisol levels at 4 and 8 hours after dexamethasone, whereas between 60-70% of PDCS will suppress at 4 hours but not at 8 hours.

In those Cushingoid patients who have no suppression at 4 or 8 hours, differentiating PDCS from ADCS is most reliably achieved by ultrasonographic examination which demonstrates roughly two normal or possibly enlarged adrenal glands in PDCS cases and in ADCS cases, an obvious difference in the size of the two adrenal glands with one gland clearly being *smaller* than normal.

Managing Pituitary Dependent Cushing's syndrome

There are effectively two broad choices for treating pituitary dependent Cushing's syndrome (PDCS), medical options or surgical options. The medical options involve the use of agents which either directly or indirectly produce adrenocorticolysis while the surgical options involve correcting the clinical problem by removing either the offending functional pituitary neoplasm or the principal target organs of the autonomously produced adrenocorticotrophic hormone (both adrenal cortices).

Medical treatment

While numerous agents have been proposed as effective in managing PDCS the only two commercially available agents that have proved adequately effective are trilostane and mitotane.

Trilostane

Trilostane is a synthetic, orally active steroid analogue that competitively inhibits 3β hydroxysteroid dehydrogenase and hence synthesis of several steroids, including cortisol and aldosterone.

In dogs peak trilostane concentrations are seen within 1.5 hours of dosing and decrease to baseline values in about 18 hours. Trilostane is variably absorbed after oral administration, at least partly due to its poor water solubility. **Absorption is enhanced by administering the drug with food and this practice should always be encouraged in the clinical setting.** The nadir serum cortisol concentration usually occurs around 3 hours after trilostane administration.

Trilostane's safety as well as both short and long term efficacy in controlling hyperadrenalism in dogs has been documented in numerous publications over the last 20 years. In essence the drug seemed to be generally effective in approximately 75-

80% of cases of PDCS although differences in opinions regarding how to optimise efficiency and minimise adverse effects has resulted in a rather broad range of dosing recommendations – both in terms of the daily dose and the dosing frequency.

Trilostane at various doses caused a significant reduction in both the mean basal and post-ACTH cortisol concentrations after 10 days of treatment in all four studies. In one study the post ACTH cortisol concentration decreased to less than 250 nmol/l within one month in 81% of dogs and in another 15% at some time whilst on treatment. These improvements were maintained in the study population for the duration of the trial. In the study targeting lower post ACTH cortisol values all dogs were well or acceptably controlled (post ACTH cortisol ≤ 75 nmol/l and ≤125 nmol/l respectively) although as mentioned previously, in some dogs these goals were only obtained by marked increases in the daily dose.

In general, trilostane is well tolerated. Approximately 10% of dogs receiving trilostane may develop mild side-effects including lethargy, depression, inappetence, vomiting and diarrhea which are usually self-limiting or corrected by adjusting the total dose or dose frequency.

One of the issues with trilostane has been the potential for the drug's effect to vary significantly between individuals, with as many as 20-25% of patients not responding satisfactorily. Additionally, the drug has the ability to modify not only the ability of the adrenal cortex to synthesize glucocorticoids but also to interfere with the bioactivity of whatever glucocorticoids are produced.

The current suggested starting dose for dogs with PDCS is 1-2 mg/kg once to twice daily given with food. As trilostane is supplied in 10, 30, 60 and 120 mg capsules, some mg/kg variation inevitably occurs and it is generally recommended to start on the lower end of the dose range. Increasingly twice daily dosing has been explored in an attempt to achieve adequate adrenal suppression. In 2026, in the author's opinion, twice daily dosing at a starting dose of 2mg/kg/12h is likely to be the most effective means of introducing trilostane to dogs with PDCS.

Interestingly, trilostane's mechanism of action in controlling canine Cushing's syndrome may be more complex than originally proposed. Increasing evidence suggests its effects maybe at least partly mediated through its capacity to modify other enzymes in the steroid synthetic pathway (including possible inhibition of 21-hydroxylase or 11α-hydroxylase) and/or up-regulation of one of the isoforms of 11-βhydroxysteroid-dehydrogenase resulting in enhanced conversion of cortisol to its inactive metabolite cortisone.

Effectively this means trilostane may also reduce the clinical signs of Cushing's syndrome by activating cortisol-deactivating enzymes in peripheral tissues, thereby

uncoupling to a variable extent the link between plasma cortisol concentrations and the biological effects of cortisol. As a result, some animals can seem to have greater clinical improvement than would be expected on the basis of their ACTH stimulation test results and also vice versa. In other words, some animals don't seem to be as well controlled clinically as would be predicted by their ACTH simulation test results, suggesting this "peripheral cortisol inactivating activity" may not always be equally expressed between individuals. This variation further supports the approach of individualising trilostane management for each patient and ensuring decisions are made on the basis of the clinical picture as well as the preferred laboratory data.

Effective monitoring of trilostane treatment remains a topic of some debate. In the author's opinion the most effective means of evaluating adequate control is to use a combination of objective criteria for improved clinical signs such as the CushQoL index together with achieving normalisation of routine clinical pathology indicative of glucocorticoid excess (normalisation of serum ALP and cholesterol, normalisation of a stress leukogram and an ACTH stimulation test with the ***post-ACTH*** serum cortisol levels in the lower 25th percentile of the reference range for ***basal*** cortisol used by the relevant laboratory.

Although trilostane is likely to be generally effective in approximately 75-80% of cases of PDCS what should we do with the approximately 25% of cases where it fails to produce a reasonable clinical response?

In these cases the attending clinician is faced with further attempted adjustments with trilostane dosing and formulations and/or considering using mitotane or surgical options.

Mitotane

The original method for managing PDCS medically was long term **mitotane** administration. The essence of this regime was to destroy most of the hyperplastic adrenal cortex by "chemical adrenalectomy". The remaining tissue provided normal plasma cortisol concentrations despite being subjected to almost supraphysiological levels of ACTH.

In other words the animal no longer had the capacity to raise its plasma cortisol level above 20-50 nmol/l. This magnitude of adrenal destruction is essential to achieve significant clinical improvement.

This "chemical adrenalectomy" can be achieved using two different protocols, one aimed at achieving complete and permanent adrenocorticolysis and the other at reducing adrenocortical activity to normal levels through partial adrenocorticolysis.

The second protocol has been generally accepted as the favoured method and involves administration of mitotane (25 mg/kg/12hr with food) for five to seven days. During this period water consumption, appetite and general demeanour are **CLOSELY** monitored. If any of these parameters change for more than 12 hours the mitotane therapy is stopped. Animals are evaluated with an ACTH response test 48 hours after the last dose of mitotane. Adrenal destruction is deemed satisfactory when both pre and post ACTH cortisols are similar and below the **MID-RANGE** for the laboratory's normal basal cortisol concentration. In approximately 15% of cases this may require a second or more courses of daily mitotane.

Once satisfactory adrenal destruction has been achieved it can be maintained by once or twice ***weekly*** mitotane administration (25 mg/kg/12hr with food on one or two days per week). Patients can be rechecked with an ACTH response test once every 8-12 weeks to ensure adequate control is being maintained.

Animals treated in this way cannot mount an appropriate stress response to trauma or illness. Consequently, from the start of mitotane therapy prednisolone should always be available and the owners instructed to administer 1-2 mg/kg orally in an emergency and continue every 24 hours until they manage to obtain input from their veterinarian.

The alternative protocol involves using mitotane on a daily basis for a longer period and attempting to bring about irreversible adrenocorticolysis and subsequently managing patients with glucocorticoid supplementation.

Both protocols have disadvantages. With complete chemical adrenalectomy approximately 30% of patients have sufficiently severe mitotane-induced adverse-effects to justify suspending treatment; while the reported relapse rate can be as high as 39%. With the more widely used method of creating and maintaining induction and remission by inducing a selective degree of adrenocorticolysis, the continuous requirement for ACTH stimulation tests along with similar relapse rates makes this regime complicated, intrusive and often expensive.

Surgical correction of PDH

As can be seen from the above, both trilostane and mitotane can be considered effective in correcting clinical signs and hormonal abnormalities in a proportion of cases with pituitary dependent Cushing's syndrome. Unfortunately, this means a *substantial* number of affected dogs will not respond to standard medical therapy and in these cases a practical alternative needs to be considered sooner rather than later. The potential frustrations of managing PDCS medically are further exacerbated by two factors. The first is the variable response of individual patients to both drugs. As a result, a great deal of time, effort and expense in modifying dosing regimes and

testing adrenal function can be required to achieve a satisfactory outcome. The second is the general concern around over-dosing with either agent and causing iatrogenic hypoadrenocorticism. Interestingly a recent study using the VetCompass data base of UK general practices demonstrated only around 15% of owners were satisfied with the management of their dogs' PDCS and approximately 90% of affected dogs had been euthanased with 18 months of starting treatment because of a lack of a significant response.

The combination of inconsistent response to a not inexpensive medical treatment and the tendency for it to be utilised in a way that would make under-dosing likely has meant alternative treatment regimes need to be entertained. Effectively these consist of two surgical options; removing the offending functional pituitary neoplasm or removing the principal target organ, the adrenal glands.

Hypophysectomy

Clinicians at the RVC and the University of Utrecht have had considerable success using hypophysectomy as a method of correcting PDH. However, this is a specialised surgical procedure and consequently is not widely available.

The principal advantage of this treatment modality is the effective removal of the underlying cause of the condition, the functional pituitary adenoma. Effective surgery generally minimises the likelihood of future problems relating to accelerated growth of the tumour and resultant clinical signs related to an expanding intracranial mass. While clearly an important consideration, any management decision needs to be moderated by the knowledge that the vast majority of pituitary tumours resulting in PDH do NOT result in clinical signs referable to an expanding intracranial mass lesion. Additionally, despite apparently complete removal of the adenohypophysis, relapses have been common.

Once hypophysectomy has been performed patients will need to be supported by long term medical management. In almost all cases this will consist of replacement therapy for thyroid, adrenocortical and vasopressin deficiencies produced as a result of the effective hypophysectomy. Generally this consists of standard replacement doses of 0.5mg/kg/24hr of cortisone acetate, 20ugm/kg/24hr of thyroxine and 1-2 drops of DDAVP eye drops every 24 hours.

While hypophysectomy is certainly a viable option for the management of PDH the various limitations associated with the procedure, its requirement for a specialist surgical and peri-operative team, the relatively high peri-operative complication rates and the disappointingly high rates of recurrence of clinical signs all suggest the need for further alternative management options. Despite apparently complete removal of

the adenohypophysis, in the past relapses have certainly been problematic. However, at least at the RVC, the increasing surgical experience the has resulted in a reduction in the peri-operative complications and it remains to be see whether this improved success rate will also translate to better long-term outcomes.

Bilateral adrenalectomy

Although this is not a new treatment, the availability of potentially effective and affordable medical options, together with the perceived difficulties in the post-operative management of the adrenalectomised patient, has probably resulted in the technique not achieving widespread acceptance as a feasible alternative to chemical adrenalectomy. However, the surgery itself is relatively simple and management of the surgically-induced "panhypoadrenocorticism" is relatively inexpensive and of course, uncomplicated by potential hyperadrenocorticoid relapses. In the author's opinion the reported difficulties with this procedure can be largely overcome by reducing the potential for perioperative glucocorticoid and mineralocorticoid deficiencies with a continuous hydrocortisone infusion at an initial rate of 0.5mg/kg/hr until the animal is taking food and water orally and then changing to a combination of cortisone acetate (0.5mg/kg/24h) with or without DOCP or fludrocortisone for long term replacement therapy.

In the author's experience this procedure has proved a practical alternative for the treatment of PDCS in the dog. Because of a general reluctance to try surgical correction when a medical alternative is available, until recently bilateral adrenalectomy has usually only been attempted in PDCS patients in which trilostane or mitotane, or both, have not been effective. However more recently the frustrations of managing PDH patients with trilostane and/or mitotane and, at least in the UK, the expense of these treatment regimens has meant in terms of efficacy, consistency and price, this form of surgical correction is becoming increasingly attractive. It is also important to highlight that at present there is no evidence that bilateral adrenalectomy increases the risk of future development of pituitary macroadenomas.

In both PDCS patients undergoing bilateral adrenalectomy and ADCS patients receiving unilateral adrenalectomy, it is imperative they receive appropriate peri and post-operative glucocorticoid replacement therapy. A relatively straight forward and reliable method for achieving this is provided in the following section.

Perioperative management:

Fluid therapy should be started one to two hours prior to surgery or as soon as the animal is anesthetised. Fluids should run at a rate of no more than "twice maintenance" levels (120 ml/kg/24 hours). Fluids alone of course will not be enough to combat the impending loss of functional glucocorticoid activity and this deficiency needs to be addressed. Currently, hydrocortisone sodium succinate (HSS) is the only

commercially available parenteral steroid with equipotent glucocorticoid and mineralocorticoid activity. Although soluble dexamethasone or prednisolone preparations can be used, their lack of mineralocorticoid activity makes them less attractive alternatives to HSS. Just as importantly, in these cases using dexamethasone and prednisolone it can be very difficult not to provide too much glucocorticoid replacement, something to be avoided as any excess glucocorticoid in these already "hypercoagulable" patients is likely to increase the risk of thromboembolism and its disastrous consequences.

Hydrocortisone sodium succinate is the succinate ester of hydrocortisone or cortisol, the principal steroid produced by the adrenal cortex in dogs. It has equipotent glucocorticoid and mineralocorticoid activity. However, it has only 25% of the glucocorticoid potency of prednisolone and less than 1% of the mineralocorticoid potency of fludrocortisone. At the recommended doses it provides sufficient glucocorticoid and mineralocorticoid activity to treat the clinical consequences of primary hypoadrenocorticism and consequently can be effectively used in the peri-operative management of these hyperadrenocorticoid patients undergoing removal of their hyper-functional adrenal cortex (ADH) or adrenal cortices (PDH).

Hydrocortisone sodium succinate is administered as an intravenous infusion at a dose of 0.5mg/kg/h until the dog is eating and drinking normally when it can be changed to oral steroid supplementation. In a dog with clinically significant hyperadrenocorticism this dose is likely to produce plasma cortisol concentrations of approximately 600 nmol/L within 2 to 3 hours. Such a cortisol concentration is likely to provide adequate glucocorticoid and mineralocorticoid replacement in stressed dogs with impaired adrenocortical function.

As there is potential for HSS to adhere to plastic or glass at low concentrations it is best to administer it in its own fluid bag made up to a concentration of 1mg/mL. It is incompatible with a variety of different solutions including ampicillin sodium and it is therefore best to dilute the HSS in normal saline.

Maintenance therapy:

Once the patient is stabilised, glucocorticoid and mineralocorticoid replacement therapy almost always needs to be maintained for the remainder of the animal's life. Traditionally, a semi-selective mineralocorticoid, fludrocortisone and a semi-selective glucocorticoid (cortisone acetate or prednisolone) are initially used together. Because fludrocortisone has approximately 15 times the glucocorticoid potency of cortisone in a proportion of patients the glucocorticoid can be discontinued and the patient maintained on fludrocortisone alone.

With the recent registration in the UK of the mineralocorticoid DOCP for the treatment of hypoadrenocorticism in dogs (Zycortal®), the "cascade regulations" demand this product be used as a first choice agent for managing hypoadrenocorticoid patients. It is also important to remember that DOCP is reported to have *no* glucocorticoid potency and thus typical hypoadrenocorticoid patients should receive *both* DOCP *and* a glucocorticoid. However, in the author's opinion the evidence supporting DOCP's lack of glucocorticoid activity is not in anyway robust and many animals are at risk of developing varying degrees of hyperadrenocorticism if managed with the recommended dose rate, especially if they are also given a glucocorticoid, again as recommended by the manufacturer.

Some of the pertinent features of the most effective drugs used to maintain hypoadrenocorticism patients are given below.

Deoxycorticosterone pivalate*:* is a potent mineralocorticoid analogue. As it has neither 11 β nor a 17β hydroxylation, it has traditionally been reported as having little if any glucocorticoid activity. However the evidence supporting this is not clear cut and there are at least two reports in the literature clearly indicating a proportion of hypoadrenocorticoid dogs can be maintained on DOCP alone. Additionally almost anybody who has used DOCP recognises the drug has underlying glucocorticoid action.

While the pivalate ester means the preparation will be relatively long-acting. Currently it has a registered recommended dose rate of 2.2 mg/kg administered subcutaneously every 25 days. Plasma electrolyte, urea and creatinine concentrations may be monitored fortnightly for the first month to determine the duration of action and help individualize the dose. Once stabilised it is prudent to check electrolytes every 4 to 6 months.
In the author's experience most dogs are well controlled on 1.0 to 1.5 mg/kg administered every 4 to 6 weeks although, occasional individuals will require more frequent dosing.

As the registration material suggests DOCP has no glucocorticoid activity it is prudent to start patients on concurrent glucocorticoid supplementation with cortisone acetate or, if the dog is over 20kg, prednisolone. The need for glucocorticoid supplementation and its relative amount can then be determined by gradual dose adjustments over the coming months.

Fludrocortisone acetate: Fludrocortisone acetate is a synthetic adrenocortical steroid with a fluoride ion substituted at the 9α position and an 11 β-hydroxyl group giving it both glucocorticoid and mineralocorticoid potency. Because of the 9□ fluoride substitution, fludrocortisone has potent mineralocorticoid activity while the 11-

hydroxylation confers significant glucocorticoid activity. By comparison, fludrocortisone has 10 times the glucocorticoid activity and 125 times the mineralocorticoid activity of cortisol.

The dose of fludrocortisone is 10 to 30 μg/kg administered orally once daily. Typically a lower dose is used initially with subsequent titration based on clinical impression and plasma electrolyte concentrations. Dose adjustments are usually made after weekly electrolyte evaluations. Once these are stable and within the normal range adjustments can be made every 3 to 4 months.

In patients receiving concurrent long-term fludrocortisone and prednisolone, it is not uncommon for the dose of fludrocortisone to gradually increase, as there appears to be a reduction in its mineralocorticoid efficacy over time. Although many possible mechanisms for this exist, it is tempting to speculate that this represents accelerated metabolism of both steroids in response to the chronic "supraphysiological" state created by long-term daily prednisolone administration. In the author's opinion, this "fludrocortisone creep" is not a feature of patients treated with concurrent cortisone acetate and fludrocortisone. In such treated patients, the fludrocortisone dose required to maintain normal electrolyte levels is generally at the lower end of the suggested dose range.

Cortisone acetate: Cortisone is a synthetic steroid with an 11-keto substitution. Once absorbed, it is rapidly activated to hydrocortisone by a distinct 11-β-hydroxysteroid dehydrogenase operating in a reductive mode. As cortisone is rapidly 11-hydroxylated to cortisol, it provides a complete replacement for any form of cortisol deficiency. As it has equipotent glucocorticoid and mineralocorticoid activity it will also provide more mineralocorticoid activity than other synthetic glucocorticoids such as prednisolone. In addition, its shorter half-life and lower overall activity means it may be less likely to create iatrogenic hyperadrenocorticism with long-term administration.
In patients with hypoadrenocorticism, orally administered cortisone acetate can be used as an effective long-term cortisol replacement. The dose of cortisone acetate must be individualized according to the severity of the condition, the response obtained and what other glucocorticoid or mineralocorticoid is being concurrently administered. In the changeover period as animals recover from an acute crisis, start eating and drinking and are changed from parenteral to oral medication most hypoadrenocorticoid dogs are started on a dose of 0.5-1.0mg/kg/12-24hr. However once they are stable, generally a dose of 0.5mg/kg/12-24 hr provides adequate additional glucocorticoid supplementation.

Prednisolone: Prednisolone is a synthetic adrenal steroid with moderately potentiated glucocorticoid activity (approximately 5 times that of hydrocortisone) and less than

10% of hydrocortisone's mineralocorticoid activity. Some clinicians advocate its use as a glucocorticoid supplement in the long-term management of hypoadrenocorticism at a dose rate of between 0.2- 0.5 mg/kg/24hr. ***The generally accepted level of endogenous glucocorticoid activity needed for maintenance in a normal dog suggests the above is a significantly excessive dose. If prednisolone is to be used to facilitate 'maintenance glucocorticoid activity' it should be at a dose rate of no more than 0.05 to 0.1 mg/kg/24hr.*** In the author's opinion cortisone acetate is a more effective alternative, especially in patients under 20kg in body weight.

Canine diabetes mellitus – what's new in management considerations in 2026

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Diabetes mellitus is not a single disease but a manifestation of various pathophysiological processes. Diabetes mellitus is perhaps best defined as clinically significant glucose intolerance caused by an absolute or relative lack of insulin. In any individual patient, the degree of insulin deficiency can fluctuate over time. These changes may be influenced by many factors. For example, any animal with pancreatitis may develop clinically significant impaired insulin secretion. This is generally reversible and resolves as the inflammation subsides.

Additionally, any condition causing chronic insulin resistance will result in a normal adaptive response of increased insulin secretion. In cats but *not* in dogs, over time this upregulation of insulin secretion may lead to so called "islet exhaustion" in susceptible individuals. This may be variably reversible depending on the length of time the islets have been exposed to this increased secretory demand and the presence of inherent individual susceptibility to the damaging effects of chronic insulin "hypersecretion".

Recent developments in managing canine and feline diabetes mellitus

Over the last five years a number of important and impactful developments in managing canine and feline diabetes mellitus have emerged and it is time to catch up to ensure we maximise the health outcomes of our patients.

Before we talk about a couple of specific developments, it is perhaps important to recognise some general principles that increasingly dominate our management priorities. First, increasingly there is an emphasis on doing what we can to ensure a reasonable quality of life for the owners of diabetic cats and dogs as well as the animals themselves. This goal requires our management strategies to be less formulaic and protocol driven; relying on applying basic principles around addressing the priorities of different owners while at the same time ensuring controls minimises the diabetic clinical syndrome.

Second but relatedly, management of diabetic cats should be focusing on achieving clinical control without the use of insulin; while management of diabetic dogs is increasingly

focused on achieving clinical control ***and*** reducing, whenever possible, the rate and severity of cataract development.

Clearly whenever underlying explanations for insulin resistance can be identified it is important, whenever practicable, to correct these as this may well result in a more easily managed diabetic patient and in, at least in cats, the patient not requiring insulin.

Some of the specific mechanisms by which these priorities are most likely to be achieved are discussed briefly below.

SGLT-inhibitors, super-longer acting insulins and extreme weight loss diets (CATS)

For years the basic components of pet diabetes mellitus management have been twice daily insulin injections and managed dietary intake.

While there is no doubt these principles remain important, with the introduction of new classes of anti-diabetes drugs, things have changed significantly.

One drug, a sodium glucose co-transporter 2 inhibitor (SGLT-2 inhibitor) called velagliflozin, is able to treat a majority of diabetic cats without the need for concurrent insulin. This oral, once daily, drug causes glucose excretion through the kidneys, resulting in rapid euglycaemia, stabilisation and improvement of clinical signs and beta-cell function.

A recent large field trial showed efficacy in virtually all naïve and most insulin pre-treated diabetic cats with a glycemic effect already noticeable within 7 days from the start of treatment. The main adverse effects included transient stool changes (soft stools or diarrhea). Expected (by some) high rates of urinary tract infections did not occur and clinical hypoglycemia was not noted. Diabetic ketoacidosis occurred in 1 in 15 newly diagnosed diabetic cats and 1 in 10 previously insulin treated cats. Use of the drug is therefore recommended in the “happy” diabetic cat (well hydrated, not cachectic, reliable appetite, active, no acute GI signs). In other scenarios its use should only be considered when insulin treatment is not possible and the owner and clinicians are prepared to accept the risk of so called “euglycemic-DKA”.

The use of SGLT2-inhibitors in dogs is currently not advocated outside of a research setting. Nonetheless, initial trials using insulin and velagliflozin together in a handful of diabetic dogs has provided promising results. More information on this is expected to become available soon which could lead to specific guidelines for the mainstream use of SGLT2 inhibitors in the management of canine diabetes diabetic dogs. This is especially exciting since improved glucose control, and in particular reduced glucose variability, will

be likely to result in a considerable decrease in the incidence of diabetic cataract, an expensive and frequent complication in the diabetic dog (leading to reduced quality of life and higher euthanasia rates).

For those animals still needing insulin injections, a new type of insulin, which evades clearance mechanisms by the body, will in future be able to be injected once every 1 or 2 weeks, rendering daily injections unnecessary. Pets (dogs and cats) could go to the vets for their insulin "shot".
Other strategies will focus on rapidly normalising body weight in cats through dietary intervention, resulting in the highest diabetic remission rates to date. Indeed, we already know that in overweight diabetic cats, achieving a degree of weight loss in the first month of their management of their makes diabetic remission up to 15 times more likely to occur.

Super-concentrated insulin Toujeo

Insulin Toujeo® is a specific formulation of highly concentrated (300U/ml) of insulin glargine. In both people and dogs, it has been shown to have a loner duration of action than glargine but most importantly it results in less day to day and within day variation in glucose control. This reduced variation means we can strive to have glucose levels maintained at lower levels as there is less risk of an unexpected hypoglycaemic event. As a result, the likelihood of diabetic cataract formation may also be reduced.

The optimal dose of Toujeo® for an individual dog is not at all predictable because a large dose range was found to be required to achieve good control.10 However, because Toujeo® has low day-to-day variability of insulin action, the effect of a given dose in an individual is relatively predictable. Therefore, when the response is closely monitored using a continuous glucose monitoring system (CGMS), clinicians can confidently and safely adjust the dose of Toujeo® rapidly to improve glycaemic control.

In a minority of diabetic dogs treated with q24h or q12h Toujeo® insulin, a substantial period of high glucose occurs for several hours following 1 or more meals during each 24 h cycle, while for the rest of the period there is very good glycaemic control. In many cases this can be resolved by changing the timing of the meals, the relative portion sizes fed at each meal and by reducing the carbohydrate content of the meal(s). Owners will often favour such an approach as an alternative to introducing an additional bolus injection of a shorter acting insulin such as caninsulin shortly (60-90 minutes) before feeding.

Continuous Glucose Monitoring Systems

The well-established considerable variation in glucose values from day to day in insulin-treated diabetic pets has taught us that relying on blood glucose curves on one particular

day and with only hourly or bi-hourly sampling, provides an inaccurate and at times random picture of the overall degree of glucose control. On this basis, many key opinion leaders advocate moving away from blood glucose curves towards the use of interstitial glucose measurement. With current available technology the interstitial glucose can be measured over several days and because the sensor is implanted subcutaneously there is no limit to the frequency with which the interstitial glucose can be estimated. Because of the possibility of measuring the interstitial glucose "continuously" this monitoring technique has become known as a "continuous glucose monitoring system" (CGMS).

Clearly, such technology brings us closer to the truth of the patient's average glycaemic control. It is important to point out that interstitial glucose is a different entity from blood glucose and therefore simultaneous blood and interstitial glucose measurement might well differ significantly at any point in time. This is not a sign that one measurement is superior to the other and simply signifies the different nature of the measured analyte in a different compartment. The magnitude of the data obtained with the CGMS can feel overwhelming, though the basics of diabetic assessment have not changed: we look at whether there are clinical signs of diabetes still present, we look at whether the glucose profile allows adjustment of the treatment protocol if there are still clinical signs or (in the dog) we worry about the development of cataract.

With the introduction of sodium glucose co-transporter 2 (SGLT-2) inhibitors as a registered treatment for feline diabetes mellitus, this oral hypoglycemic therapy is likely to be the first choice for managing diabetic cats.

Despite these future developments, in 2026 the vast majority of dogs with diabetes mellitus and a proportion of diabetic cats will need insulin supplementation for the rest of their lives making it hugely important that the insulin and feeding regime is tailored to suit the needs not just of the patient but also the owner.

Which insulin is best?

Insulins such as lente, protamine zinc and isophane insulins have been the mainstay of insulin therapy for cats and dogs for some time. Over the last 10 years the synthetic insulin analogues, insulin glargine and insulin determir, have been recommended for use as a twice daily exogenous insulin, especially in cats. The perceived advantages of these two insulins is an allegedly relatively "peakless" profile and longer duration of action than is seen with insulin lente or PZI. It is possible that these products' relatively "peakless activity profile" may make them a more suitable insulin for those cats that are keen to "graze".

However, when studies are “corrected” for diet, currently there is no substantial clinical data that suggests cats receiving twice daily glargine or determir are any better controlled or any more likely to go into remission than cats receiving twice daily lente or protamine-zinc insulins.

Additionally, while recent work at the Royal Veterinary College suggested diabetic cats changing from lente insulin to protamine zinc showed some clinical improvement, ***there was no significant difference in diabetic control between diabetic cats receiving glargine insulin verses matched diabetic cats receiving protamine-zinc insulin. Numerous indicators of diabetic control were utilized including average blood glucose, fructosamine, diabetic clinical score and insulin dose.***

Regardless of whichever of these “standard insulins” are used, the overall aim of standard insulin therapy is to administer the insulin and feed the animal such that the time of peak activity of the insulin corresponds to the peak demand i.e. when the blood glucose is rising after feeding. The aim is to lower blood glucose concentration over the 24-hour period and to minimise fluctuations as much as possible.

With the recent introduction of longer acting and more realistically peakless insulins such as the 300U/ml glargine insulin, insulin toujeo®, a different focus can now be explored. When using this type of insulin, the timing of the insulin and the meals are not particularly important as the principle is to try and achieve a lower average blood glucose for the majority of the 24 hour period while accepting inevitable episodes of increased blood glucose during the post-prandial periods. The aim is to have the overall average blood glucose kept closer to the reference range for normal blood glucose levels. With traditional insulins this inevitably meant an increase in the likelihood of clinically significant hypoglycemia. With the reported increased consistency of insulin toujeo, its proponents suggest this hypoglycemic risk is likely to be at a far more acceptable level.

Nevertheless, this type of insulin and feeding regime should never be undertaken without placing a continuous glucose monitor on the patient for the introduction of the insulin and its dosing adjustments.

Regardless of the insulin used, successful insulin therapy is based around reducing the average blood glucose concentrations to levels where clinical signs are effectively controlled. While it is perhaps too early to tell how best to manage the longer-acting basal insulins such as toujeo; certainly with the traditional insulin regimes, achieving these goals of controlling clinical signs and minimising the risks of clinically significant hypoglycemia invariably requires:

(i) insulin being administered at least twice daily with
(ii) the meal being offered an hour to an hour and a half ***after*** the insulin
(iii) some form of monitoring of the insulin and meal's effects on serial glucose levels. The mechanism for assessing how effectively the management is controlling blood glucose varies and the optimum means of doing this can be different for different patients and owners. Ideally a stable well controlled patient can be monitored with regular serial glucose 'curves'. In 2025 ideally this should be via a continuous glucose monitoring device measuring interstitial glucose
(iv) regardless of the method(s) used to quantify blood glucose levels the owners and members of the veterinary team should be encouraged to use the **"Diabetic Clinical Score"** (DCS) to "semi-quantify" the degree of diabetic control being achieved.

The DCS is a score out of 12 made up by giving a rating of between "zero" and "three" to four clinical parameters associated with poor diabetic control. The four components are:
(i) weight loss over the past 2 months,
(ii) degree of polydipsia and polyuria,
(iii) degree of increased appetite and
(iv) degree of decreased activity and attitude.

To facilitate the regular use of the DCS the Royal Veterinary College has developed the ***Pet Diabetes App*** which is freely available from all usual "App stores" for iPhone and android smartphone platforms.

Feeding and exercising the diabetic patient (dog or cat)

The diet must be consistent - the animal should consume the same caloric intake morning and night and from day to day. The food should be controlled and consistent:

- Commercial canned preparations with variable amounts of dry food are a convenient food type for diabetics as their caloric content is relatively predictable and consistent. However they may not be attractive to all patients, particularly if they have not been a traditional part of the animal's diet.

- When using relatively short duration insulins it is vital that all the offered food is consumed within a short space of time. In other words, caloric content and palatability are equally important. As a result, some diabetic dogs and cats need to be stabilised on standardised portions of their usual diets.

- Whatever the form of the calories fed to the diabetic patient, the timing and the caloric content must remain consistent. Any changes in the animal's diet should only be made if the overall caloric content remains unchanged.

- Ordinarily snacks should be provided minimally and ideally be low in carbohydrate concentrations; they can be valuable in providing "positive reinforcement" when the animal is being injected.

- As a general guide feed 50-60 kcals/kg body weight per 24 hours, fed as two meals 60-90 minutes after insulin injection.

- There is some evidence to suggest cats fed a low carbohydrate diet will have improved diabetic control (see below).

- There is no evidence that adding fibre to a diabetic's diet is likely to significantly improve poor diabetic control.

Diabetic nutrition: Low carbohydrate, high protein diet for cats and high fibre diets for dogs

Lowering the overall carbohydrate intake by feeding a diet low in carbohydrate and high in protein may theoretically lower insulin requirements and/or improve diabetic control. Over the last few years numerous clinical trials have demonstrated improved diabetic control and reduced insulin requirements in diabetic *cats* fed a low carbohydrate-high protein diet. In a number of these studies the 'control' group were fed a high fibre (& hence high carbohydrate) diet of differing starch sources (which may have an impact on the glycaemic index). However, notwithstanding these limitations the collective results strongly suggest feeding cats diets with constituent composition more closely resembling the composition of their natural carnivorous diet will increase diabetic remission rates, especially in those cats with marked insulin resistance through obesity. Further, to the author's knowledge, there is no evidence to support differences the way the high-protein diet is achieved be it commercially available products or so called "natural diets".

Also remember a low carbohydrate diet is effectively any commercial wet or canned food, it does not need to be a specific diabetic prescription diet.

The proportion of cats with uncomplicated diabetes mellitus likely to achieve a state where they no longer require exogenous insulin after receiving a combination of exogenous insulin and being switched to a high protein, restricted carbohydrate diet is still a point of conjecture.

However, it is generally accepted that upto 30% of early diabetics may ultimately be able to be managed without exogenous insulin and a recent study being undertaken at the RVC suggests this figure may be as high as ~60%. This invariably requires an initial period of exogenous insulin administration combined with ongoing high–protein diet and weight-loss to a normal body condition score.

While feeding high protein diets is likely to have benefits for improving diabetic control in the cat, there is no evidence that this type of diet or indeed the more traditionally recommended high fibre diet, has any role or impact on improving control of diabetes mellitus in the dog.

Exercise

Generally, exercise has the capacity to lower insulin requirements hence exercise should be consistent and encouraged. As diabetics are usually older animals that have been obese due to overfeeding and lack of exercise, over-exercise is not usually a problem.

Otitis Media – Diagnosis and Therapy

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Introduction

Otitis is one of the most common reasons for veterinary consultation in small animal practice. Approximately 15–20% of dogs and 6–7% of cats present with some form of ear pathology. Clinical signs are often recognized late by owners, and many patients are only presented once the condition has become chronic and severe.

Typical clinical signs include head shaking, scratching, erythema of the pinnae, otic discharge, and pain upon palpation. When the middle or inner ear is involved, additional neurological signs such as head tilt, facial nerve paralysis, Horner's syndrome, vestibular dysfunction, or hearing loss may occur. In rare cases, neurological deficits are the primary presenting complaint.

Otitis externa is primarily a clinical diagnosis. However, in chronic or treatment-resistant cases, concurrent otitis media must be ruled out. Importantly, an intact tympanic membrane does not exclude middle ear disease. Studies have shown that up to 59% of dogs with confirmed otitis media still had a macroscopically intact tympanic membrane.

Pseudomonas infections represent the most severe form of otitis. More than 50% of these cases involve the middle ear, and in chronic infections the prevalence may reach up to 75%. Therefore, comprehensive diagnostics including imaging, culture, and video otoscopy are mandatory.

Clinical Examination

As with all dermatological cases, the diagnostic approach begins with a thorough history. Primary causes such as allergic dermatitis, parasitic infestation, endocrine disorders, or keratinization defects must be identified. In allergic patients in particular, other body areas (paws, axillae, inguinal region) are frequently affected.

The ear examination consists of inspection (erythema, lichenification, hyperpigmentation, quantity and odor of discharge), palpation (pain, cartilage

elasticity, mineralization), and otoscopy (erythema, edema, epithelial hyperplasia, type and amount of exudate, erosions, ulcerations, parasites, foreign bodies, stenosis, glandular hyperplasia, and condition of the tympanic membrane).

Otoscopy

Otoscopy can be challenging in painful or heavily exudative ears. Even after flushing under general anesthesia, visualization of the tympanic membrane may remain difficult. One study demonstrated that the tympanic membrane could only be clearly identified in approximately 28% of cases after anesthetic ear flushing.

In cases of severe stenosis or marked epithelial hyperplasia, a 7–10-day course of a high dose of systemic corticosteroids without concurrent topical therapy is recommended. This reduces inflammation, edema, and pain, facilitating subsequent evaluation. Avoiding initial topical treatment in severely painful ears can also prevent the development of head shyness, which is particularly important in chronic cases requiring prolonged therapy.

Cytology and Bacteriology

Cytological examination of otic discharge is an essential diagnostic step in every otitis case. It enables identification of cocci, rods, yeast organisms, inflammatory cell types and biofilm, thereby guiding therapeutic decisions.

Bacterial culture and susceptibility testing are indicated in suspected or confirmed otitis media, presence of rod-shaped bacteria, marked neutrophilic inflammation, ulceration within the ear canal, or recurrent and treatment-resistant otitis. Whenever possible, samples should be obtained directly from the middle ear to increase diagnostic accuracy.

Biofilm in Otitis

Biofilm formation is a major factor in chronic and treatment-resistant otitis externa and media, particularly in infections with *Pseudomonas aeruginosa* but also with Staph. pseudintermedius and even Malassezia spp. infections. Within a biofilm, bacteria are embedded in an extracellular polymeric matrix that protects them from antibiotics, antiseptics, and host immune responses. Bacteria in this state exhibit reduced metabolic activity, which markedly decreases antibiotic susceptibility. Consequently,

the minimal biofilm eradication concentration may be up to 1000 times higher than the MIC of planktonic organisms.

Clinically, biofilms contribute to persistent gram-negative rods, cocci or yeast despite appropriate antimicrobial therapy and to rapid relapse after treatment cessation.

Diagnostic Imaging

Imaging is indicated in suspected otitis media or interna. Radiography has a sensitivity of approximately 46%, whereas computed tomography (CT) reaches a sensitivity of 83% and specificity of 89%. CT provides valuable information regarding soft tissue opacification of the bulla, canal stenosis, mineralization, osteitis, or osteolysis.

Magnetic resonance imaging (MRI) is particularly useful in acute cases, soft tissue assessment, and suspected inner ear involvement.

A recent study in cats (Chan et al., Veterinary Dermatology, 2025) demonstrated that ultrasonographic evaluation of the tympanic bullae achieved a sensitivity of 92% and specificity of 100% for detecting fluid accumulation. Overall diagnostic accuracy reached 97%, indicating that ultrasound may represent a valuable non-invasive diagnostic tool in feline otitis media.

Video Otoscopy and Myringotomy

Video otoscopy has both diagnostic and therapeutic value. It allows visualization and flushing under direct vision, collection of biopsy samples and bacterial cultures, removal of foreign material and debris, and direct instillation of medication into the bulla.

Myringotomy is indicated in confirmed or strongly suspected otitis media. The tympanic membrane is perforated in the posteroventral quadrant of the pars tensa (approximately the 5–7 o'clock position). The procedure allows drainage of middle ear contents, collection of cytological and bacteriological samples, and thorough lavage of the bulla.

Therapeutic Principles in Otitis media

Successful treatment of otitis media relies on bulla lavage under general anesthesia, control of inflammation with systemic corticosteroids (e.g., prednisolone 1–2 mg/kg once daily for 2–4 weeks), and systemic antimicrobial therapy based on culture and

susceptibility testing for 4–8 weeks. Insufficient duration increases the risk of relapse. In cases of multi-resistant bacterial infection, topical antibiotic therapy is the only option.

Topical Therapy

Topical therapy is usually administered twice daily. Pretreatment with Tris-EDTA may enhance antimicrobial efficacy especially in gram-negative infections. Fluoroquinolone-, polymyxin-, or aminoglycoside-containing preparations are commonly used.

In the presence of biofilm, effective management requires mechanical disruption via thorough lavage under video otoscopy, ideally with high-volume saline flushing of the bulla. Adjunctive biofilm-disruptive agents such as Tris-EDTA or N-acetylcysteine may enhance antimicrobial penetration. High local antibiotic concentrations achieved through topical therapy are critical. Without adequate biofilm control, long-term resolution of chronic otitis media is unlikely.

In-vitro resistance does not necessarily predict failure of topical therapy, as local drug concentrations can far exceed systemic levels. Ototoxic agents should be avoided if tympanic membrane integrity is uncertain. High volumes (0.5–1 ml per ear) are necessary to ensure adequate distribution.

Follow-Up and Prognosis

Re-evaluations are recommended every 10–14 days for 6–8 weeks. Cytology is essential for monitoring therapeutic success. Lack of clinical improvement, persistence of gram-negative rods, or ongoing neurological deficits may necessitate surgical intervention such as bulla osteotomy.

Conclusion

Otitis media requires systematic diagnostics and prolonged therapy. An intact tympanic membrane does not exclude middle ear involvement. Video-otoscopy, adequate lavage, appropriate systemic and/or topical antimicrobial therapy, anti-inflammatory treatment, and control of the primary underlying cause are essential for long-term success.

The Use of Cold Atmospheric Plasma in Veterinary Dermatology

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Introduction

Cold atmospheric plasma (CAP), commonly referred to as cold plasma, represents an emerging non-thermal therapeutic modality in veterinary dermatology. Plasma is considered the fourth state of matter and consists of partially ionized gas containing reactive oxygen and nitrogen species (RONS), charged particles, UV photons, and transient electric fields. In medical-grade devices, CAP is generated at near-physiological temperatures, allowing safe application to living tissue without thermal injury.

Generation of Cold Plasma

Cold plasma is typically produced by applying high-voltage energy to a carrier gas such as argon, helium, or ambient air. This process results in partial ionization and formation of biologically active reactive species including superoxide, hydrogen peroxide, nitric oxide, peroxynitrite, and hydroxyl radicals. These reactive species are responsible for the biological and antimicrobial effects of CAP.

Mechanism of Action

Cold plasma exerts broad-spectrum antimicrobial effects through oxidative damage to bacterial cell membranes, DNA and protein oxidation, and disruption of metabolic pathways. It also destabilizes the extracellular polymeric substance matrix of biofilms, increasing bacterial susceptibility to host immunity and topical therapy. Importantly, CAP is effective against multidrug-resistant organisms and has not been associated with clinically relevant resistance development. This is particularly valuable in chronic otitis and deep pyoderma.

Beyond antimicrobial effects, CAP modulates redox signaling pathways. Controlled exposure activates Nrf2 signaling, leading to upregulation of endogenous antioxidant enzymes, and modulates NF-kB activity, reducing excessive inflammatory cytokine production. CAP stimulates keratinocyte migration, enhances fibroblast proliferation, and promotes angiogenesis. Thus, it provides a controlled oxidative stimulus that supports the transition from chronic inflammatory phase to proliferative wound healing.

CAP also exerts anti-inflammatory effects by reducing local levels of pro-inflammatory mediators such as TNF-alpha and IL-1 beta while improving microcirculation and tissue oxygenation. Clinically, this contributes to reduced edema, pain relief, and improved wound environment.

Clinical Benefits in Veterinary Dermatology

In veterinary dermatology, cold plasma has demonstrated potential benefits in chronic non-healing wounds, surgical wound complications, superficial and deep pyoderma, acral lick granuloma, chronic, biofilm-associated infections, vasculitis and otitis externa. Advantages include non-invasive application, absence of systemic side effects, minimal discomfort, reduction of systemic antibiotics, and suitability for repeated treatments, even by the patient owners.

Treatment duration typically ranges from thirty seconds to several minutes per lesion depending on device type and lesion size. CAP is commonly carried out two to three times weekly in chronic conditions. Application may be direct or near-contact depending on the technology used.

In feline patients, CAP offers a rapid, contact-limited and generally well-tolerated intervention that can reduce the need for extensive topical treatment. Potential feline indications include chronic wounds, feline acne, eosinophilic granuloma complex lesions. Although controlled feline-specific studies remain limited, early clinical experience suggests favorable tolerability and safety.

Cold atmospheric plasma also shows promise in papillomavirus-associated lesions. CAP exerts antiviral effects through oxidative damage to viral capsid proteins and nucleic acids and may impair viral replication. Additionally, local immune modulation induced by CAP may enhance innate antiviral responses. In canine papillomatosis, CAP may contribute to regression of persistent lesions by combining mild cytotoxic effects on hyperproliferative keratinocytes with immunostimulatory mechanisms.

In fungal infections, CAP demonstrates direct antifungal activity through oxidative disruption of fungal cell walls, membranes, and intracellular components. Reactive species can impair ergosterol-containing membranes and reduce spore viability. In vitro data support susceptibility of dermatophytes and yeasts relevant to veterinary medicine, including Microsporum and Malassezia species. Clinically, CAP may serve as an adjunctive therapy in localized dermatophytosis, particularly in patients that poorly tolerate topical antifungal therapy or when systemic antifungal use is limited by contraindications. Nevertheless, CAP does not replace systemic antifungal therapy in generalized dermatophytosis or deep mycoses, as penetration depth is limited and fungal elements within hair shafts or deeper tissues may persist.

Cold plasma operates at temperatures generally below forty degrees Celsius, preventing thermal tissue damage. Adverse effects are rare and may include mild transient erythema. Compared to conventional antiseptics, CAP demonstrates lower cytotoxicity toward mammalian cells when appropriately dosed.

Conclusions

In summary, cold atmospheric plasma represents a promising antimicrobial and redox-modulating technology in veterinary dermatology. Its dual action of biofilm disruption and regenerative signaling makes it particularly suitable for chronic, therapy-resistant dermatologic conditions and may contribute to reduced antibiotic use and improved long-term outcomes in small animal practice.

Advances in feline CKD

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Feline CKD is a progressive, multifactorial disease in which earlier diagnosis, disease staging, and individualised management are recognised as increasingly important. This lecture focuses on recent advances in biomarkers (FGF-23), early dietary intervention, the gut-kidney axis, development of hypercalcaemia in cats fed a renal diet, and anaemia of CKD, with the practical goal of improving quality of life and survival in affected cats.

FGF-23

FGF-23 is one of the most important new biomarkers in feline CKD. It is secreted by osteoblasts in response to phosphate-related signals and acts to decrease circulating phosphate by increasing renal phosphate excretion and reducing calcitriol production. In cats, FGF-23 rises with declining renal function and has been associated with progressive CKD, making it a potential early marker and prognostic biomarker. FGF-23 is also increased before overt azotaemia develops. The International Renal Interest Society (IRIS) recommends increased FGF-23 in the presence of normophosphataemia as a marker to indicate initiation of feeding a renal diet.

Dietary management for early stage CKD

Diet remains the cornerstone of CKD management, but the evidence for when to initiate diet in early disease is still evolving. Traditional renal diets are lower in protein, phosphate, and sodium, and often include increased omega-3 fatty acids. The strongest nutritional evidence still supports phosphate restriction.

Early renal diets designed for cats before overt azotaemia are less restricted in phosphate but trials have been underpowered and possibly of too shorter length to demonstrate effects on delaying disease progression in early CKD populations. A major concern raised is that too much phosphate restriction in cats that do not truly need it may contribute to ionised hypercalcaemia.

Development of hypercalcaemia in cats fed renal diet

Diet-induced hypercalcaemia is increasingly recognised in cats. Cats fed phosphate-restricted renal diets can develop increased total or ionised calcium, particularly when phosphate is already within target range. This suggests that overly restricting phosphate in early disease may have unintended consequences.

The mechanism is not fully understood. Although secondary and tertiary hyperparathyroidism and calcitonin responses are considerations, the evidence suggests it is related to the calcium: phosphate ratio of renal diets being too high resulting in increased calcium absorption. Therefore, if a cat develops hypercalcaemia when fed a standard renal diet then transitioning to an early renal diet that has less phosphate restriction should be considered.

Gut-kidney axis

Cats with CKD can develop intestinal dysbiosis, which may increase colonic production of uraemic toxins such as indoxyl sulfate and p-cresol sulfate. These toxins are associated with inflammation, disease progression, and broader systemic effects.

Several interventions have been explored, including probiotics, adsorbents, fibre supplementation, and dietary protein modification. Evidence is mixed, but more fermentable fibre sources appear preferable to less fermentable ones in some studies, and the overall concept is that modifying the intestinal environment may help reduce uraemic toxin burden. Constipation and excessive antibiotic use may worsen dysbiosis and absorption of uraemic toxins, so they should be avoided when possible.

Anaemia in CKD

Anaemia is common in more advanced feline CKD. When CKD-related anaemia is present, impaired erythropoietin production, inflammation, iron dysregulation, and reduced red blood cell lifespan may all contribute.

Iron assessment is important because functional iron deficiency can occur even when total body iron stores are not completely depleted. Treatment options include iron supplementation, darbepoetin, recombinant erythropoietin products, and newer HIF-inhibitors, though each carries potential adverse effects and monitoring requirements.

Clinical approach

Feline CKD management increasingly combines serial biomarker monitoring, dietary individualisation, careful calcium-phosphate regulation, gut-directed strategies, and treatment of complications such as anaemia. Improved outcomes

may result from earlier recognition and a more tailored approach for each individual cat.

Feline cardiorenal syndrome

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Feline cardiorenal syndrome refers to cardiac and renal dysfunction, where disease in one organ contributes to disease in the other, or both are affected by the same systemic process. In cats, the concept is still emerging, however it is increasingly recognised that clinically relevant cardiorenal interactions do occur and should be considered in how we monitor and treat feline patients.

Definition and classification

Cardiorenal syndrome is defined as dysfunction in either the heart or kidney leading to dysfunction in the other organ, or concurrent dysfunction from systemic disease. A veterinary cardiovascular-renal disorder classification has been suggested, which parallels human classifications and groups disease into five types based on whether the primary problem is cardiac, renal, or systemic, and whether it is acute or chronic.

C_vRD_H (unstable) is acute cardiac disease causing acute kidney injury, C_vRD_H (stable) is chronic cardiac disease causing progressive CKD, C_vRD_K (unstable) is acute renal dysfunction causing acute cardiac dysfunction, C_vRD_K (stable) is chronic kidney disease contributing to cardiac dysfunction and C_vRD_O describes both organs being affected secondary to another systemic disease such as sepsis.

Does it exist in cats?

There is some evidence that cardiorenal syndrome exists in cats, even if the exact boundaries are not always easy to define clinically. Several retrospective studies show azotaemia or worsening creatinine in cats with congestive heart failure, and renal injury appears particularly common around initiation of furosemide therapy. In one study, 44% of cats with CHF were azotaemic at diagnosis, and 66% had at least one episode of acute-on-chronic kidney injury during follow-up.

Other studies suggest a relationship between structural heart disease and renal complications. Cats with renal infarcts were more likely to have hypertrophic cardiomyopathy, and echocardiographically detectable cardiomyopathy was more common in cats with renal infarction than in those without. Conversely, CKD and hypertension are associated with cardiac structural changes, including myocardial fibrosis, myocyte hypertrophy, and microvascular changes.

Pathophysiology

The pathophysiology is multifactorial including haemodynamic factors, thromboembolism, inflammation, RAAS activation, sympathetic stimulation, uraemic toxins, oxidative stress, electrolyte abnormalities, renal congestion, and endothelial dysfunction as major contributors. These mechanisms often overlap and amplify each other rather than occurring in isolation.

In cats, systemic hypertension is important. High blood pressure can damage renal arterioles and glomeruli, contributing to arteriosclerosis and renal injury. Chronic kidney disease is also associated with inflammatory markers such as serum amyloid A and hepcidin, supporting the idea that CKD behaves as a chronic inflammatory state. Uraemic toxins such as indoxyl sulfate rise with CKD stage and may drive vascular and cardiac disease, while FGF-23 is a potential contributor to cardiac remodelling even though current feline data are limited.

Diagnosis and monitoring

There is no single test for cardiorenal syndrome in cats. Diagnosis depends on identifying evidence of both cardiac and renal disease and monitoring patients with one disease for early signs of development of the other. Renal assessment commonly relies on surrogate markers of GFR including creatinine and SDMA, and also renal imaging. Cardiac monitoring can include thoracic radiographs, echocardiography, cardiac troponin, and NT-proBNP. Troponin reflects myocyte injury, while NT-proBNP is released in response to myocardial stretch and volume overload.

Management

Management is a balancing act between protecting the heart and preserving kidney function. In cats with CHF, the priority is controlling fluid overload, but excessive diuresis can reduce renal perfusion and worsen kidney injury. Urine output, body weight, respiratory rate, heart rate, blood pressure, and renal markers, electrolyte status should all be monitored closely.

In cats with CKD and cardiac disease, treatment should aim to avoid volume overload, correct electrolyte disturbances, and consider the renal handling of cardiac drugs. Monitoring and management of hypertension with drugs such as amlodipine and telmisartan are also important.

Prognosis

Cardiorenal syndrome likely worsens prognosis, but the influence in cats remains to be fully determined. Older cats with CHF and azotaemia in particular may have a guarded prognosis, especially because age itself is a major predictor of survival. Overall, the field of cardiorenal syndrome is still evolving, and better biomarkers and definitions are needed to improve diagnosis, monitoring, and outcome prediction

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Surgical approach to emergency obstructions: GDV, intestinal occlusion, and foreign bodies

1. General considerations

Stabilization is the main objective for any gastro-intestinal obstruction before going to surgery. Animals are at risk of fluid and electrolyte imbalances. Hypovolemia, hypokalaemia, hyponatremia, hypochloraemia, and compression of the organs should be corrected before the surgical correction. Surgery may or may not be delayed depending on the origin of the obstruction.

2. Gastric dilatation and volvulus

a. Timing of the surgery

First the animal is stabilized, needle decompression of the stomach and fluid resuscitation are performed immediately on admission of the animal. Recent studies have not detected any difference in survival after surgery performed after a rapid (median time of surgery 2.1 hours after presentation) or a prolonged medical stabilization (median time of surgery 9.8 hours after presentation). This implies that surgery can be planned after aggressive stabilization and delayed until the next day if the animal can be monitored during the night.

b. Surgical exploration

Surgical exploration is indicated in all cases of GDV, due to the high risk of recurrence without gastropexy (80%).

A cranial laparotomy is performed. Due to the rotation, the greater omentum covers the stomach, the pylorus is sought on the left cranially and an anticlockwise movement is performed whilst pushing the body of the stomach caudally and to the left to reposition the stomach. The orogastric tube is reinserted by the anaesthesia team, the surgeon can control the passage through the cardia.

The fundus and the greater curvature are inspected, the short gastric arteries are checked for haemorrhages.

The spleen is inspected, in the event of torsion, tearing of the vessels, or a lack of pulse in the splenic artery, the spleen should be removed.

The viability of the greater curvature and fundus is assessed. The four criteria to be evaluated are the colour, palpation, presence of persistaltic waves, and capillary perfusion. If the wall is dark red, purple, or black,

ischaemia is present and the affected section of the stomach should be removed. If it is not possible to distinguish between the muscular/serosal and the mucosal/submucosal layers on palpation, it is because the latter is necrotic and a gastrectomy is indicated. The absence of peristaltic waves is also a sign of non-viability. Incision of the serosa should result in arterial bleeding. Up to 60% of the stomach can be removed. The stomach is isolated using moist abdominal swabs, Doyen forceps are placed to isolate the section of stomach that requires resection. The wall is cut using a scalpel and the defect closed using an appositional simple continuous suture with resorbable monofilament. The use of several cartridges of staggered staples makes closure easier, faster with less risk of spillage, then the suture is buried with a simple continuous inverting suture pattern. A gastrectomy is performed in 10% of GDV cases.

c. Gastropexy

A gastropexy is always performed on the right-hand side between the cranial body wall and the pyloric antrum. Several technics have been described, the incisional gastropexy is the simplest and mostly used:
A 4-5 cm longitudinal incision is made in the seromuscular layer at the middle of the pyloric antrum (taking care to avoid penetrating the submucosa). A 4-5 cm incision is made in the transverse abdominal muscle parallel to and 6-8 cm from the laparotomy incision on the right side, opposite to the stomach just behind the last rib. The edges of the incision in the stomach are sutured with the margins of the transverse abdominal muscle using two layers of simple continuous sutures with 2/0 or 3/0 resorbable monofilament.

d. Postoperative care

Fluid therapy is continued until the animal can feed normally again. Potassium levels are monitored and supplemented where necessary. Water and food should be offered from the day after surgery.
The animal should be fed in 3-4 small meals per day for the rest of its life.
An ECG is repeated daily for 3 days and any ventricular premature contractions (VPC) treated with lidocaine.
Metoclopramide is given for 5 days.

e. Prognosis

Negative factors include cardiac arrythmias, hypoglycaemia, gastrectomy, splenectomy, and high lactatemia post resuscitation.
The survival rate is around 75% with surgery.

3. Intestinal occlusion

The causes of intestinal occlusion are intraluminal (foreign body, intussusception), intramural (tumour), and extramural (volvulus, incarceration, strangulation). Foreign body occlusion will be discussed in the next paragraph.

a. Intussusception

This is the invagination of an intestinal segment (intussusceptum) into the adjacent segment (intussuscipiens). The most common location is at the ileocolic junction (the small intestine invaginates into the colon). The entire gastrointestinal tract is inspected as several intussusceptions may be present. The intussusception can be reduced by delicate traction on the intussusceptum and pressure on the intussuscipiens. If it is not possible to reduce it due to fibrosis or if a segment is not viable, an enterectomy / anastomosis is performed. It is important to ensure that no more than 75 to 80% of the small intestine is removed, and, when possible, preserve the ileocolic junction. Recurrence at a different site occurs in 3–25% of cases regardless of resection, with idiopathic intussusceptions carrying the highest risk.

Enteroplication may prevent recurrence but is associated with 19% complications rate.

It is recommended to prevent recurrence when no cause is identified. The enteroplication should start at the duodeno-colic ligament and finish at the ileocolic junction: the small intestine should not be too folded over on itself, the plicature sutures are placed at regular intervals, the intestinal anastomosis (if performed) should not be at the extremity of a bend, the sutures used are simple interrupted resorbable sutures penetrating the submucosa and placed halfway between the mesenteric and antimesenteric borders. Enteroplication has not demonstrated any benefit in cats.

b. Tumours

The most common small bowel tumours causing obstruction in dogs and cats are adenocarcinomas, leiomyosarcomas, and gastrointestinal stromal tumours (GISTs). Regardless of the histopathological findings, tumour resection improves the animal's survival rate. Adenocarcinomas are characterized by intraluminal growth, leading to small bowel obstruction. Leiomyosarcomas and GISTs, on the other hand, exhibit extraluminal growth that can lead to intestinal perforation.

	Cat		**Dog**
	No surgery	**Surgery**	**Surgery**

		No metastasis	With LN metastasis	No metastasis	With LN metastasis
Adenocarcinoma	22 days	843 days	358 days	544 days	
Leiomyosarcoma	rare			237-599 days	
GIST				356-1266 days	

c. Volvulus

Mesenteric volvulus is a rare and often fatal condition in which the bowel twists on its mesenteric axis, resulting in strangulating mechanical obstruction and ischemia of the small intestines. It is a true surgical emergency. If diagnosed late, most of the digestive tract is already ischemic and necrotic, and animals often die or must be euthanized during surgery. Survival is possible after derotation alone, derotation followed by resection and anastomosis, or resection and anastomosis (duodenoileal anastomosis) without derotation. Intestinal derotation can worsen the systemic response due to reperfusion injury.

If the torsion is segmental, resection and anastomosis without derotation offer a better prognosis.

d. Incarceration and strangulation

Intestinal incarceration is the abnormal displacement and entrapment of bowel within omental defects, hernias, or mesenteric tears and may quickly progress to strangulation, leading to intestinal ischemia and septic or endotoxic shock. Management requires prompt stabilization followed by surgical correction of the displacement and its underlying cause, with intestinal resection when needed; surgery must not be delayed if strangulation or necrosis is suspected, and nonviable herniated segments should be resected and anastomosed before returning the remaining bowel to the abdominal cavity.
The prognosis for intestinal incarceration depends on the severity of vascular compromise and the resulting systemic inflammatory response, being good when involvement is limited and cardiovascular parameters remain stable but guarded to poor when extensive ischemia and endotoxemia are present.

4. Foreign bodies

Foreign material can result in partial or complete intestinal luminal obstruction, tissue necrosis, intestinal perforation, or a combination of these.
The various surgical approaches include advancing the foreign body into the stomach or colon, enterectomy, and enterectomy-anastomosis in cases of severe ischemia or perforation. The surgeon must inspect the entire digestive tract, as multiple foreign bodies may be present. The choice of surgical technique depends on the mobility of the foreign body and the degree of intestinal ischemia. (table 1).

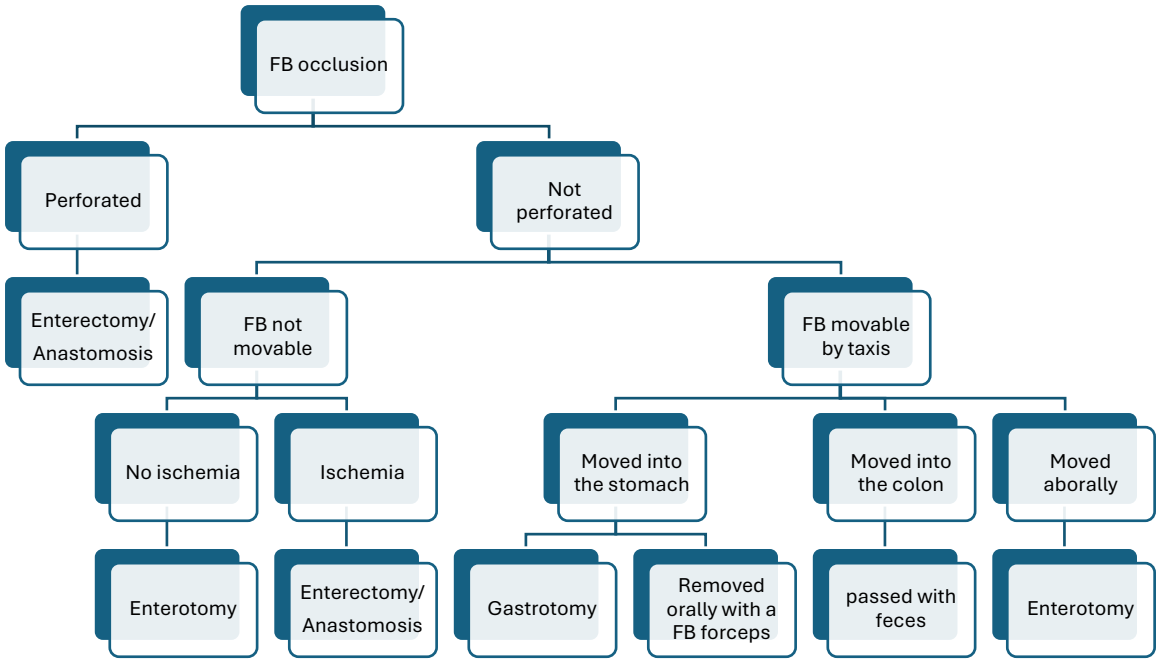


Table 1: Decision making for foreign body occlusion

5. Conclusion

Successful management of GDV, intestinal occlusions, and foreign bodies relies on rapid stabilization, timely surgery guided by bowel and gastric viability, and meticulous perioperative care, as prognosis is primarily determined by the duration and vascular consequences of the obstruction.

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Urethrostomy: surgical approach

Urethrostomy is the creation of a permanent opening between the urethra and the skin.

1. Anatomy

In cats, the urethra is 8.5 to 10.5 cm long. The preprostatic urethra is around 2.0 mm in diameter, the post prostatic and the pelvic urethra are 1.9 to 2.6 mm in diameter. At the level of the bulbourethral grand the urethra is around 1.8 mm in diameter, and the penile urethra is very narrow with a diameter of 0.7 mm. This implies that to have the biggest urethral diameter, the urethra must be opened up to the bulbourethral glands at distal end of the beginning of the pelvic urethra.

2. Indications

The most common indication for perineal urethrostomy is recurrent urethral obstruction despite appropriate management for feline lower urinary tract disease. Other indications include lesions distal to the bulbourethral glands like penile urethra stenosis, rupture, and acquired or congenital malformations.
The surgery is rarely done on emergency.

3. Techniques

Several locations of the stoma have been described. Perineal urethrostomy is the preferred and main technique used. Other techniques include transpelvic, subpubic, and prepubic urethrostomy.
Various techniques and modifications have been described for perineal urethrostomy in male cats. The original technique describes cutaneous anastomosis with the penile and beginning of the pelvic urethra. Other techniques preserve the preputial mucosa in order to suture it to the opened urethra.
Another variation of the technique is to perform the surgery the cat in dorsal recumbency.
Sutures at the stoma can be in a simple interrupted or simple continuous pattern, with a resorbable or non-resorbable suture material 4/0 or 5/0.
Regardless of the technique used, the keys steps of the surgery are:

- Dissection of the penis
- Section of both ischio-cavernosus muscles

- Ventral dissection of the entire pelvic urethra from the pelvis in order to be able to translate the blocked urethra/bladder caudally
- Opening of the dorsal urethra up to the bulbourethral glands
- Checking the diameter of the opening with a 8 or 10 Fr urinary catheter.
- Suture the urethral wall to the skin using a simple continuous suture pattern with a 4/0 resorbable suture.

4. Surgery (https://www.youtube.com/watch?v=pel7q4gBHrg)

Premedication: Medetomidine 2µg / kg, methadone 0.2 mg / kg,
Induction: alphaxalone
Maintenance: isoflurane
Epidural: morphine 0.2 µg / kg, bupivacaine 5% 0.1ml / kg

The author describes a perineal urethrostomy with preservation of the preputial mucosa in ventral recumbency. The perineum is raised; an anal purse string suture is placed.
A new urinary catheter is inserted; the bladder is emptied to reduce the gravitational pulling during the dissection of the penis. The preputial mucosa is incised longitudinally dorsally and circumferentially at the base of the glans (picture1). An elliptical incision is performed around the scrotum dorsally in continuity with the preputial incision. The incision is enlarged laterally on both sides to open widely the stoma (picture 2). The penis is dissected from the preputial mucosa and from the surrounding tissues (picture 3). It is moved dorsally and its base is dissected with Metzenbaum scissors ventrally and laterally, the ischio-cavernous muscles are identified (picture 4). The ischio-cavernous muscles are sectioned at their insertion on the ischial tuberosity to limit bleeding. The fibrous insertion of the pelvic urethra is dissected totally from the ventral pelvis with scissors or the finger until entering the abdominal cavity (picture 5). Then the block penis/bladder can be translated caudally. The penis is then placed ventrally. Dorsally, the penile retractor muscle is removed from the penis. The urethra is then dissected dorsally paying attention to the rectum until the bulbo-urethral glands are visualized (picture 6). The urinary catheter is removed. The penile urethra and penis are amputated just distal to the attachments of the ischiocavernosus muscles (picture 7). The two cut corpus cavernosus are grasped with an Allis tissue forceps and the urethra is recatheterized. The dorsal aspect of the urethral is incised longitudinally with an 11 blade over 2 to 3 mm (picture 8), Metzenbaum scissors are then used to open the urethra longitudinally up to the bulbourethral glands (picture9). The diameter of the urethra is checked by inserting an 8 or 10 Fr urinary catheter (picture 10). Three 4/0 to 5/0 resorbable monofilament simple continuous suture patterns are used to close the surgical site. The first continuous suture is placed

between the dorsal wall of the urethra and the dorsal beginning of the skin incision. It then continues to the right, encompassing the skin, urothelium, and part of the corpus spongiosum (the urothelium being too fragile to be sutured alone) (picture 11). Once the distal end of the right border of the urothelium is reached, the right corner of the preputial mucosa is lifted, and they are sutured together (picture 12). The running suture continues horizontally between the preputial mucosa and the distal portion of the urethra. Once the left corner of the distal urethra and the preputial mucosa are reached, the running suture is continued to the left, encompassing the skin and the left border of the open urethra, and finally returning to its dorsal starting point. Two small continuous running sutures are then made at 4 and 8 o'clock between the skin and the preputial mucosa (picture13).
The 8 or 10 Fr mm urinary catheter is then reinserted, and the bladder emptied and flushed several times to remove sand, blood, or fibrin clots.

5. Post-operative care

The purse string suture is removed, and a buster collar is placed for 15 days. Nonsteroidal anti-inflammatory drug is given for 3 to 5 days for pain management. In the early postoperative period shredded paper or commercial foam litter is recommended in place of regular clay litter, and petroleum jelly may also be applied to the peristomal region to prevent urine scald.

6. Complications

Postoperative complications include hemorrhage, stricture, urine extravasation, and wound dehiscence. The most recent studies report 10 to 12.8% complications rate, the two most important of these are stricture and urine extravasation.
Strictures most commonly develop secondary to excessive tension on the stoma from a failure to free the pelvic urethra from the floor of the pelvis, and from insufficient dissection of the urethra with creation of the stoma too caudal. Urine extravasation typically results from imprecise urethrocutaneous apposition. Long-term complications were reported in 28% of cats that underwent perineal urethrostomy, with bacterial urinary tract infections and recurrent FLUTD being most common. In the latest studies the long-term complication rate is very low.

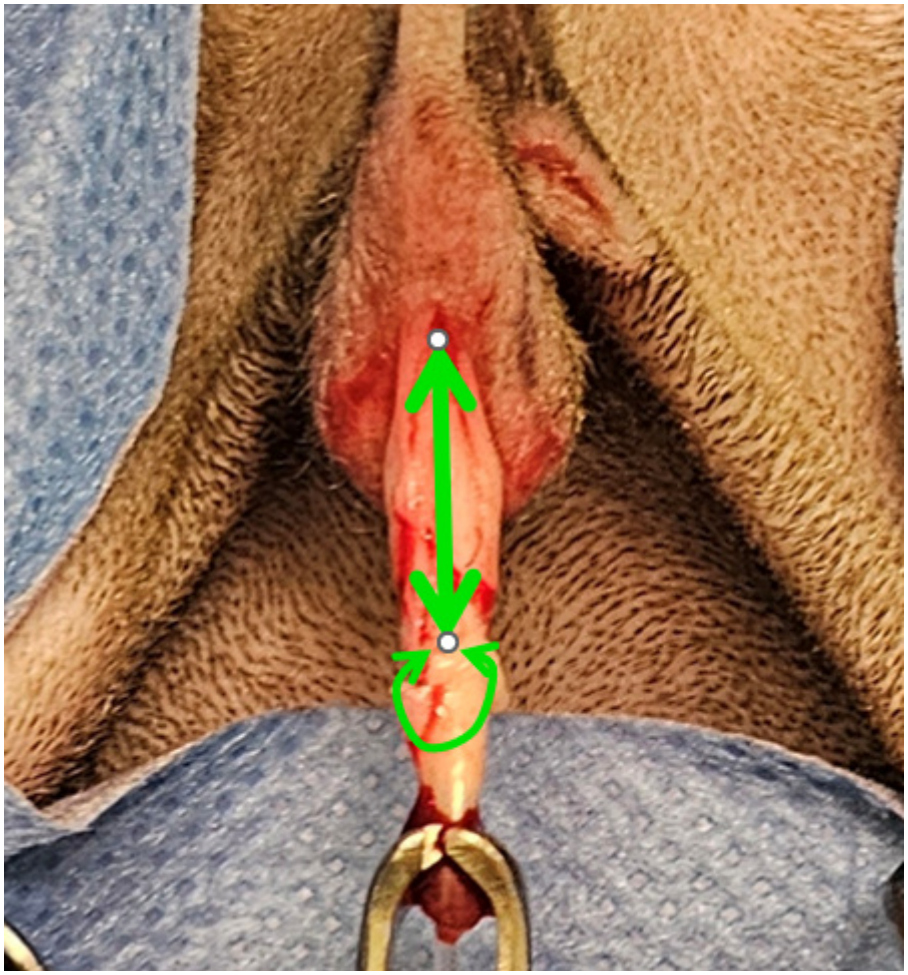
Perineal urethrostomy provides a good long-term functional outcome in cats when performed correctly.

To Watch

- https://www.youtube.com/watch?v=pel7q4gBHrg

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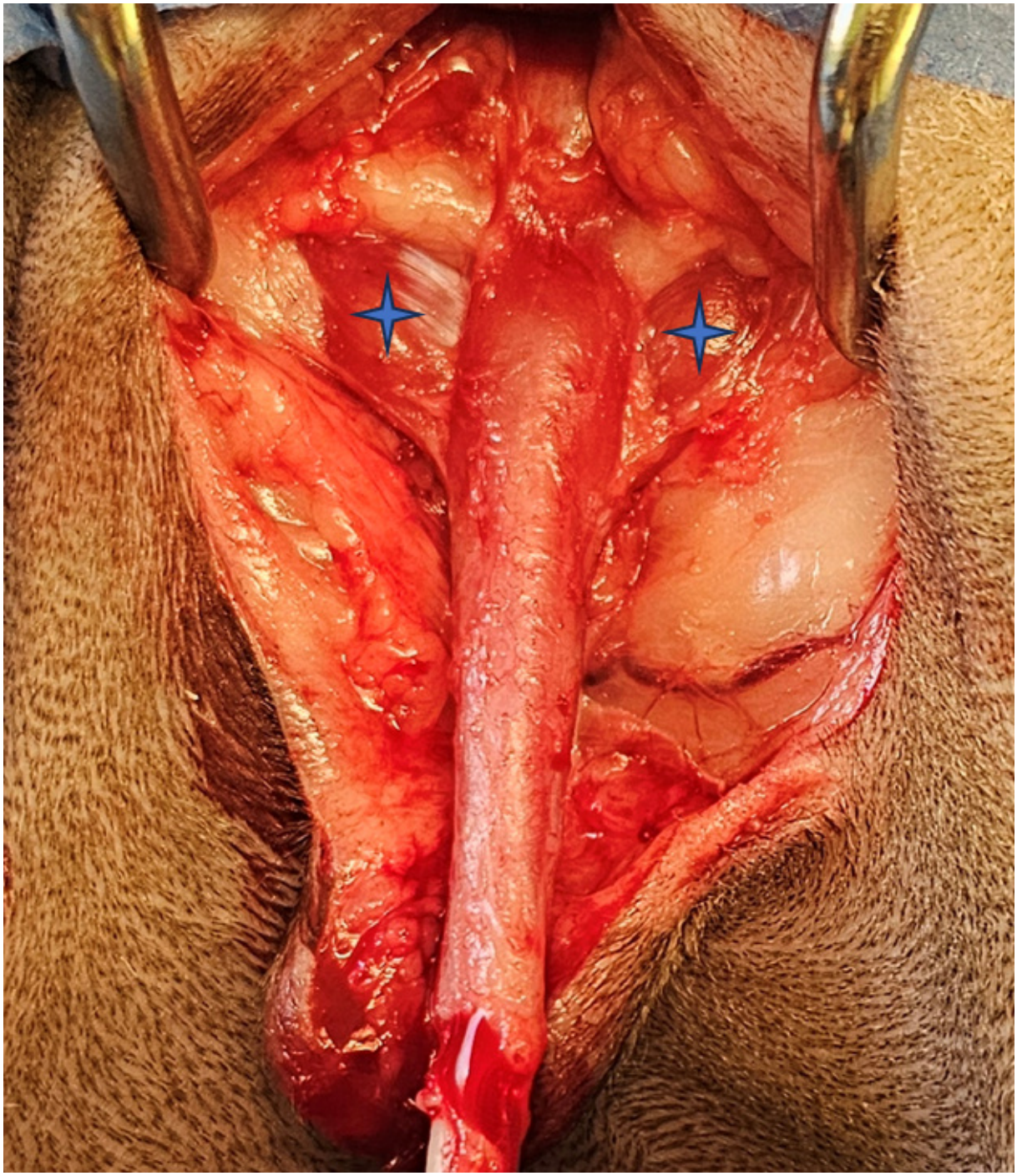
Picture 1: preputial incisions



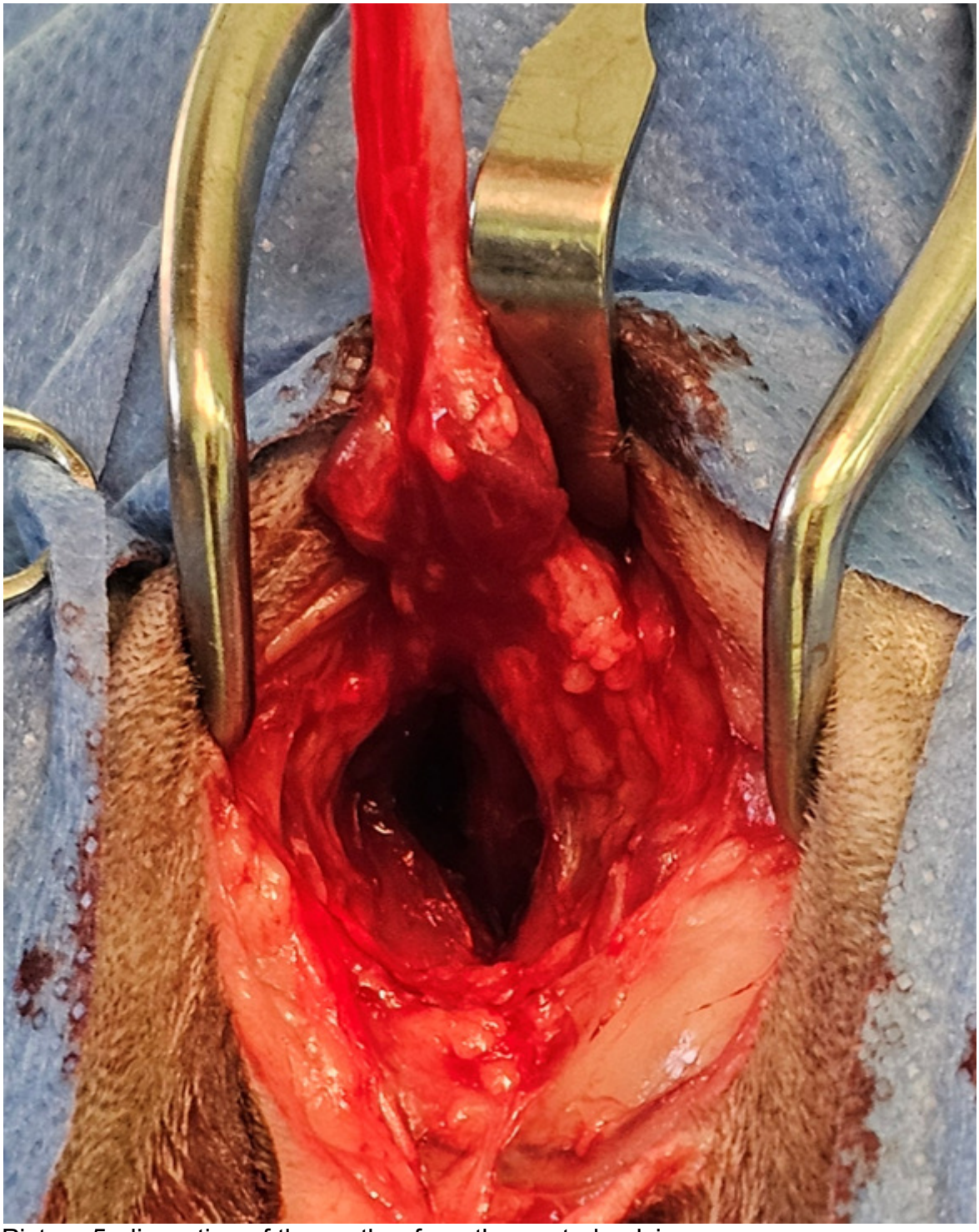
Picture 2: skin removal at the site of the scrotum



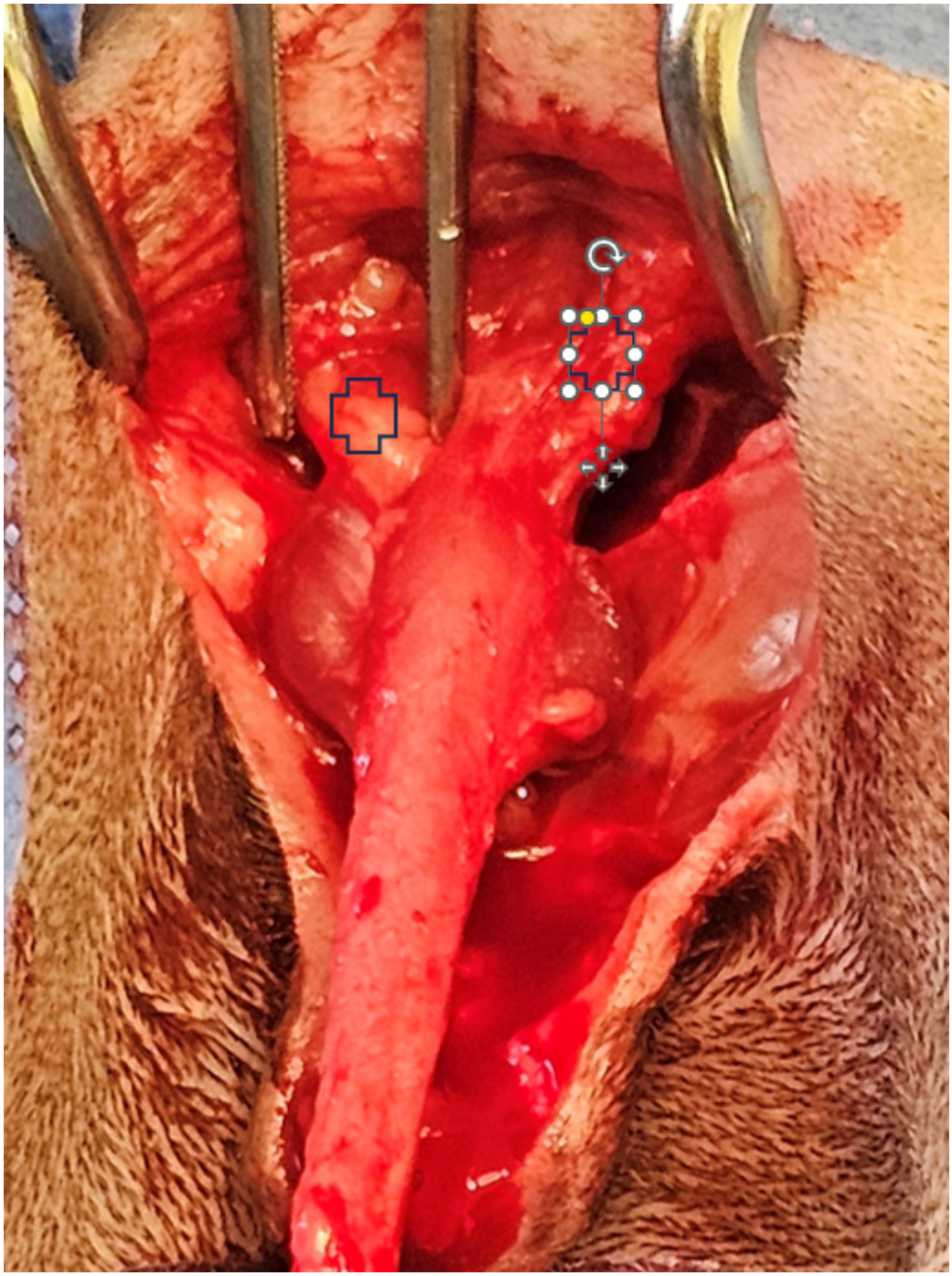
Picture 3: the penis has been dissected from the surrounding tissues



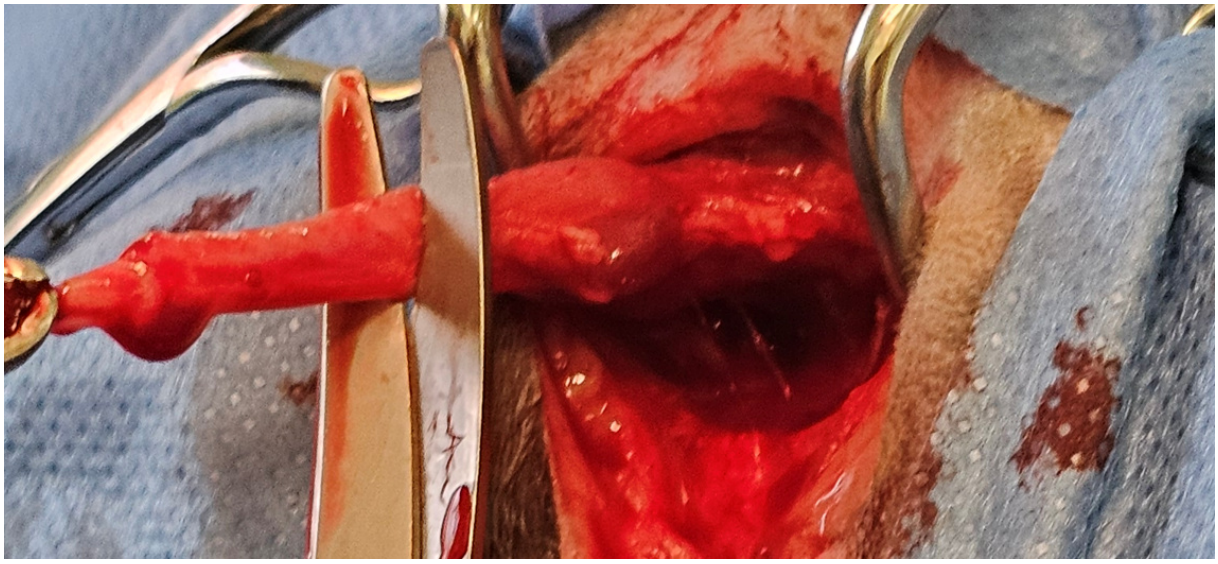
Picture 4: dissection at the base of the penis with visualisation of the ischio-cavernosus muscles (stars)



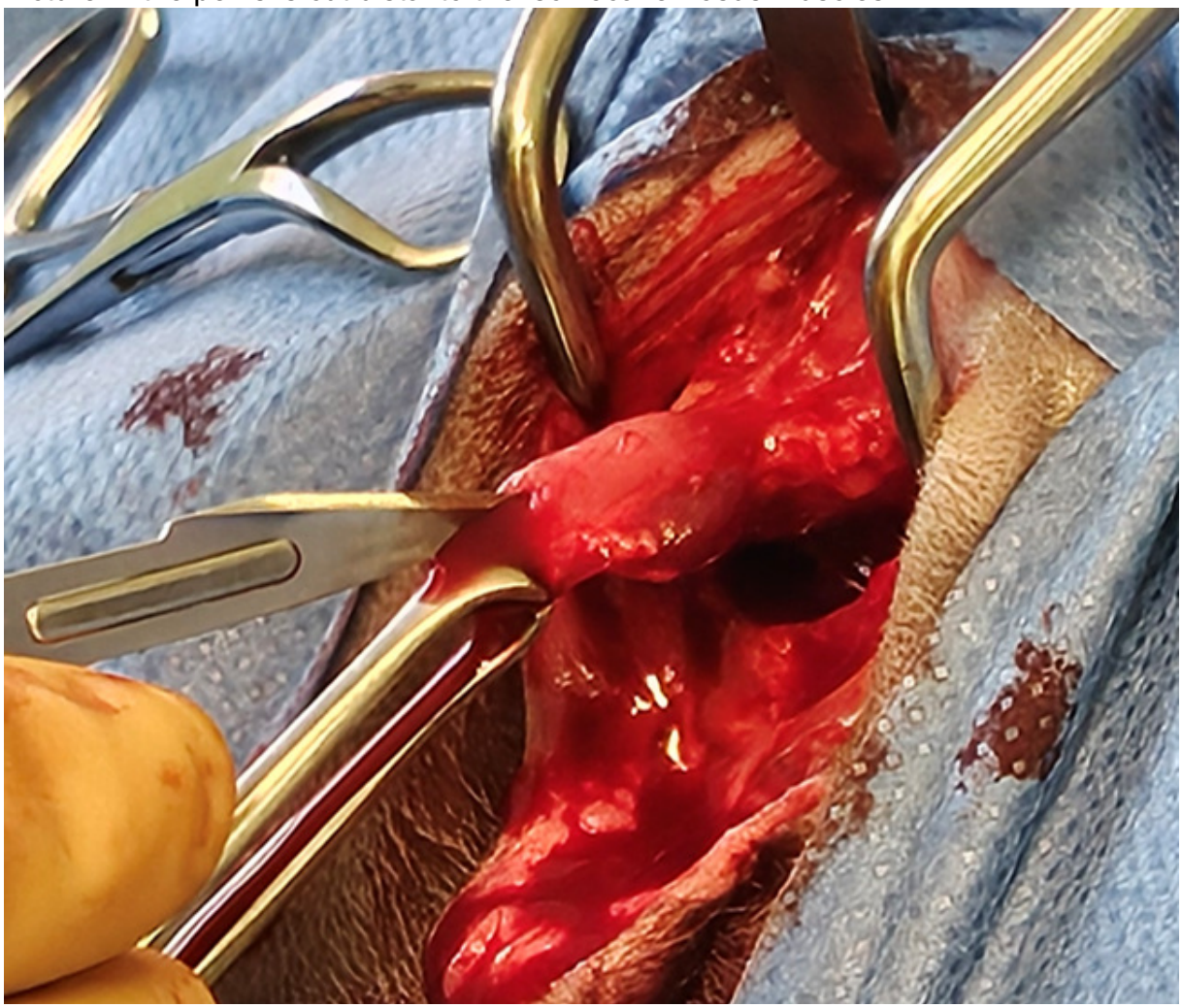
Picture 5: dissection of the urethra from the ventral pelvis



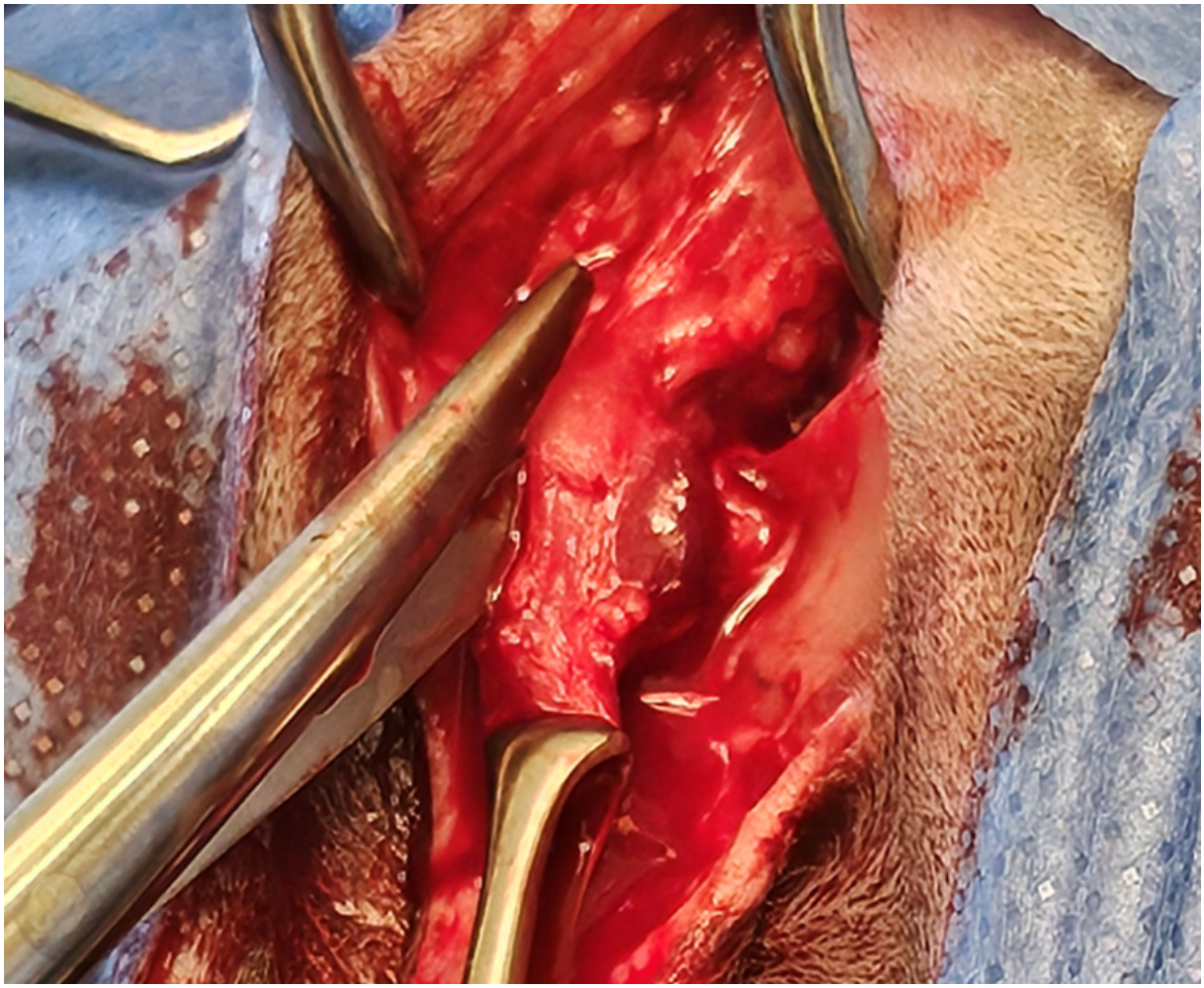
Picture 6: the ischio-cavernosus muscle have been cut, the bulbourethral glands are visualized (cross)



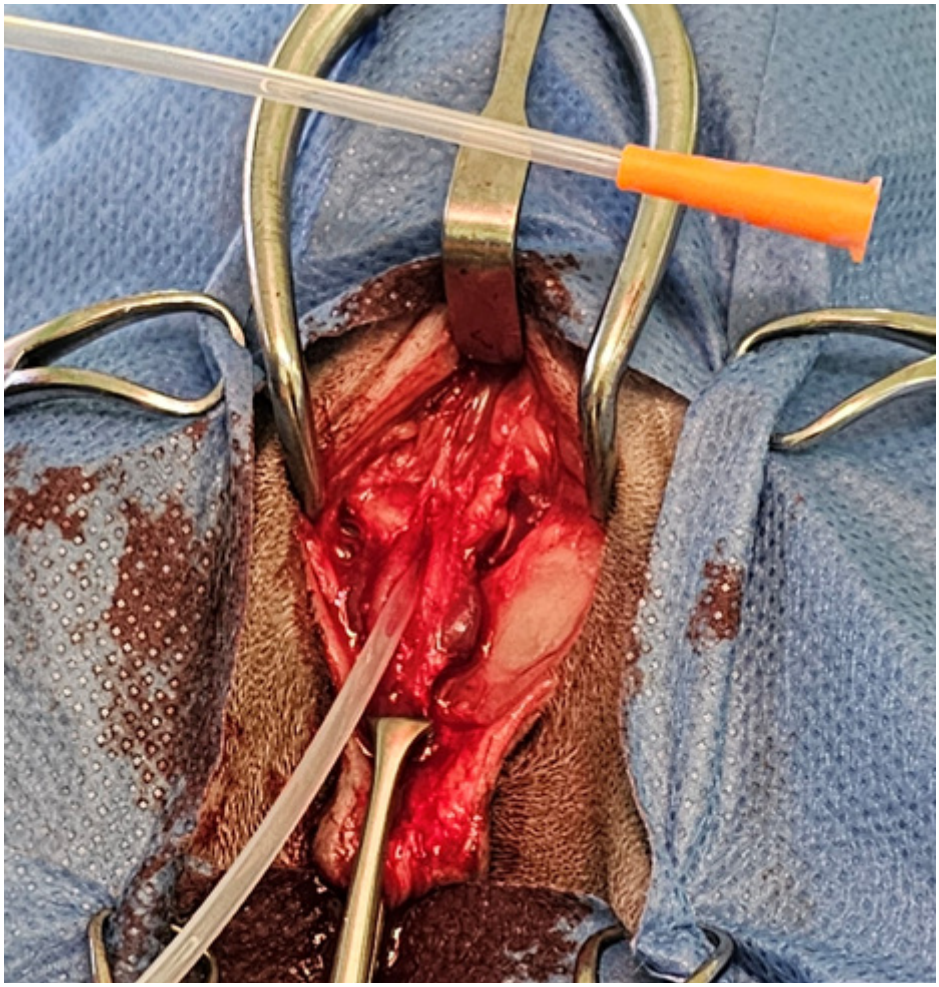
Picture 7: the penis is cut distal to the ischiocavernosus muscles



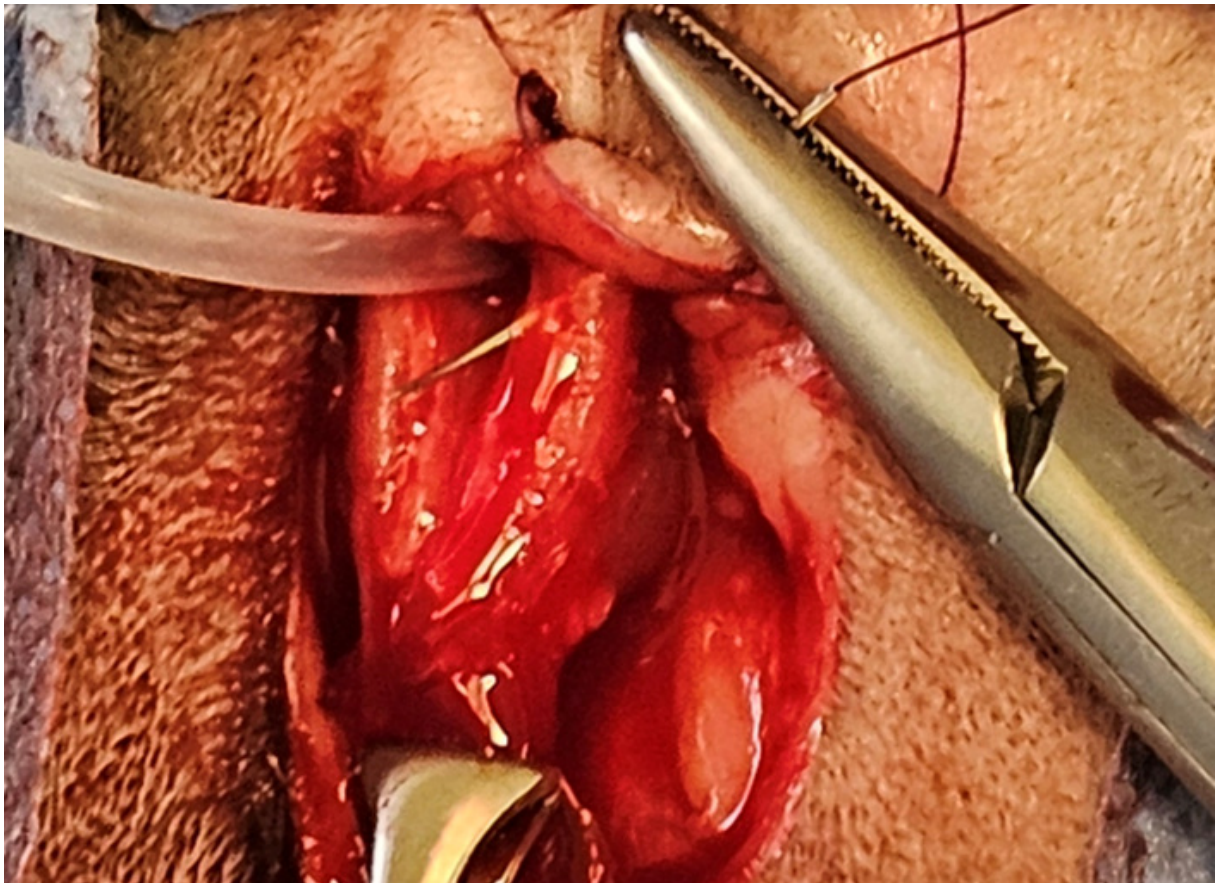
Picture 8: incision of the urethra with an 11 blade



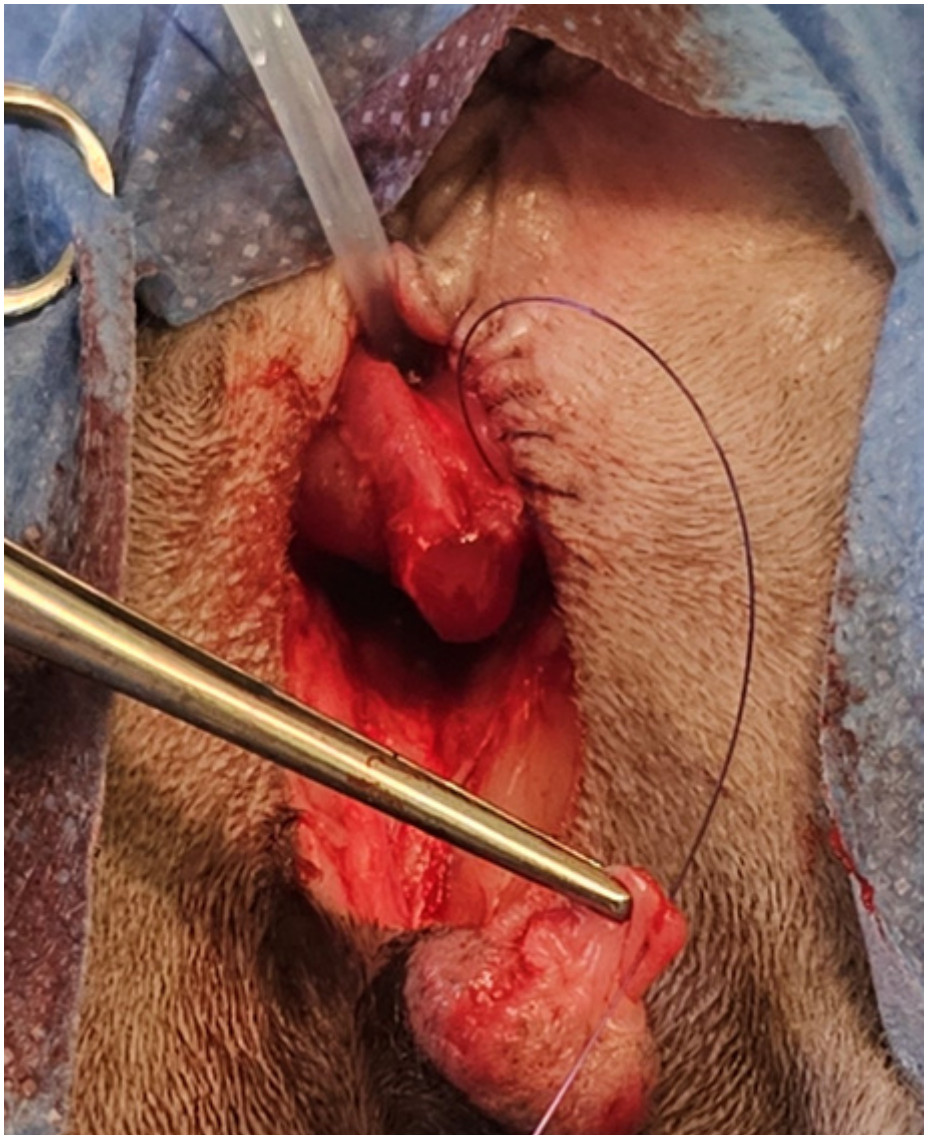
Picture 9: incision of the urethra with Metzenbaum scissors



Picture 10: The diameter of the urethra is checked by inserting an 8 Fr urinary catheter



Picture 11: The first continuous suture is placed between the dorsal wall of the urethra and the dorsal beginning of the skin incision. And the run on the right side.



Picture 12: the right border of the urothelium and the right corner of the preputial mucosa are sutured together



Picture 13: final result of the perineal urethrostomy with preservation of the preputial mucosa.

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Surgical exploration of abdominal cavity and abdominal biopsies

1. Introduction

Exploratory laparotomy is a systematic evaluation of the abdominal cavity. Despite major advances in imaging over the past two decades, which allow faster and more precise preoperative visualization, laparotomy remains indicated when lesions require biopsies that cannot be obtained by ultrasound-guided techniques or when imaging reveals warning signs such as focal steatitis. The presence of septic effusion or pneumoperitoneum confirms gastrointestinal peritonitis and justifies immediate surgical exploration.

2. Specific equipment

Essential instruments include self-retaining abdominal retractors (Gosset or Balfour), laparotomy sponges, and a Poole suction tip. To reduce abdominal wall trauma, a moistened sponge is placed between the retractor and the body wall and the retractor is periodically released. Suction facilitates removal of effusion and abdominal lavage. Sponges must be counted at the beginning and end of the surgery, or they may include a radiopaque marker woven into the fibers noticeable on radiograph postoperatively.

3. Patient preparation

A ventral midline celiotomy is performed with the patient in dorsal recumbency, clipped from the sternum to the pubis. In male dogs, a parapreputial approach is used caudally. After skin incision, subcutaneous tissues are dissected to expose the linea alba, which is opened from the xiphoid to the pubis. The falciform ligament is removed to improve cranial exposure and facilitate closure.

4. Exploration

Exploration must be exhaustive and performed in a consistent sequence to avoid omissions. A system-based approach is often preferred. Routine improves efficiency and diagnostic yield, although the order may be adapted to the suspected pathology.

a. Spleen, liver, biliary tract

The spleen is examined first because of its mobility; surgical biopsies are rarely required.

The liver is evaluated on its visceral surface. Surgical biopsies are indicated when percutaneous samples are insufficient for diagnosis or copper quantification. At least five samples from two lobes should be collected. Punch

biopsies (3–5 mm) taken perpendicular to the surface provide representative tissue to a depth of less than half the lobar thickness to avoid damaging large hepatic vein branches. Haemostasis is achieved with a square of absorbable gelatin sponge place in the biopsy site (pictures 1).
The extrahepatic biliary tract is inspected. The gallbladder is not biopsied, but bile may be aspirated for culture when infection is suspected, as it is more sensitive than hepatic culture. The gallbladder is emptied as completely as possible to prevent bile leakage from the needle insertion site.

b. Digestive tract and pancreas

The gastrointestinal tract is examined methodically from stomach to colon (or in reverse), palpating the cardia, stomach, duodenum, jejunum, ileum, cecum and colon while assessing mesenteric lymph nodes. The right limb of pancreas is examined while lifting the duodenum and the body of the pancreas is caudal to the pylorus. The omental bursa is opened to visualize the left limb of the pancreas, which is shorter and thicker.
Full-thickness surgical biopsies are indicated for diffuse or infiltrative disease, when endoscopic samples are inadequate, or when differentiation between inflammatory bowel disease and lymphoma is required. Biopsies are taken on the antimesenteric border using a longitudinal full-thickness incision and closed with a monofilament appositional pattern (pictures 2). Tru-Cut biopsies are faster and will give adequate material for diagnosis, the bowel is closed with a simple interrupted suture (pictures 3). Mesenteric lymph node sampling is performed by partial excision to avoid vascular injury of the mesenteric vessels (picture 4).
Pancreatic biopsies are obtained from the edges and the extremities of the lobes to avoid ducts and major vessels with a scalpel blade or cup biopsy forceps (picture 5); two samples are recommended because lesions may be focal.

c. Urinary tract and adrenal glands

The kidneys and ureters are retroperitoneal and exposed by reflecting the duodenum (right) or colon (left). The adrenal glands lie craniomedial to the kidneys, adjacent to the phrenicoabdominal vessels and caudal vena cava on the right. The bladder is intra-abdominal and attached by lateral and median ligaments.
Renal biopsies should include cortex only to avoid severe hemorrhage. Although most are performed percutaneously under ultrasound guidance, surgical biopsies using cup forceps allow controlled cortical sampling from the convex surface, one per kidney (picture 6).

d. **Reproductive tract**

The ovaries lie caudal to the kidneys within the ovarian bursa, which is closed in the dog and open in the cat. The uterus extends from the horns to the cervix, partly within the pelvic canal. The prostate surrounds the proximal urethra and

becomes progressively more abdominal with age. These organs are inspected and sampled when indicated.

e. Other considerations

The abdominal wall and diaphragm must always be examined before closure.

5. Minimally invasive surgery

Minimally invasive techniques, including laparoscopy and laparoscopic-assisted procedures, have become an important alternative to exploratory laparotomy for selected indications. They allow complete or targeted abdominal exploration with reduced surgical trauma, improved visualization due to magnification, decreased postoperative pain, shorter hospitalization, and faster return to function. In addition, the panoramic view of the cranial abdomen and the possibility of close inspection of specific organs often result in excellent diagnostic accuracy.

Diagnostic laparoscopy is particularly useful for evaluation of the liver, gallbladder, spleen, pancreas, abdominal lymph nodes, kidneys, adrenal glands, reproductive tract, and peritoneal surfaces. It enables targeted biopsies under direct visualization while avoiding the morbidity associated with a full celiotomy. Hepatic biopsies obtained laparoscopically are larger and less prone to crush artefact than percutaneous samples and allow reliable histopathology and copper quantification. Laparoscopic biopsy of abdominal lymph nodes and adrenal glands is also well described.

However, minimally invasive exploration has limitations. Palpation of the entire gastrointestinal tract is not possible, which reduces sensitivity for small mural lesions, foreign bodies, or focal infiltrative disease. Full-thickness intestinal biopsies can be performed using laparoscopic-assisted exteriorization of the bowel, but this requires advanced equipment and surgical expertise. In cases of septic peritonitis, hemodynamic instability, large masses, severe adhesions, or when rapid therapeutic intervention is required, open exploratory laparotomy remains the procedure of choice.

Patient selection is therefore critical. Minimally invasive approaches are best suited for stable patients requiring targeted biopsies or staging procedures, whereas open surgery is preferred when diffuse disease, obstruction, perforation, or the need for extensive manipulation is suspected. Rather than competing techniques, laparoscopy and exploratory laparotomy should be viewed as complementary, and the choice depends on the clinical question, expected diagnostic yield, patient stability, available equipment, and surgical experience.

6. Conclusion

Exploratory laparotomy remains an essential complementary procedure to advanced imaging, allowing systematic abdominal evaluation and collection of

high-quality full-thickness or organ biopsies that frequently provide a definitive diagnosis when minimally invasive techniques are inconclusive. Its success relies on a consistent exploration routine, precise anatomical knowledge, appropriate instrumentation, and meticulous tissue handling to maximize diagnostic yield while minimizing morbidity

To Read:

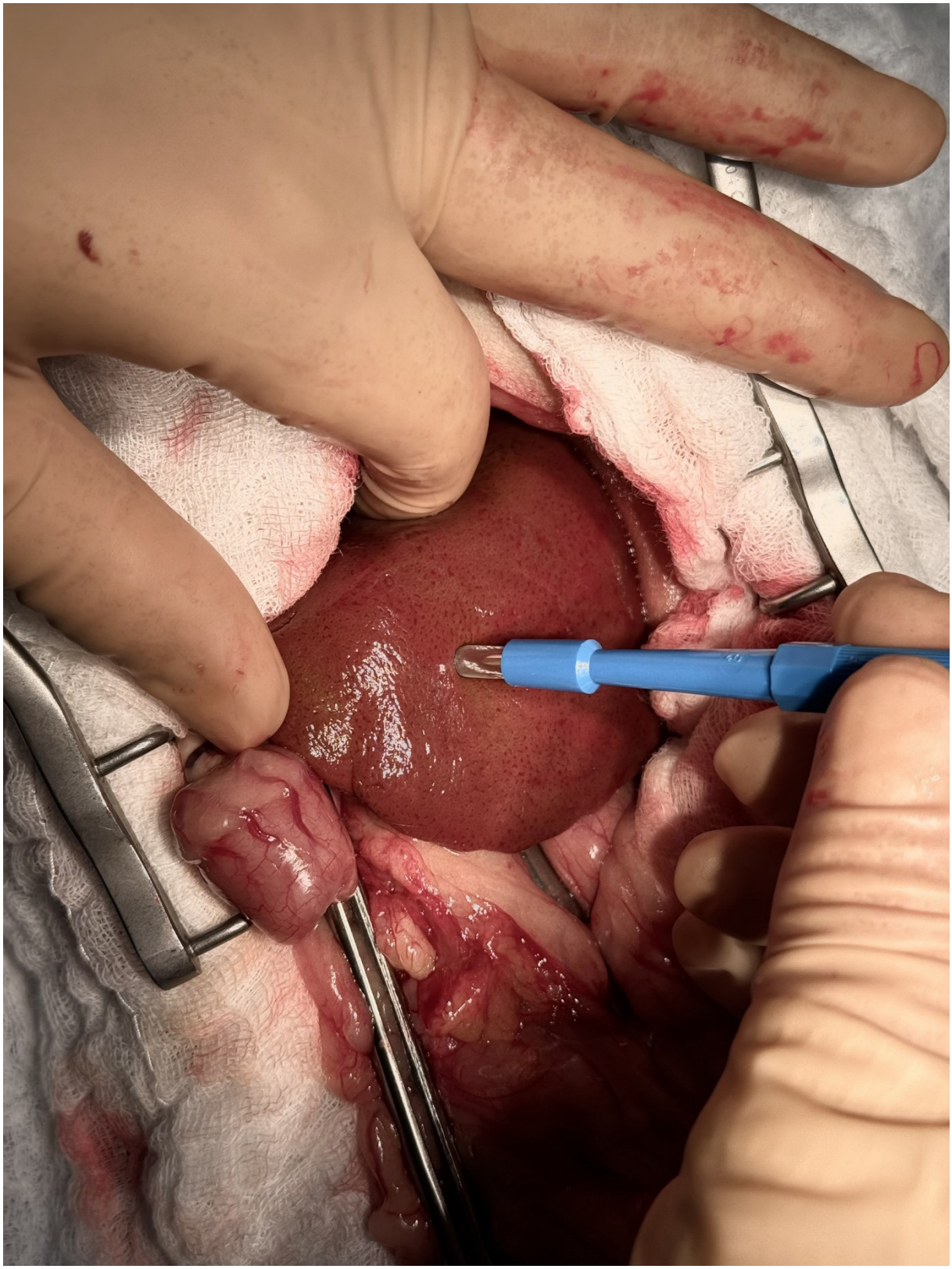
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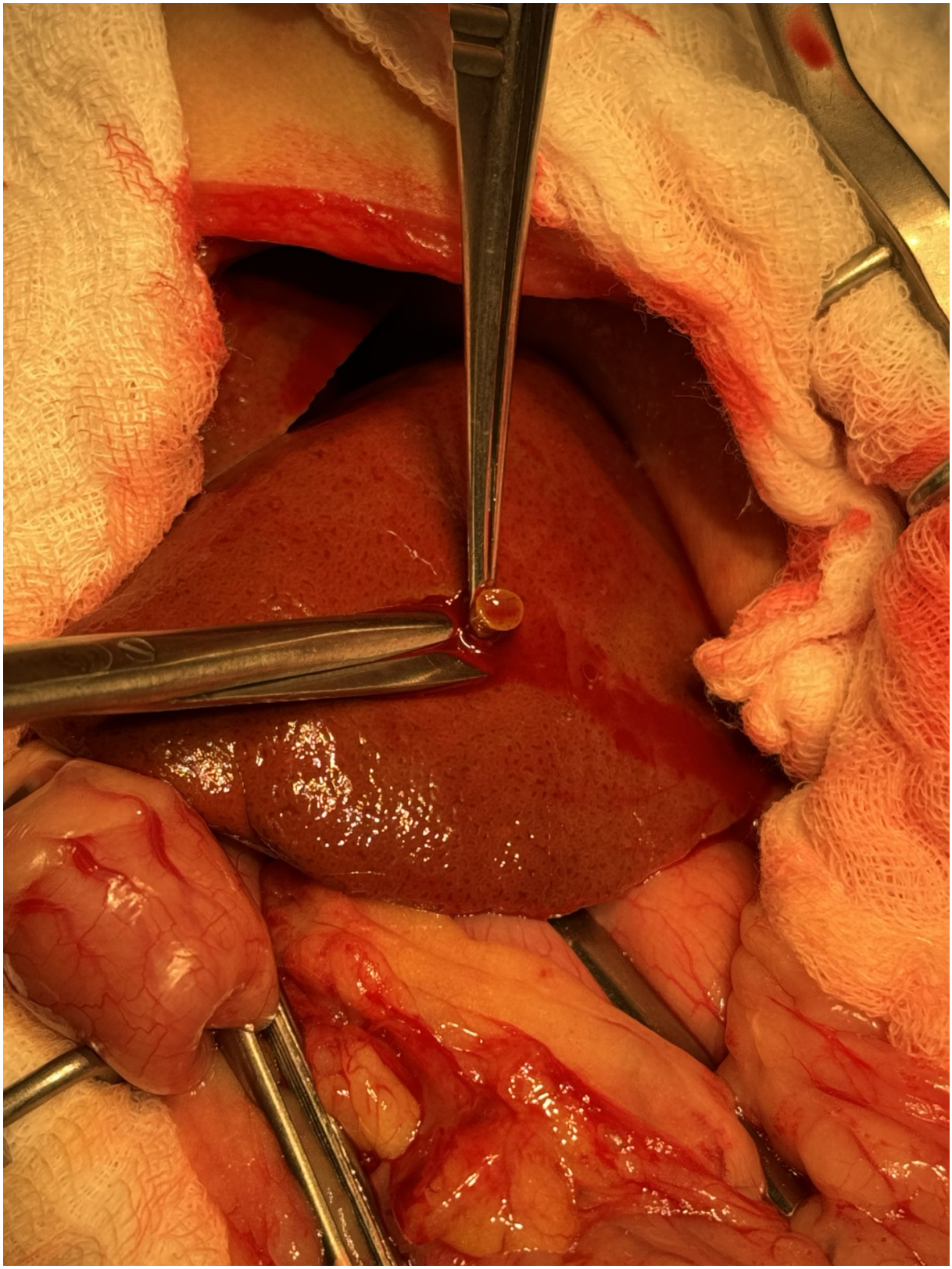
Picture 1. Open surgical liver biopsy.
1a The biopsy punch is inserted perpendicular to the surface of the lobe to a depth of less than half the lobar thickness to avoid damaging large hepatic vein branches.
1b The sample is retrieved by gently lifting it and transecting its base with Metzenbaum scissors.
1c A square of gelatin sponge is prepared.

1d The square of gelatin sponge is placed into the biopsy defectIt shrinks as it fills with blood.

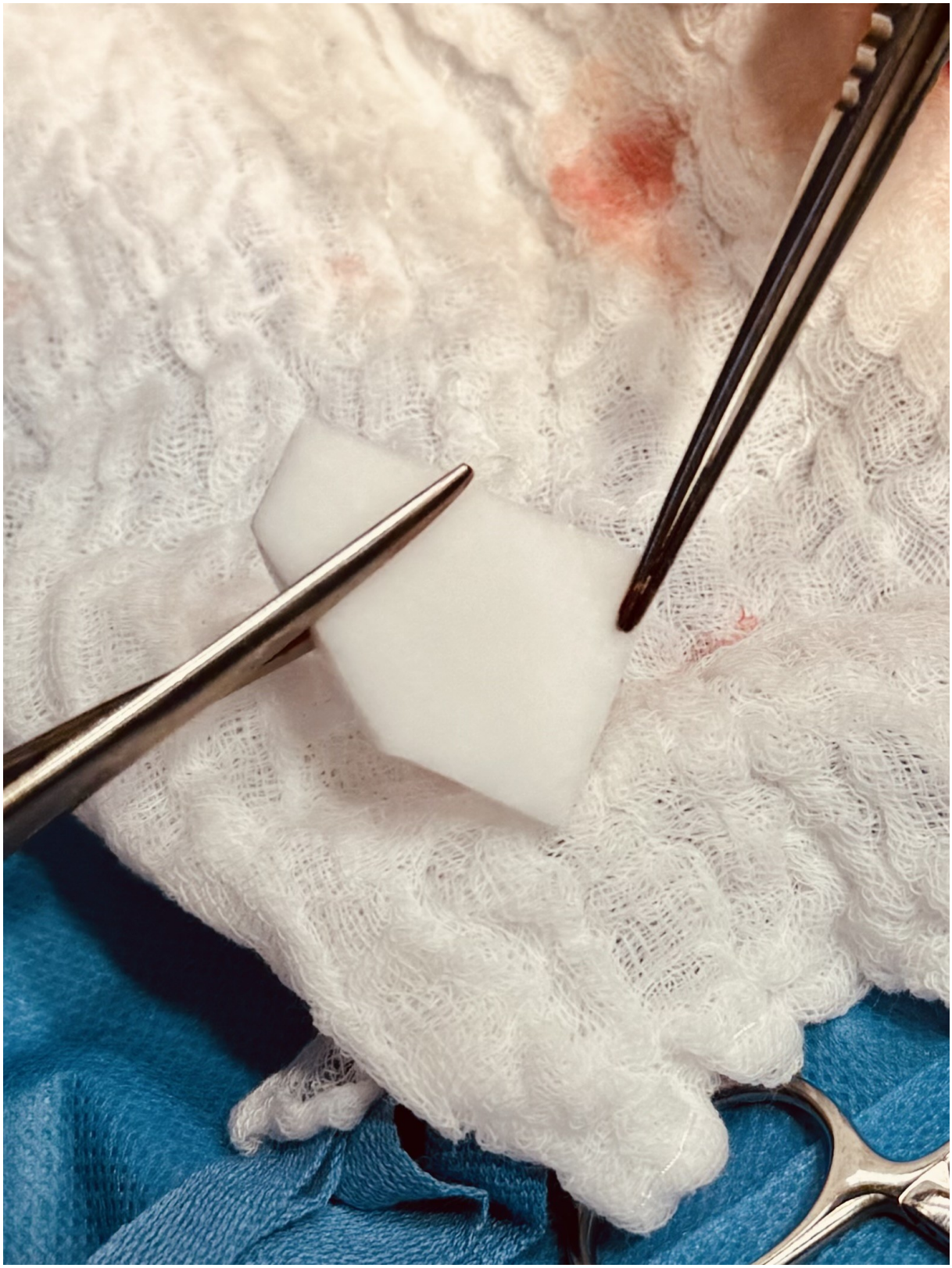
1e The square of gelatin sponge decreases in size as it becomes engorged with blood.



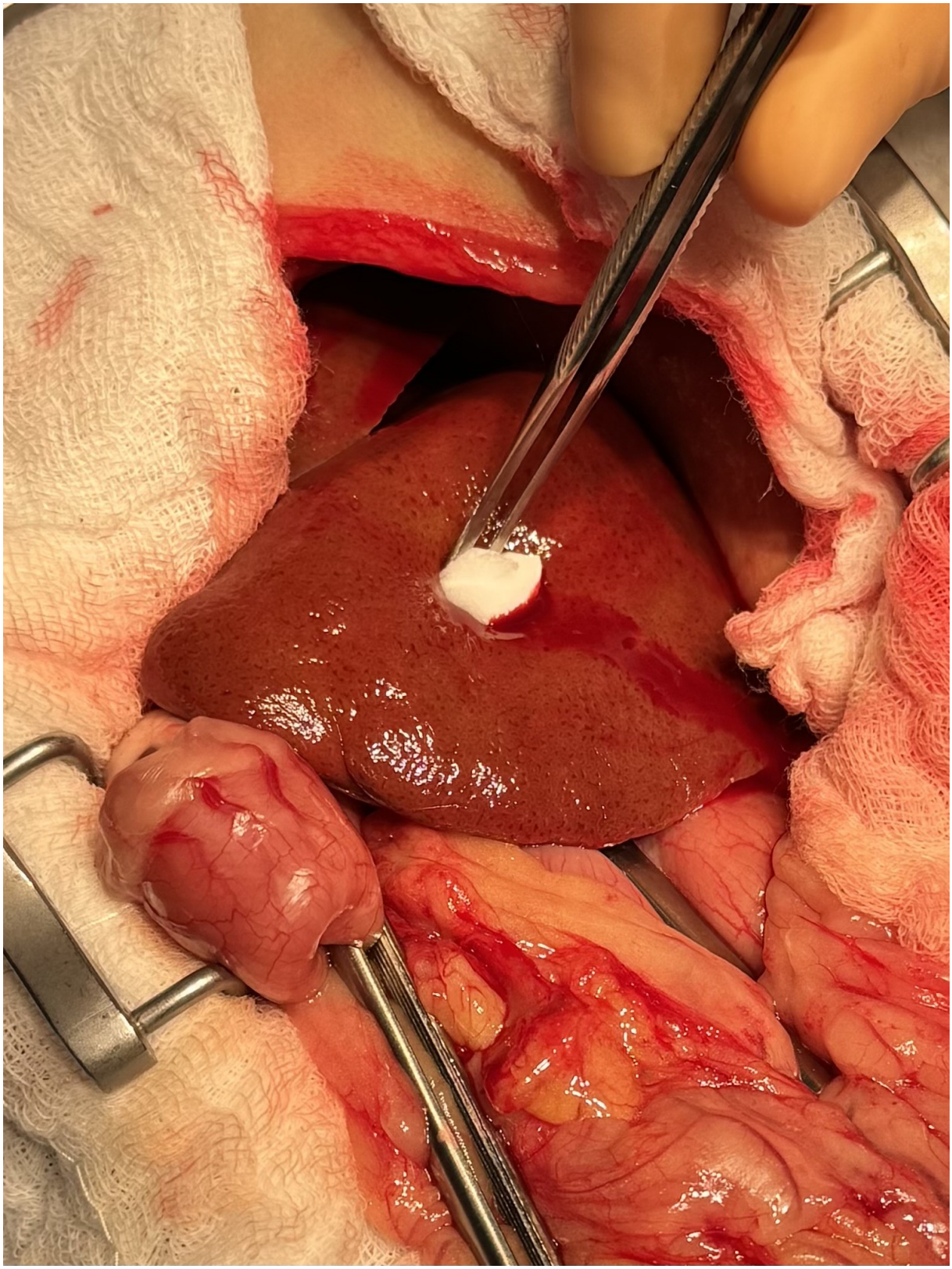
1a



1b



1c



1d

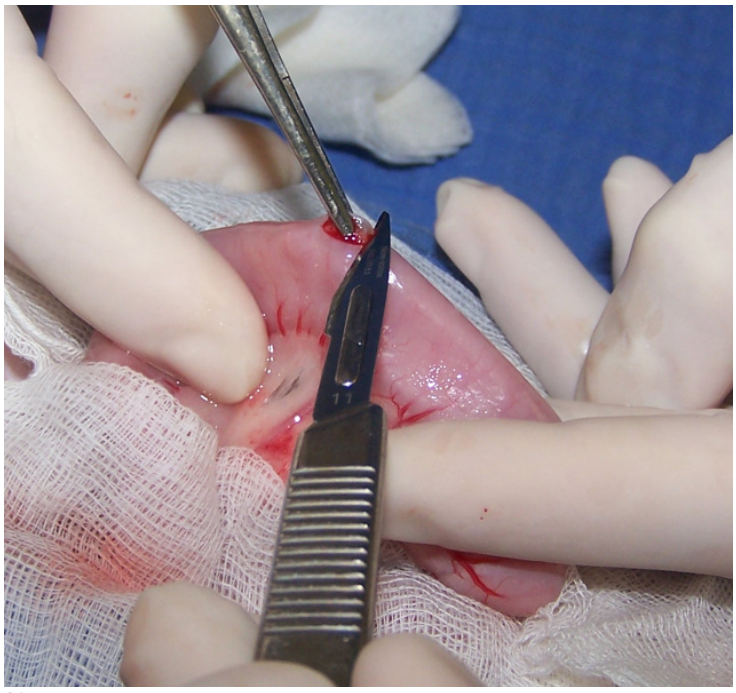


1e

Pictures 2: small intestinal biopsy using an 11 blade
2a a longitudinal incision is made through the full thickness of the wall of the intestine
2b the wall is lifted with a Debay thumb forceps and another longitudinal incision is made to get the full thickness biopsy
2c the biopsy site is close using a simple continuous suture with a monofilament resorbable suture starting and ending outside the incision site



2a

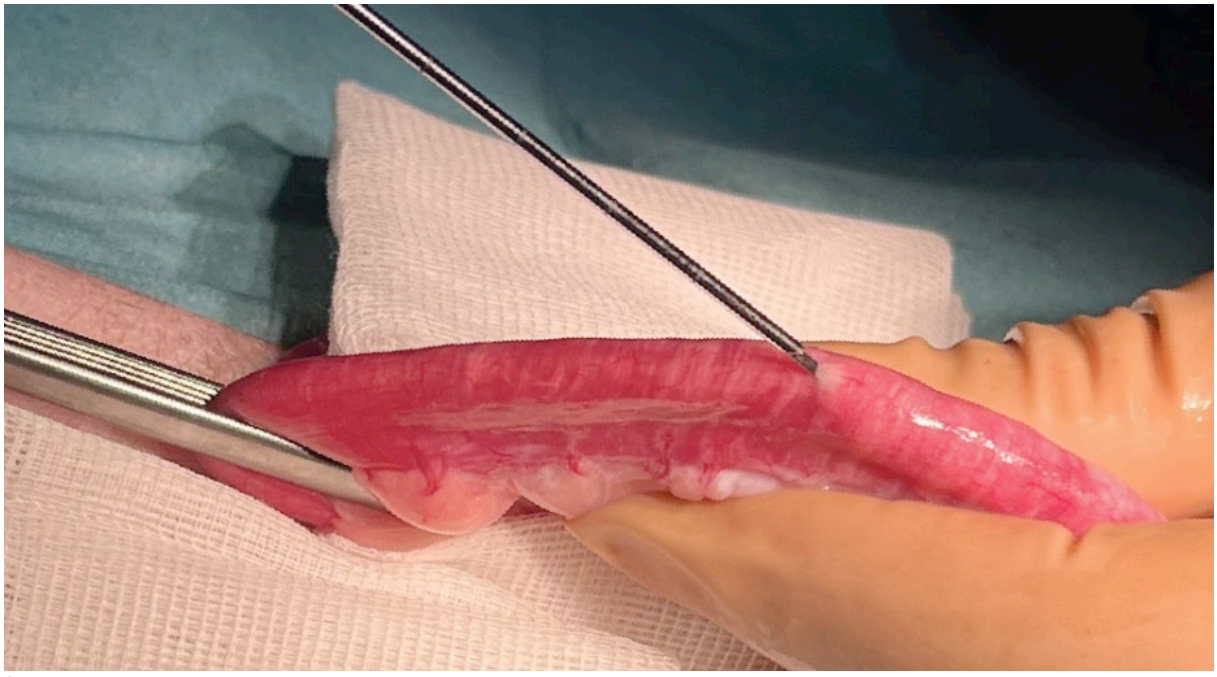


2b

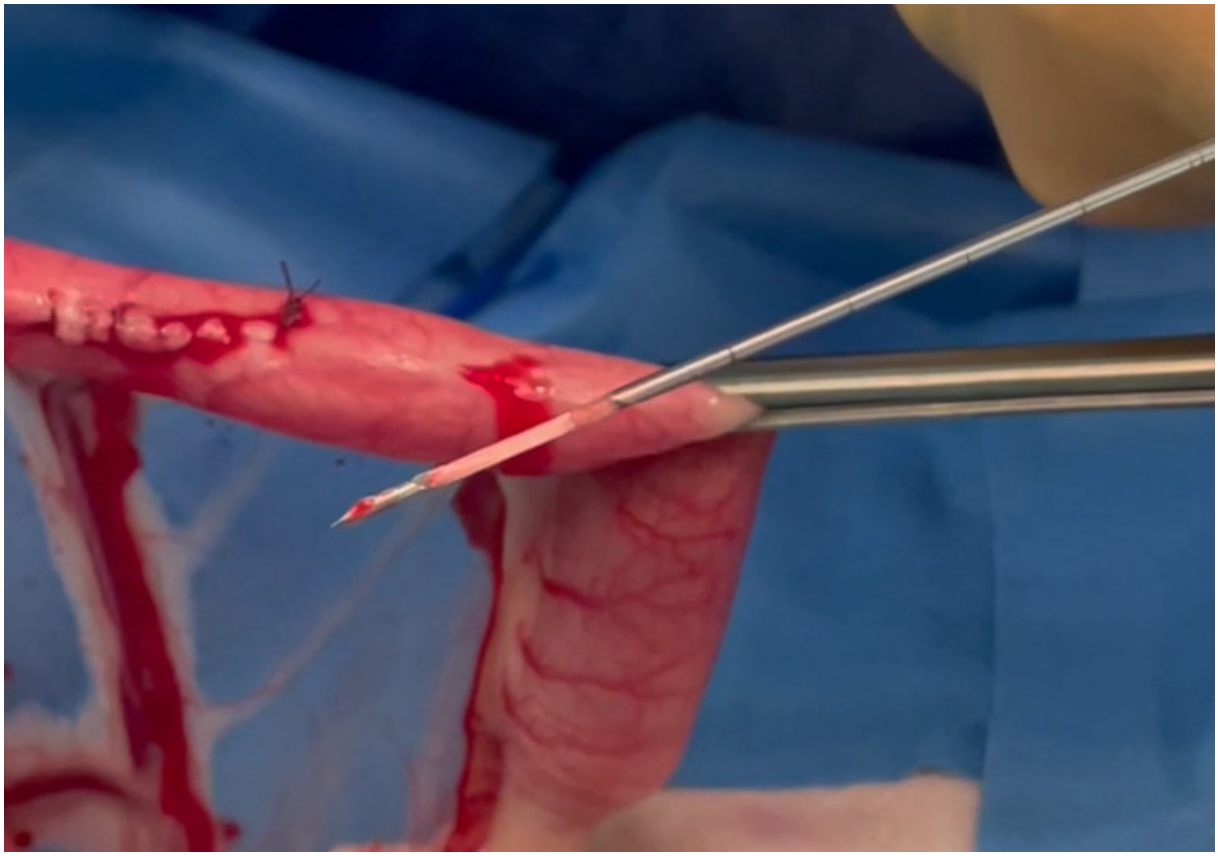


2c

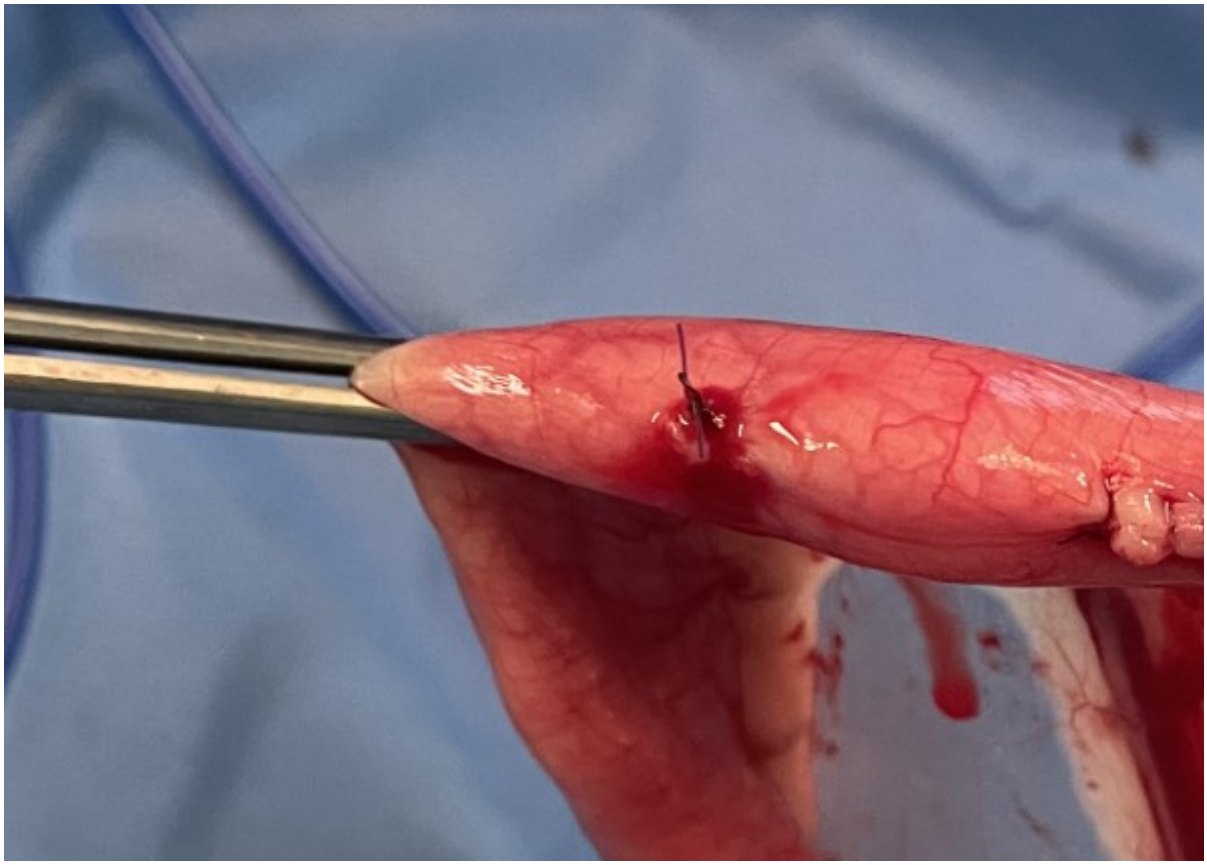
Pictures 3: Intestinal full-thickness needle-core biopsy
3a A 16-gauge Tru-Cut biopsy instrument is opened and introduced into the intestinal lumen at a tangential angle.
3b The Tru-Cut biopsy instrument is fired, removed from the intestine, and opened to get the sample
3c A simple full thickness interrupted resorbable suture is placed



3a



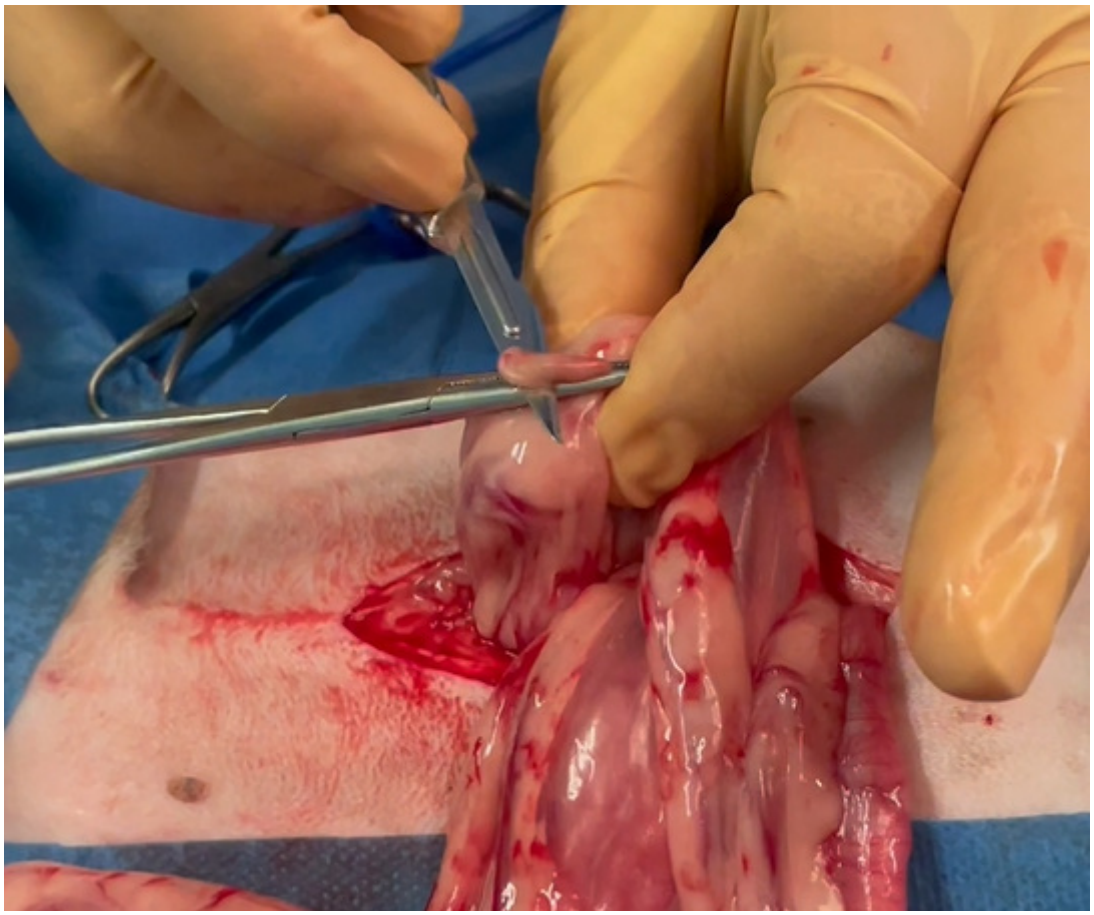
3b



3c

Picture 4: partial mesenteric lymph node biopsy

4a A hemostatic clamp is placed on the third of the superficial end of the mesenteric lymph node. Using an 11 blade, the superficial part of the lymph node is excised.
Ab A cross resorbable suture is placed to close the defect

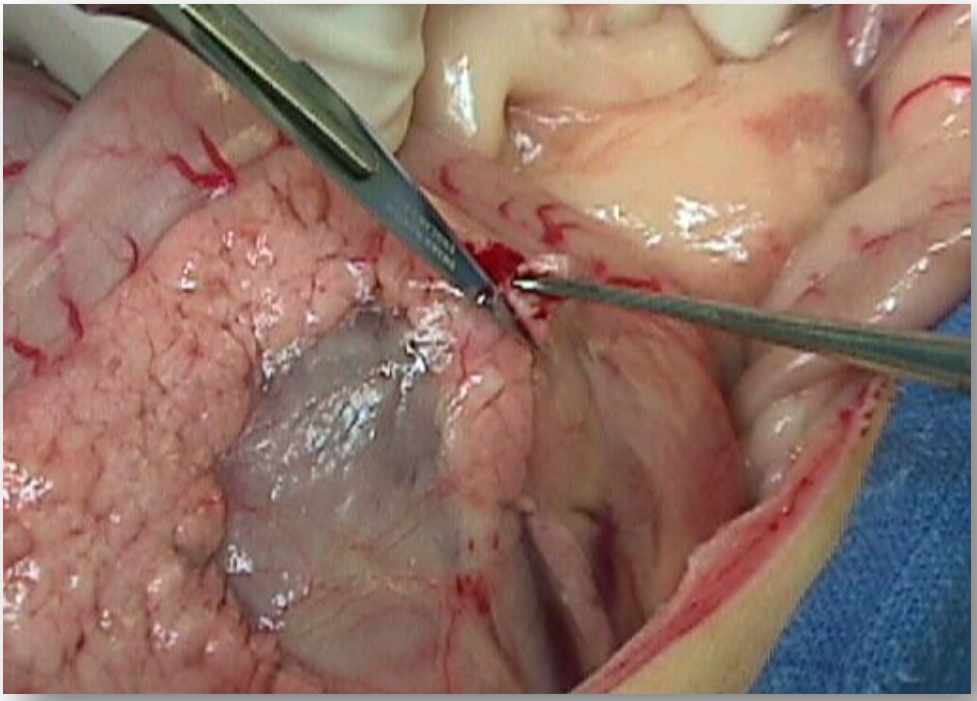


4a

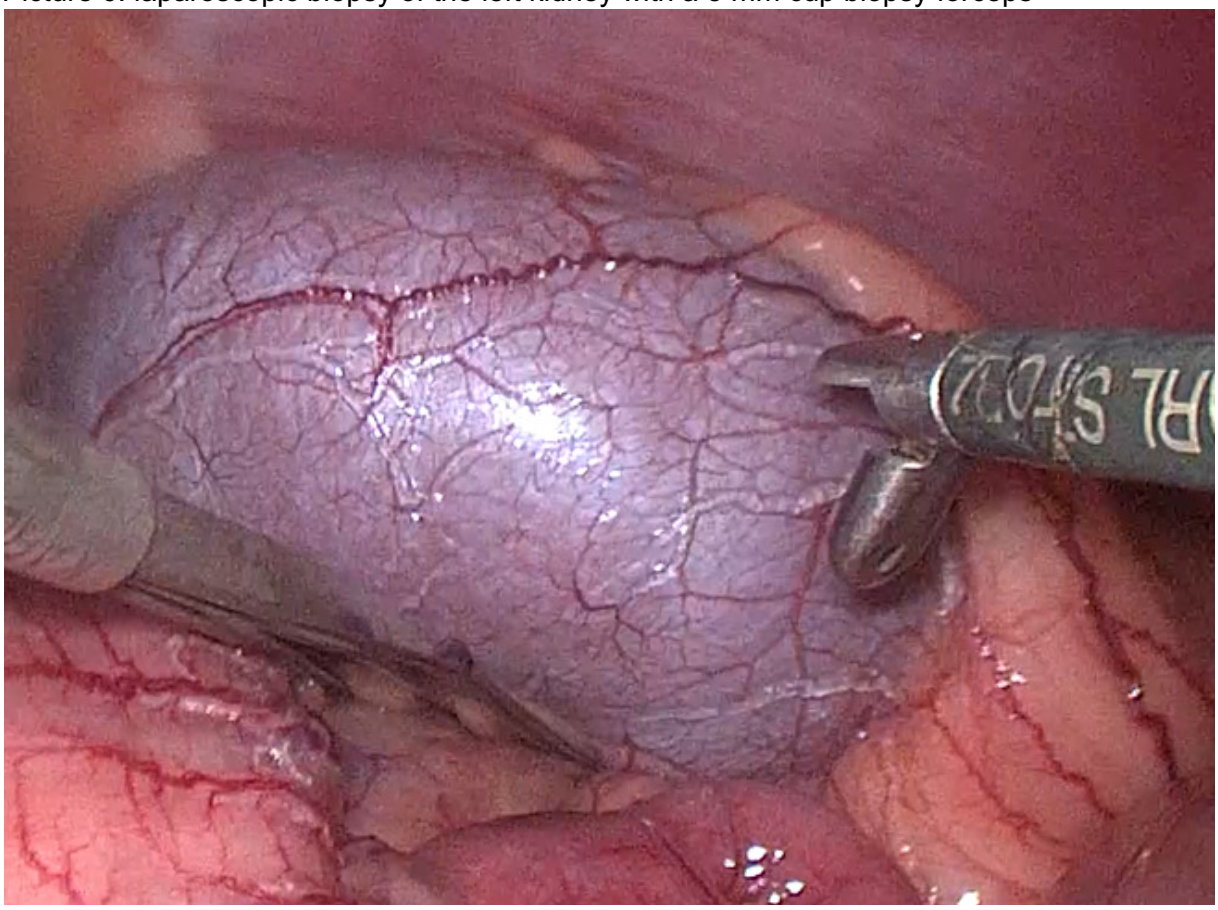


4b

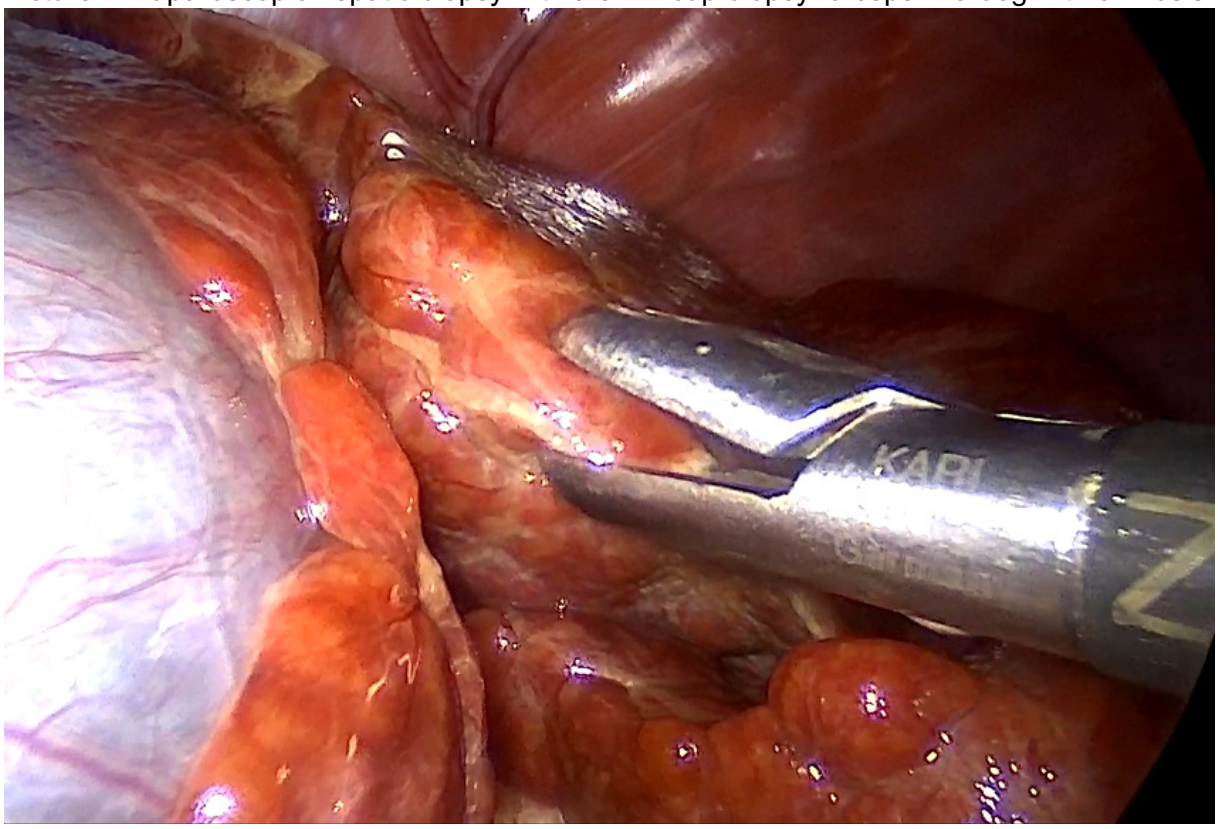
Picture 5: peripheral biopsy of the right limb of the pancreas



Picture 6: laparoscopic biopsy of the left kidney with a 5 mm cup biopsy forceps



Picture 7: Laparoscopic hepatic biopsy with a 5mm cup biopsy forceps in a dog with cirrhosis



Neuro-clinical reasoning - a case-based approach

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Neurology is the Queen discipline of clinical problem-solving. Logically and systematically thinking can help you narrow down the differential diagnosis list to a handful of possibilities. Advanced imaging is useful for verifying the location of a lesion but should not be used to localise it.

“Pattern recognition” is not inherently bad, but how it’s often done is problematic. Pattern recognition is often based on two or three factors, which is unreliable. Key factors often used are breed, age, and one clinical sign. Before the patient is seen, you might already have made a diagnosis, making it difficult to be open-minded during the consult. For example, a Boxer, 8 years old, with a behavioural change could be diagnosed with a brain tumour based on just two or three factors. However, using nine or more factors increases the chances of getting the right diagnosis to 99%. This level of expertise and up-to-date knowledge is required to achieve this.

“Well, I do some bloods” can help solve a clinical problem, but it can also be a fishing exercise if you don’t know what you’re looking for. Many neurological diseases, some of which are life-threatening, may not result in significant blood changes. What then is your plan B? MRI?

Having an intellectual framework for working up a case in a structured problem-based approach will help you solve “not-as-straightforward” cases and advance your knowledge.

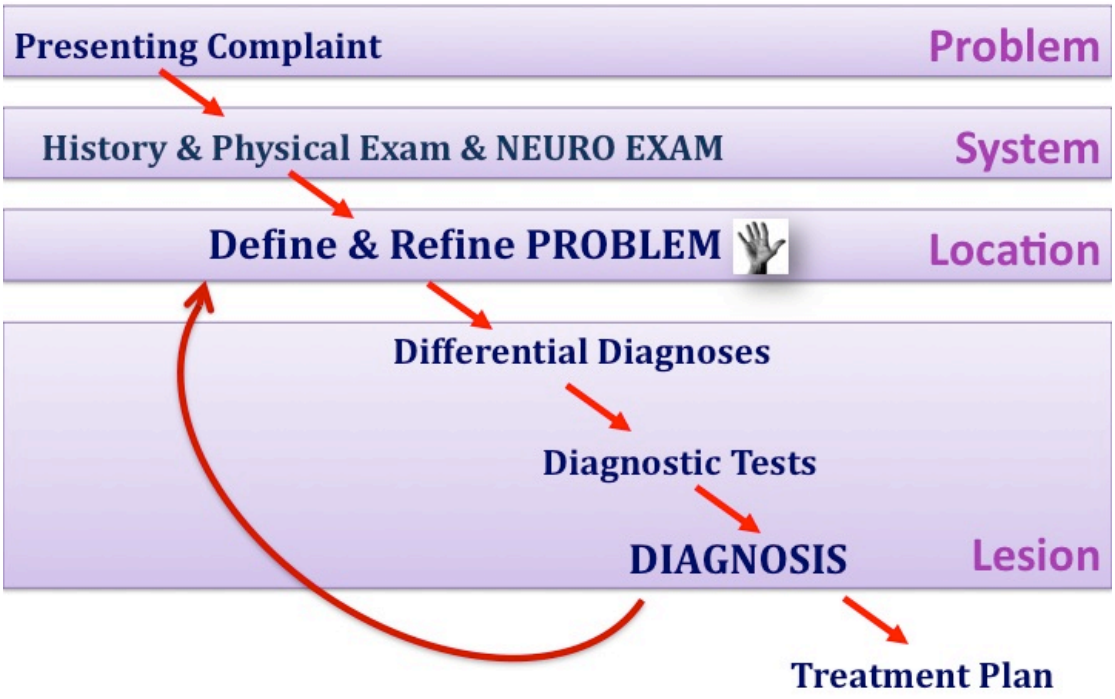
Construct a problem list then….

2. Define the problem

3. Define the system (and how it may be involved)

4. Define the location within the system

5. Define the lesion



Constructing a problem list

A problem list:

- Helps to define clearly the clinical signs

- Helps prevent overlooking less obvious but nevertheless crucial clinical signs
- Helps clarify the temporal relationship and if there is only one or multiple problem(s) in the animal

Define and refine the problem

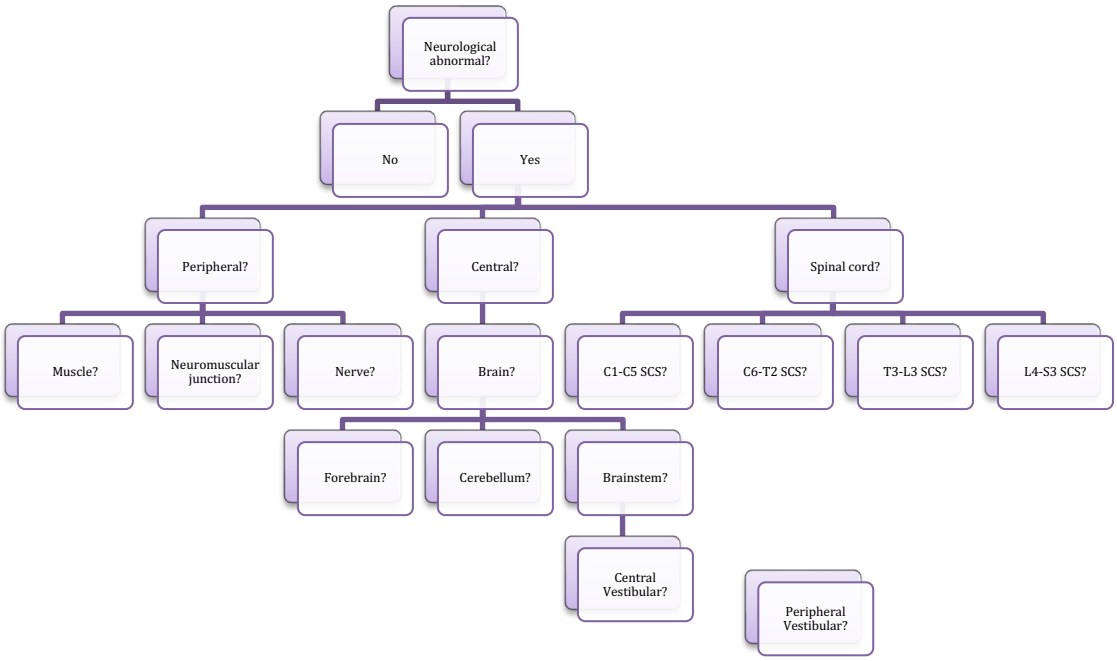
When considering the important clinical signs the patient is exhibiting, it is essential to try to define the problem as accurately as possible. “A problem well defined is a problem half solved” is a good motto to work from. Try to define the problem at the highest level of your understanding at the time.

Define the body system and how is it affected

Once the problem is defined, the next step is usually to consider the systems involved. The key question is: “What systems could be causing this clinical sign?” or “Do I have a primary, structural problem of a body system, or a secondary, functional problem where the system involved is affected by other factors?” For example, seizures can be caused by intracranial or extracranial causes, and the body system involved is the forebrain.

Define the location within the system

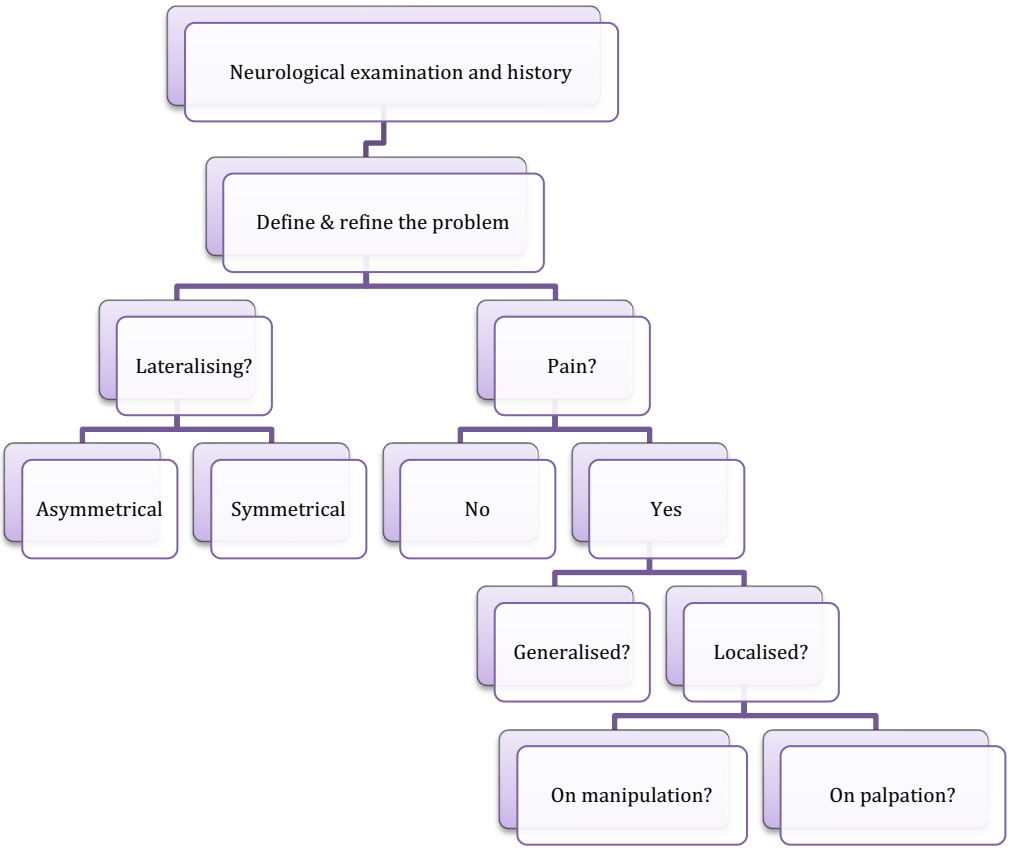
If you suspect the neurological system may be involved, a neurological examination is necessary to pinpoint the lesion more precisely within the nervous system. Below is a flow chart to aid in this process. Peripheral vestibular is a separate box because it can only be localised by ruling out brainstem signs, which would indicate a central vestibular problem.

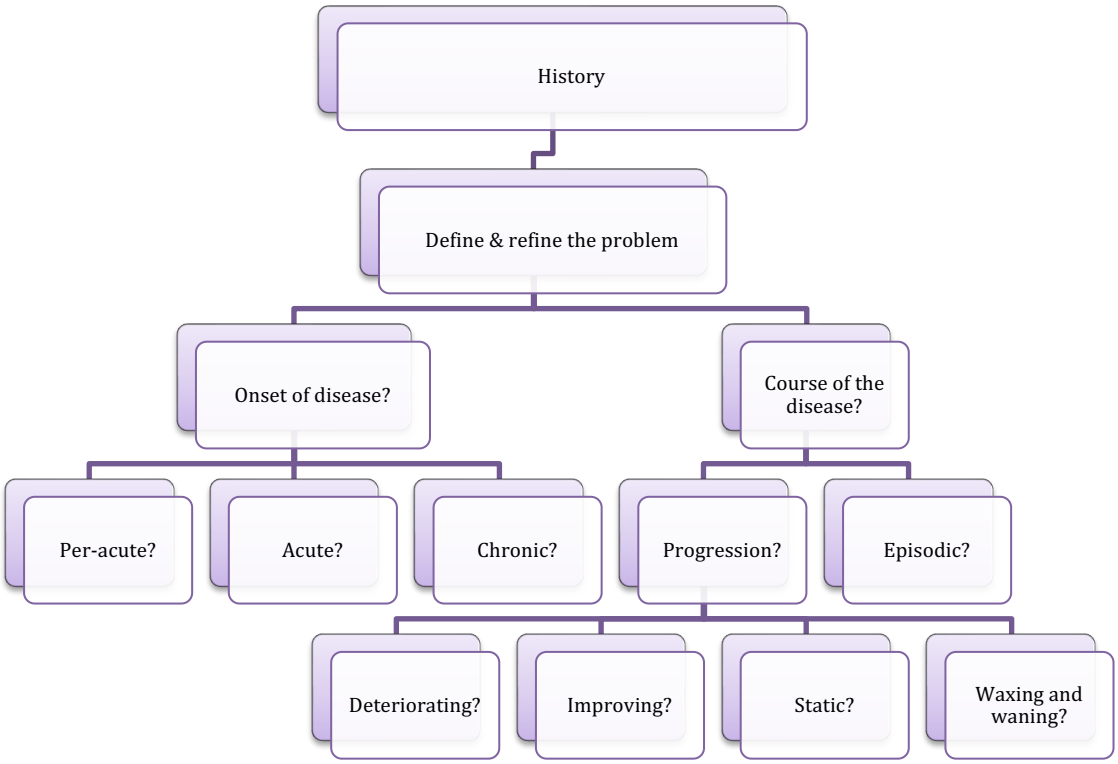


Define and refine the lesion

Once the location of a problem within a body system is determined, the next key question is "What is it?" This involves defining the pathology. It can be helpful to remember the broad types of pathology that can occur using the DAMNIT-V scheme (see details below): degeneration, anomaly, metabolic, neoplasia/nutritional, inflammatory/infectious/idiopathic, trauma/toxic, or vascular. The type of pathology most likely to occur will depend on the body system or organ involved, the clinical course of the disease presentation, symmetry of the deficits, pain involvement, and the patient's signalment (species, breed, age, sex, etc.). Additionally, the geographic location of the patient and what's common in that population should be considered.

Below are some flow charts that can help you define your problem as precisely as possible to create a differential list of lesion possibilities before considering further diagnostics.


Neurological examination and history
Define & refine the problem
Lateralising?
Pain?
Asymmetrical
Symmetrical
No
Yes
Generalised?
Localised?
On manipulation?
On palpation?


History
Define & refine the problem
Onset of disease?
Course of the disease?
Per-acute?
Acute?
Chronic?
Progression?
Episodic?
Deteriorating?
Improving?
Static?
Waxing and waning?

I found using the five-finger ("Neuro-hand-rule") rule very helpful in clinical problem-solving. It helps me to consider the onset, clinical course, pain, lateralisation, and neurological localisation of a patient's symptoms. For example, I might use the rule to diagnose acute onset, progressive, painful, asymmetrical T3-L3 myelopathy, or chronic deteriorating, non-painful, asymmetrical forebrain disorder. Once I've determined the problem using the five-finger rule, I can then use the signalment to refine my diagnosis further.

Now, think pathophysiologically using the DAMNIT-V Scheme (use it to un-lock your memory potential):

1. Degenerative
2. Anomaly
3. Metabolic
4. Neoplastic, Nutritional
5. Inflammatory, Infectious, Idiopathic
6. Trauma, Toxic
7. Vascular

1. Which of the disease processes listed above cause **lateralising (asymmetrical) signs?** Always? Any exceptions?
2. Which of them cause **pain?** Always? Any exceptions?
3. What about **onset and course of disease**?

After you have come up with your main differentials think about which diagnostic test to perform and why.

Diagnostic tests (see lecture for details):

1. Clinical pathology
2. Diagnostic imaging
3. Functional assessment

Seizures -Epilepsy or Not?

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A patient with a history of paroxysmal episodes, or 'fits', can be challenging for experienced clinicians. They usually appear normal during the presentation, and identifying the episode type relies heavily on eyewitness descriptions. These episodes are unpredictable and uncontrollable for the owner, leading to biased observations. However, obtaining a comprehensive patient history is crucial before diagnosis. Many owners now record these events on video, which can aid characterisation.

Paroxysmal events like syncope, narcolepsy/cataplexy, pain, compulsive behaviour disorders, vestibular attacks, certain movement disorders, neuromuscular weakness, and seizures share common clinical presentations. The inter-episodic clinical examination may be normal, but some animals may exhibit deficits, providing guidance for decision-making and localisation. Occasionally, animals may manifest these episodes at the clinic, such as prolonged seizures (status epilepticus >10 minutes), cluster seizures (≥2 seizures/day), or vestibular dysfunction.

Define the problem

Paroxysmal episodic disorders can affect posture, muscle tone, cause uncontrolled movements, and alter behaviour. Identifying triggers or clinical signs before or shortly after episodes is crucial (Table 1). Common episodic events requiring differentiation include syncope, narcolepsy/cataplexy, behaviour changes, vestibular attacks, movement disorders, neuromuscular weakness, and seizures.

When an animal presents with a history of episodic weakness, fatigue, or collapse, it's challenging to define the problem accurately. Owners may describe these episodes as "collapsing," but clinicians must gather key information to precisely define the issue.

- What happens before or after the episode?
- What was observed during the episode?
- Does the animal lose consciousness during the episode? (Seen in syncope or seizures.)

This will enable the clinician to ascertain whether:

- The animal loses **consciousness** (indicating syncope or seizures).
- There is NO evidence of **convulsive** activity (more likely syncope than seizures).
- The animal is normal in between the episodes (**episodically weak)**, whether weakness is precipitated by exercise (**fatigability**) or the animal is consistently weak (**persistently weak)**.
- The animal shows a **spastic** or **flaccid** 'weakness' associated with or without incoordination (**ataxia**).

Other common presenting complaints that may be seen in the flaccidly weak patient include:

- Regurgitation
- Paresis
- Difficulty rising
- Exercise intolerance
- Episodic weakness
- Fatigability
- Altered voice
- Change in musculature
- Stiff stilted gait
- An inability to lift the head up normally or, especially in cats, a state of persistent
- cervical ventroflexion

Syncope

Syncope is a sudden, brief loss of consciousness and posture. Animals become flaccid but may jerk before collapsing. It's common in cats with 3rd degree atrioventricular block and can be mistaken for seizures. Most animals don't show signs before or after an episode.

Syncope often happens when animals move, not when they're resting. Recovery is usually quick. Some animals have multiple episodes in a day, which may not respond to anti-seizure drugs. In fact, these drugs can worsen the episodes.

Paroxysmal Behaviour Changes

Episodic pain can cause behavioural responses similar to focal seizures, such as nerve root impingement or disc protrusion/extrusion. This can lead to 'freezing', jerks, spasms, or fasciculations. Similarly, behaviour disorders like aggression or compulsive changes (e.g., rhythmic pacing, licking, vocalisations) may resemble sensory seizures. Dogs and cats usually return to normal between episodes, but

compulsive changes aren't linked to muscle tone or consciousness, and there's usually a clear trigger.

Vestibular Episodes

Transient vestibular episodes are a rare phenomenon characterized by the same cardinal signs as non-intermittent vestibular disease, including head tilt, nystagmus, and ataxia. While nystagmus and gait abnormalities can also be seen in seizures, it is uncommon for a seizure to cause a head tilt. Patients experiencing transient vestibular episodes typically do not exhibit altered consciousness during an episode and return to a fairly normal state before and after the episode. These episodes do not respond to standard antiepileptic drug treatment.

Paroxysmal Movement Disorders

Our understanding of and ability to identify paroxysmal movement disorders has improved in the last decade. These disorders are triggered or worsened by excitement or stress and are usually associated with movement, rarely occurring at rest or sleep. They are episodic, involving an increase in muscle tone (dystonia) but not affecting consciousness. Some were previously mistaken for seizures but don't respond well to standard antiepileptic drugs. They resemble human movement conditions and are now classified as movement disorders. Some have even been genetically characterised.

- When presented with a purebred dog experiencing a paroxysmal episode without autonomic dysfunction, normal recovery, non-generalised tonic-clonic seizure, appropriate mentation during the episode, even with bilateral muscle activity changes, and poor response to antiepileptic drugs, consulting breed-specific movement disorder databases is advisable.
- These disorders usually lack:
  - An identifiable precipitating event like an aura (sensory seizure activity, such as behaviour change, lasting a couple of minutes before the motor seizure).
  - Autonomic signs (e.g. hypersalivation, urination, defaecation).
  - Generalisation of increased motor activity (e.g. generalised tonic or tonic-clonic seizure).
  - Impairment of consciousness. Dogs with impaired consciousness won't look in the owner's eyes during the event, which is a good question to ask the owner. Animals will also often not listen to the owner due to the impairment of consciousness, though this is often falsely underreported due to the owner's perception of the event.

Seizures

The brain is a complex structure with limited ways of expressing dysfunction. Seizures are clinical signs caused by forebrain dysfunction and are not specific diseases. Various structural and functional causes can lead to seizures. Their forms

depend on the affected brain areas, e.g., a specific part of the sensory cortex can cause behavioural changes, while a part of the motor cortex can cause oro-facial automatisms. The location of the 'symptomatogenic' zone usually overlaps or is close to the epileptogenic zone, indicating the seizure's origin. Seizure semiology, using clinical signs of cerebral dysfunctions, confirms the event as a seizure and provides information about its origin. It's simple, clinically effective, and differentiates motor, sensory, and vegetative changes and automatisms.

Is it a seizure?

In brief:

- Increased muscle tone is far more likely in seizures. The most common recognised seizure is a generalised tonic-clonic seizure. Most commonly, the animal first goes stiff (tonic phase), loses proprioception and collapses into lateral recumbency, then the tonic-clonic phase (rhythmic alternating muscle contractions) starts followed often by running movements (automatisms). Atonic seizures are very uncommon and a 'floppy' collapse should guide the clinician to 'think' syncope or cataplexy.
- Rhythmic alternating muscle contractions are common in both focal and generalised seizures.
- Seizures often first involve the head and facial muscles (eye or facial muscle twitching).
- Stereotypical - most animals will have only one (or two) type(s) of seizure (seizure onset generalized, focal seizure onset with or without secondary generalisation). Seizures in an animal typically originate from the same epileptic focus and spread following the same brain pathways.
- The ictus (seizure itself) normally lasts 1-2 minutes.
- Most seizures exhibit several stages:
    - Pre-ictal behaviour changes (prodrome [hours to days] and/or aura [minutes]).
    - Ictus.
    - Post-ictal behaviour or neurological deficits (hours to days).

Apart from the seizure itself, it is the post-ictal changes that are recognised by the owner.

- Common post-ictal dysfunctions are:
    - Behaviour changes such as fear, aggression and disorientation.
    - Increased appetite.
    - Compulsive pacing.
    - Blindness, usually with normal pupillary light reflexes consistent with "central" blindness.
    - Menace response deficits.

    - Miosis contralateral to the lesion (if one lesion [secondary to disinhibition of the oculomotor nucleus]).
    - Gait abnormalities especially ataxia and “conscious” proprioceptive (paw position) deficits.
- Seizures often, but not always, occur at rest or while sleeping.
- Seizures usually impair the consciousness of the animal.
- Most of the seizure disorders will at least initially respond to antiepileptic treatment.

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Intervertebral Disc Disease – diagnosis and management

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 identified 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.

M**yelopathic 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:

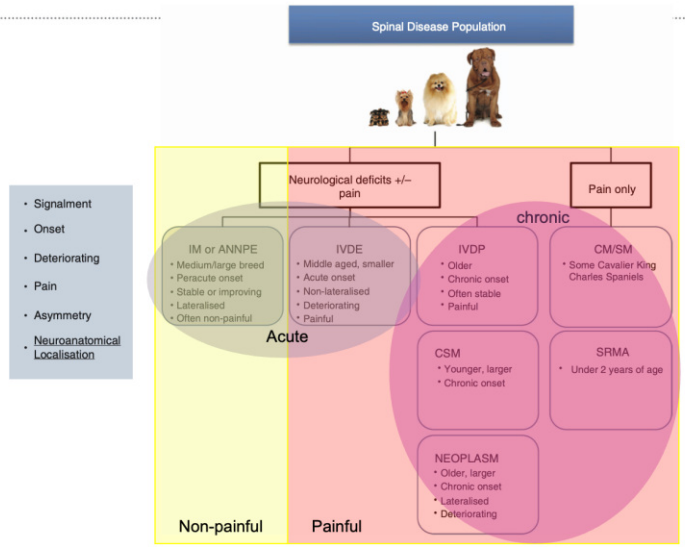
- Patients who present with peracute, non-progressive or improving, largely non-painful and lateralised neurological deficits (most often in T3-L3 spinal cord segments) have a 98% chance of having an ischaemic myelopathy such as fibrocartilaginous embolism (FCE) or acute nucleus pulposus extrusion (high 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 occasion- ally lateralised myelopathy (often at T3-L3 spinal cord segments). Ninety percent of patients presenting

with these clinical signs will have Hansen type-I disease. [SEP]

- 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 neu- roanatomical localisation (multiple spinal cord segments and/or brain). Many of the animals will also have mentation changes and cranial nerve deficits. [SEP]

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) Hemmorrhage (Y,A) Vascular malformations (Y)		

ESAVA Congress, "Kahun 1"

Prof Jane Ladlow MA VetMB, CertVR, CertSAS DipECVS

Where Are We With Brachycephalic Airway Syndrome?

As the name suggests, BOAS is found predominately in the extreme brachycephalic breeds (the Pug, French Bulldog and Bulldog).

There are many definitions of brachycephaly. The most common one is shortened skull length to width (width >80% of length) which results in both nasal cavity and pharyngeal compression. According to this skull index definition, there are numerous brachycephalic breeds, including the Pug, French Bulldog, Bulldog, Boston Terrier, Cavalier King Charles Spaniel, Pekingese, Japanese Chin, Dogue de Bordeaux, Shih-tzu, Boxer, Pomeranian, Griffon, and Lhaso Apso.

We define BOAS as upper airway obstruction related to the restricted length of the skull, particularly the nasal cavity. This definition however is non-specific, giving little information about the site and severity of the airway obstruction.

The prevalence of BOAS within the extreme breeds is not yet clear. In the last 10 years we have been studying the extreme brachycephalic breeds in order to develop an objective respiratory function test. Although there is a perception from some veterinary professionals that all brachycephalic dogs are affected to some extent, we have seen dogs from the extreme brachycephalic breeds that have no upper airway noise and normal exercise tolerance. In our study population (excluding clinical cases) we have classified approximately 40% of Bulldogs, 46% of French Bulldogs and 60% of Pugs with clinically significant disease (Liu et al 2017).[3]

Dogs usually present with BOAS between 1 and 4 years of age although severe cases may be noted when only a few months of age.

If a puppy does develop signs of upper airway obstruction however it will usually get worse with age.

There are some risk factors that mean that a dog is more likely to develop BOAS over its life span. Interestingly, in pugs, females are more likely to be affected than males whilst in bulldogs and French bulldogs more males are affected. The main risk factors for developing BOAS are having closed nostrils and being overweight. There are some external conformational factors (shape of the head) that have a smaller effect on developing BOAS, in the pug these are having a wider head and having wide set eyes. In the bulldog it is the wide head and thick neck whilst in the French bulldog it is short neck length and shorted muzzle.

Clinical signs are exacerbated by heat and exercise.

In Pugs particularly, obesity is linked to an increased risk of BOAS[7] (Liu et al 2016) and weight reduction can improve respiratory function. One of the risk factors for BOAS is neck girth and this may be partly a reflection of body condition score (Packer et al, 2015; Liu et al. 2017).

Normal Respiration

Dogs normally breathe through their noses. Air enters into the nostrils and moves to the nasal cavity past the nasal turbinates. The turbinates are bony structures covered by mucus membranes, these membranes serve to warm and humidify the air before it moves to the lung. The air then passes over the soft palate, through the back of the mouth, and into the larynx to enter the trachea. When the dog is excited or exercising, nasal breathing will switch to open-mouth breathing in order to increase the efficiency of inspiration and reduce heat by panting.

Disease Syndrome-Pathophysiology

The upper airway obstruction may manifest as respiratory noise, dyspnoea, exercise intolerance, heat sensitivity, sleep disorders, cyanosis or collapse (Roedler et al 2013). Gastro-intestinal signs include regurgitation and vomiting with oesophagitis from increased gastro-oesophageal reflux. There is also an increased incidence of hiatal hernia in these dogs. Excessive ingestion of air when during dyspnoeic episodes can result in gastric tympany and distention and increased gastrin secretion.

Vet Compass has been used to show that the extreme brachycephalic breeds died younger than equivalent sized dogs with a higher proportion of deaths due to upper respiratory disease (O'Neil et al, 2015).

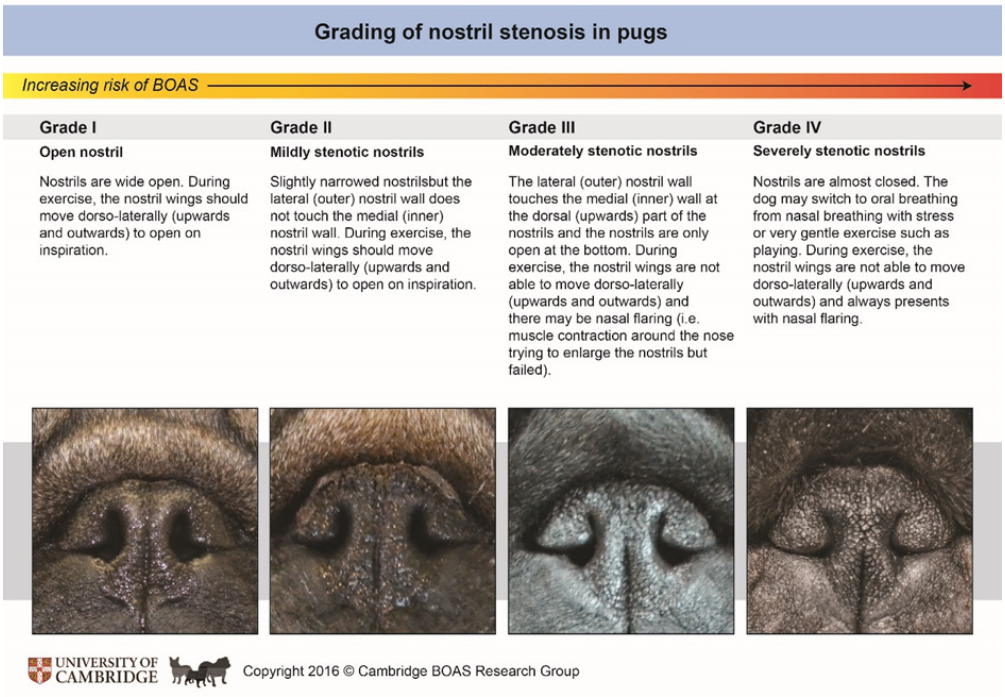
Lesion sites

The lesion sites that are linked to BOAS include stenotic nares, a thickened and overlong soft palate, hypoplastic trachea and bronchial collapse, hyperplastic tongue and excessive turbinate crowding in the nasal cavity.

These lesions can cause secondary areas of collapse and inflammation, notably laryngeal collapse, laryngeal saccule eversion and tonsillar hypertrophy. The increased thoracic pressures may result in pulmonary hypertension and cor pulmonale in addition to the gastrointestinal signs of reflux and hiatal hernia with the accompanying risk of aspiration pneumonia.

We have developed a nostril grading system to assess the effect of degree of nasal stenosis on BOAS incidence and we regard open and mildly stenotic nostrils as acceptable and are encouraging breeders to breed away from moderate and severely stenotic nostrils (Figure 5). Grade 0 and 1 are not associated with a greater risk of airway disease whereas grade 3 (moderate stenosis) and grade 4 (severe stenosis) are linked to a fourfold increase in risk of developing BOAS.

Nostril grading system



Laryngeal saccules or ventricles sit in the larynx, between the vestibular and vocal folds. The saccule lies medial to the thyroid cartilage and lateral to the vocal and vestibular folds. This is the first stage of laryngeal collapse as the saccules are place of least resistance in the wall of the larynx and therefore evert under the action of negative pressure.

We further divide laryngeal collapse into three stages:

1. Everted laryngeal saccules (seen in 50% of cases)
2. Deviation of the cuneiform cartilage medially
3. Medial collapse of the cuneiform and corniculate cartilage of the arytenoid obstructing the airway

Both severely and mildly affected dogs may have aberrant turbinates, stenotic nares and long soft palates with the severely affected dogs more likely to have grossly thickened soft palates. This demonstrates how difficult it is to look at lesion sites independently and we also need to remember that static images on endoscopy, radiography or CT may be affected by the presence of an ET tube, phase of respiration, head position and degree of mouth opening, and thus need to be interpreted with caution.

BOAS Respiratory Assessment

Disease recognition and Functional Grading

A major problem with BOAS is that it is often under recognized by both the public and veterinary professionals. A questionnaire-based study on disease recognition revealed that approximately 60% of owners did not perceive their dogs to have airway obstruction when clinical examination and history were compatible with the disease (Packer et al, 2012).

This inability to clearly define BOAS, along with clinical signs that are often described as ‘normal’ for the breed, makes determining the prevalence of BOAS within breeds very difficult. Often anatomical lesions are used as a definition of disease, however, not all dogs with stenotic nares will show signs of BOAS.

While upper airway noise is an indication of respiratory obstruction, many animals seem to tolerate some mild degree of respiratory noise and still live a seemingly good quality of life, albeit with some snoring or stertorous breathing when exercising or stressed.

As the site of obstruction can vary amongst dogs, the clinical signs or problems that the dogs have differs amongst individuals. The usual problems are struggling to exercise (exercise intolerance), noisy breathing, struggling to

thermoregulate (keep cool) in warm weather, regurgitation of food and/or saliva and sleep disorders.

The noisy breathing usually gets worse when the dog is excited or exercising and there are two sounds we are listening for. The first is a lower pitched noise (or stertor) which is usually the soft palate vibrating because it is too long or because the nasal cavity is obstructed.

https://youtu.be/iD0vWtjgCs0

The second sound we are listening for is a higher pitched noise or stridor which is usually a sign that the larynx or voice box is collapsing due to high pressures when breathing in (think hosepipe with a large amount of suction applied). Laryngeal collapse is more common in pugs than in bulldogs or French bulldogs. Pug can also suffer from collapse of the trachea and main stem bronchi though we think this is often secondary to the upper airway obstructions.

https://youtu.be/pQd0xbddfvY

Other signs of BOAS are using the abdominal muscles to breathe or the skin at the front of the chest (neck) moving when breathing.

https://youtu.be/_dADh1gbExs

It can be more complex as many French Bulldogs will have nasal stertor and we have seen Pugs with nasal stridor and Bulldogs with both nasal and pharyngeal stertor. Some dogs have no nasal noise due to the fact they have minimal nasal airflow but the lack of nasal stertor does not mean these dogs have a better airway compared to dogs with obvious nasal airflow noise.

There are other signs of obstructed nasal airflow including flaring of the skin just behind the nasal planum, lack of nasal wing mobility or excessive panting.

The clinical picture is further obscured by some dogs, particularly in the Pug breed, that appear to have reasonable respiratory function until stressed or exercised when they then show marked dyspnoea.

BOAS: Clinical Signs

- Noisy breathing:
- Stertor- pharyngeal noise, lower pitched, snoring and snorting
- Stridor- laryngeal noise, mostly inspiratory and higher pitched
- Exercise and heat intolerance
- Sleep apnoea/ disorders
- Hyperthermia
- Cyanosis and collapse
- Regurgitation and vomiting
- Flatulence

Respiratory Functional Grading (RFG)

When we auscultate these dogs we listen directly over the larynx, with the stethoscope held gently against the side of the larynx, not ventrally.

For diagnosis we use a functional grading system for BOAS that includes an exercise test. We exercise the dogs at a moderate trot (4 mph) for 3 minutes and note refusals, airway noise, effort, and extreme respiratory signs pre and post exercise. We grade airway noise as:

- 0 – not present
- 1 – mild (only audible with a stethoscope)
- 2 – moderate (intermittent and heard without a stethoscope)

- 3 – severe (easily audible without a stethoscope and constant).

While this system is not perfect, as exercise tolerance may be affected by ambient temperature and sometimes the animal's fitness levels, it does expose those animals that are teetering on the edge of obstruction when calm and firmly tipped into 'affected' once stressed.

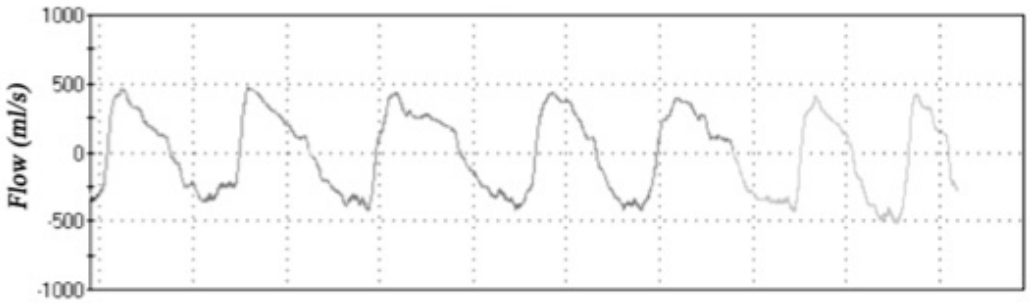
Exercise tests also reveal clinical signs in those dogs that only have respiratory noise when mouth breathing (often the long but not particularly thickened soft palates) after switching from nasal breathing in the consult room.

We find that about 40% of dogs that show few clinical signs in the consult room will be classified as affected after the exercise tolerance test. We consider no airway noise (grade 0) and mild airway noise (only audible with a stethoscope – grade 1) as clinically unaffected as in these dogs we do not detect any decrease in quality of life, with normal exercise tolerance and no other associated clinical signs. The grade 2 and 3 dogs have airway obstruction that affects quality of life and are regarded as clinically affected.

This grading system enables us to advise on treatment – grade 3 dogs are surgical candidates, grade 2 dogs are likely surgical candidates but if obese, weight loss could be initiated prior to surgery. We can also monitor dogs with the grading system and use it in addition to our objective respiratory function test.

In human respiratory medicine, in addition to clinical examination, respiratory function would be tested with whole-body plethysmography in conjunction with forced spirometry. An objective measure of respiratory function in the brachycephalic dogs would help to determine which dogs require treatment, response to such treatment and to guide the breeding of healthier individuals. Unfortunately, our clinical patients are not compliant with requests to breathe out into a mouthpiece with maximum force or breath-hold before inspiration.

The technique that we use for the extreme brachycephalic breeds dogs is whole body barometric plethysmography (WBBP) where dogs are placed in a sealed chamber with biased airflow moving across it.



A trace from WBBP performed on a French bulldog, from these traces parameters that can be generated include breathing frequency; total inspiratory volume, time and peak flow; total expiratory volume, time and peak flow; lags between breath cycles (if any) and hence minute respiration volume

Over the last 10 years we have tested more than 1000 dogs with WBBP. We have used 20 consecutive breaths when the dogs are relaxed yet awake to record flow time traces. Approximately 10–20% of dogs do not give a usable test result due to constant panting, barking or movement.

By looking at three different markers of respiratory obstruction, we have developed a test for respiratory obstruction in the Pug, French Bulldog and Bulldog that is >95% accuracy compared with clinical functional assessment (Liu et al 2015, Liu et al 2016). This test has been further developed to give a BOAS index; a numerical score from 0–100% to describe how severely an individual dog is affected with BOAS, where 0% is non-affected and 100% is our most severely affected individuals.

The BOAS index is important as we now have an objective measurement with which to evaluate risk factors for BOAS and the effectiveness of our surgical treatments, looking at the whole upper airway rather than individual sections. The BOAS index can also be used to objectively describe the phenotype which

is required for genetic work and can aid breeders by screening the breeding animals.

BOAS: Diagnosis

- History
- Clinical signs at rest and during exercise- functional grading
- Oral examination under sedation/ GA
- Head/ thoracic CT or radiographs
- Examination of the nasopharynx with an endoscope

Imaging

As usual in veterinary medicine, this unfortunately depends on the owner's budget. Direct laryngoscopy is sufficient to evaluate the larynx, tonsils and gives a good idea of soft palate length and thickness.

A CT scan of the head will reveal turbinate morphology and the soft palate thickness. Rhinoscopy is a complementary and probably more accurate method of assessing turbinate crowding, with a rigid scope used for the rostral nasal cavity combined with a flexible scope retroflexed behind the soft palate to assess caudal aberrant turbinates, trachea and bronchi and oesophageal lesions.

CT of the thorax enables assessment of bronchial collapse, aspiration pneumonia, oesophageal diverticulum and may pick up hiatal hernia (De Lorenzi et al 2009). Radiographs of the thoracic cavity allow the tracheal diameter to be gauged and aspiration pneumonia excluded if CT is not possible.

If finances are very limited then it is reasonable to treat without imaging, particularly as we think a thorough clinical examination is the most important diagnostic test. However, in cases that do not respond successfully to conventional surgery, nasal obstruction or aspiration pneumonia may have been missed.

Opening Airways: Surgical Solutions for BOAS in Practice

BOAS - how to facilitate treatment

- Diet if overweight
- Treat intestinal signs- Omeprazole, sulcralfate
- Soft palate shortening and thinning
- Opening of nostrils
- Resection of laryngeal saccules

- Resection of aryepiglottic fold
- +/- turbinate resection

Life-style management

Weight

A number of studies have shown a correlation between BOAS status and weight. Excessive fat around the neck, pharynx and thoracic wall increases respiratory obstruction and decreases tidal volume. Reducing an obese dog's weight is very likely to improve respiratory function though this has not yet been proven in clinical BOAS cases.

Gastrointestinal Medication

One study (Poncet 2005) reported 98% cases (out of 73 dogs) had chronic gastritis, 90% had lymphoplasmocytic duodenitis, and there was a positive statistical correlation between severity of respiratory and GI signs. In 80% of these cases GI signs resolved after BOAS surgery, with and without GI medication, although immediate post op vomiting and regurgitation were reduced if the dogs were given 0.7mg/kg omeprazole, 0.2mg/kg cisapride and 1g sucralfate. The authors also believed that concurrent medical management of GI tract pathology may have helped reduce the progression of the respiratory disease (laryngeal collapse).

We currently use 1mg/kg omeprazole, initially once daily up to twice daily in frequent regurgitators. A small number of dogs will develop vomiting and diarrhoea on omeprazole, in which case we switch to cimetidine or ranitidine.

Other lifestyle amendments include avoiding exercise in hot weather and the use of cool jackets in the warmer summer months when out.

Surgical options

If lifestyle management is not sufficient, we have surgical options for this disease.

Preparation

If the animal is a frequent regurgitator then starting an antacid such as omeprazole or ranitidine 5-7 days prior to surgery may improve clinical signs.

Stressed animals are more difficult to treat effectively. If the dog is anxious or of a stressful temperament, then we usually dispense trazadone (5-10 mg/kg) and/or gabapentin 10-20 mg/kg to be given 2 hours prior to admission and for these dogs we would try and admit the morning of the procedure rather than hospitalising overnight.

Equipment

Table arms are very useful to suspend the head in theatre, otherwise drip stands can suffice. A customised BOAS stand is produced by VI (designed by me) which maximises exposure.

When positioning we tie the lower jaw and tongue down to maximise the space at the back of the pharynx.

Equipment includes delicate long handled debakeys and metzenbaum scissors, delicate curved haemostats and microvascular scissors for the larynx. The nasoplasty is done with No 11 scalpel blades.

Large sterile cotton buds are useful and we use bipolar electrosurgery for haemostasis, vessel sealers and monopolar electrosurgery are also often commonly used.

BOAS - Anaesthesia guide and the emergency patient

To anaesthetise brachycephalic dogs safely does need some forward planning.

Ideally assess the dogs prior to their procedure, specifically noting any airway disease and/ or regurgitation.

Fasting. The latest anaesthetic papers suggest there is no benefit to prolonged fasting of brachycephalic dogs prior to general anaesthesia and this may increase regurgitation during anaesthesia. We fast from midnight.

Equipment Needed

- Laryngoscope with a good light and suitable length blades
- Variety of ET tubes- silicon with high volume/ low pressure cuffs.
- Suction
- Q tips
- Swabs
- Cuff inflator- manometer or syringe
- Monitor- pulse oximeter and capnography and ECG
- Tracheostomy tubes
- Emergency kit

Premedication

A light premed is used- such as methadone (0.2mg/kg) IM followed by low dose medetomidine (0.001mg/kg) IV slow (0.002mg/kg IM fractious dog). In fractious dogs we add 1mg/kg alfaxalone IM

Butorphanol/ methadone or acepromazine (anti-arrhythmic/ methadone are also acceptable.

ACP needs 10-15 minutes of very calm environment to be effective.

Intravenous catheter placement is achieved and the dog is pre-oxygenated prior to induction.

Induction

Pre-oxygenate for 5-10 minutes, if the dog has airway disease it is likely to decompensate when induced – it makes sense to preload with oxygen.

I tend to use IV propofol given relatively slowly at 2-4mg/kg. Alfaxalone is also a suitable induction agent.

It is sensible to have a range of ET sizes available and, if any signs of laryngeal collapse are evident then smaller tubes such as urinary catheters or a stylet can be useful. Induction should be intravenous and to effect, we like to keep the dogs under a light plane of anaesthesia and assess the laryngeal movement prior to intubation. A low-pressure high volume cuffed ET tube is used to protect the airway during surgery.

Use large cotton buds to remove any obstructing saliva/ foam and have suction ready in case of regurgitation (unusual).

Secure the ET tube and place on oxygen at a suitable flow rate, gently inflate the ET cuff, checking for leaks or using a manometer. Start inhalation agent- isoflurane is usual though sevoflurane is fine.

Check capnography and ensure breathing. Check ECG/ pulse oximetry. Keep a check on temperature and make sure they don't become hyperthermic.

We give omeprazole (if not given previously) and metoclopramide to any dogs with airway disease or with regurgitation. Also consider maropitant for BOAS cases.

Maintenance

If the anaesthetic is not stable consider ventilation – also if ET CO_2 > 65, excessive panting or frequent arousals.

Use frequent eye lubrication as brachycephalics are prone to corneal dryness- this should continue for 24 hours post-surgery.

If regurgitation occurs then gentle suction the oesophagus, lavage with water and suction again.

For BOAS surgeries:

Intravenous dexamethasone is given at induction to try and avoid airway swelling. Local nerve blocks (maxillary) are effective at reducing arousal during nasoplasty. We use bupivacaine – upto 1 ml (not more than 2mg/kg split between each side).

We use xylometazoline (otrivine), 0.3ml per nostril which is an alpha 3 agonist and shrinks the nasal mucosa. I use it pre and post scoping to see the consistency of the turbinates. It increases nasal airspace and there is an argument for using it in any French bulldog or Pug with nasal signs that undergoes anaesthesia for any procedure.

Be aware of the vasovagal or trigeminal cardiac syndrome when working in the nose/ on eyes/ changing position and have atropine close by.

Emergencies

- Check temperature and cool appropriately.
- Flow by oxygen and place IV catheter
- Try intravenous dexamethasome
- Nebulised adrenaline - 0.05mg/kg in 5 ml of saline. Effective in laryngeal oedema
- Judicious sedation – medetomidine/ dexmedetomidine
- Be ready to intubate…..

Avoiding problems with anaesthesia

Complications after anaesthesia in brachycephalic dogs increase with duration of anaesthesia and thus if possible split elective procedures, particularly if the dogs have BOAS or are regurgitators.

Surgery

Surgical technique order

1. Tonsillectomy- removal of extruded part of hypertrophic tonsil
2. Soft palate reduction
3. Laryngeal surgery if required
4. Nasal Surgery

Tonsillectomy

We perform resection of hypertrophic tonsils that extrude outside the tonsillar crypt (Fasanella 2010, Oechtering 2016).[9] Most dogs with clinical signs of

BOAS have hypertrophic tonsils that may not look particularly important when the dog's mouth is open with an elevated head. However, when the mouth is closed, these tonsils put pressure on an already compromised nasopharynx (http://www.vet.cam.ac.uk/boas). [22] By removing only the extruded part of the tonsil, less haemorrhage occurs, although we do use either bipolar coagulation, a harmonic scalpel or a laser for this procedure.

Soft Palate Resection

Conventional surgery consists of soft palate surgery, either staphylectomy or, if the palate is thickened, a folding flap palatoplasty where the thickness of the palate is also reduced at the time of surgery (Findji 2008). The staphylectomy has become more radical over the years and now an extended staphylectomy involves trimming the palate in a smooth U-shape to the rostral tip of the tonsils. Carbon dioxide laser resection techniques have been reported- the results are very similar to incisional techniques.

When shortening the soft palate, the aim is for the palate to be at rostral/mid edge of tonsils, just contacting the epiglottis when the mouth is closed- extended staphylectomy.

Staphylectomy

In a cut and oversew technique, place an absorbable suture at the designated resection site on one side, cut 1/3 of the palate and the suture nasal mucosa to oral mucosa. Continue along soft palate using simple interrupted or continuous sutures of 1.5 M vicryl.

Modified Folded flap palatoplasty:

U shaped incision is made through the oral mucosa from the back of the palate extending to rostral to the palate, the underlying stroma and muscles are stripped from the nasal mucosa and leaving the nasal mucosa 1-3 cm longer than the oral mucosa. This nasal mucosa is folded over and sutured. Sutures are simple interrupted vertical mattress sutures of 1.5 M vicryl. This results in a thinner section of caudal soft palate. There are vessels entering the palate dorsolaterally that need addressing with electro surgery.

Nasoplasty

Other procedures that are included in the conventional surgery are widening of the nares and everted laryngeal saccule resection. The nares surgery we perform now involves resection of the inner alar fold within the vestibule (alar fold or second stenosis) along with resection of the wing of the nares (Oechtering personal communication). We use a Trader technique for the external nares and the combination of these two nares techniques results in a very much enlarged nostril.

Although the nostrils can look rather raw at first, in a few weeks they pigment and appear normal

Laryngeal Surgery

The laryngeal sacculectomy is controversial with some advocating leaving saccules in position as they are likely to regress after improvement of the other areas of the upper airway dogs (Poncet et al. 2006, Riecks et al. 2007).The everted laryngeal saccules seen in dogs are not fibrotic on histopathological examination but rather oedematous. As such, regression is possible but this was

not found in an elegant study by Cantatore and colleagues in 2012 where they performed unilateral sacculectomy at the time of conventional surgery and re-scoped the 10 dogs 4–6 months later when none of the remaining everted saccules had regressed.

On presurgical laryngoscopy with no ET tube in position, everted laryngeal saccules are sucked into the rima glottides on inspiration and can occupy the ventral 50% of the airway (http://www.vet.cam.ac.uk/boas).Immediately after surgery there is some degree of airway swelling and it seems sensible to remove as much obstructive tissue as possible to aid recovery. Removal of the saccules is relatively simple and unlikely to cause many post-operative problems (Koch et al. 2003, Lorinson et al. 1997, Torrez & Hunt 2006) although a recent paper reported more post-operative complications in dogs that underwent sacculectomy (Hughes et al 2017).

We also resect the laryngeal arytenoid cuneiform process in dogs that have a grade 2 or 3 laryngeal collapse, (Oechtering personal communication). Removal of the cuneiform results in less tissue that can be sucked into the rima glottidis on inspiration and appears to aid stabilization of the larynx. After unilateral surgery the larynx appears to heal well with no stenosis. There is a small vessel which is inclined to bleed after resection of the cuneiform but this is addressed by topical adrenaline on a swab.

Recovery

I usually recover BOAS cases in the OR as it is quiet, has all the monitoring and if intervention is needed it's easier. We wake the dogs up in sternal recumbency, slowly, over ten-twenty minutes, with supplemental oxygen. If the dogs has had airway surgery check the pharynx carefully for blood clots prior to extubation.

We only remove the tube when the depth of anaesthesia is light and the dog is swallowing. Prior to extubation, I usually check if oxygenation is maintained on

room air. If not, delay recovery, position in sternal if they were in lateral and assess if any lung injury e.g. from aspiration. Once saturation is maintained on room air we will wake up, using supplemental oxygen for 10-20 minutes post recovery.

We observe pulse oximetry and respiratory effort for the first 20 minutes and then monitor closely for the next couple of hours. If the dog becomes agitated and analgesia is sufficient then a dexmedetomidine infusion (0.0015-0.001mg/kg per hour) can be used though we find that usually recovering with a nurse close by is sufficient to calm most brachycephalics.

As dogs are usually given steroids (dexamethasone) at induction we usually chose paracetamol for analgesia, using an oral suspension (off cascade) for the smaller dogs twice daily for 5 days. For large dogs Pardale V can be used.

Antibiotics are not routinely used unless there was evidence of aspiration pneumonia.

Omeprazole is usually continued for 5-7 days after surgery for frequent (daily) regurgitators.

We nebulise the dogs with steam every 3-4 hours to moisten airways and prevent crusty secretions forming. If any dogs are showing respiratory effort after surgery nebulizing with adrenaline (0.05mg/kg in 5 ml of saline) will reduce some airway swelling and seems particularly effective in pugs.

If the dog recovers from traditional surgery and is still obstructed, we initially place a temporary tracheostomy tube, which is usually left in place for 24–48 hours while the surgical swelling resolves. We use temporary tracheostomy tubes in less than 5% of our cases, the majority of which then go on to have a reasonable recovery. Many of the tracheostomy tubes are placed pre-emptively in the grade 3 laryngeal collapse cases to allow a smooth and less distressing recovery for the dog.

If possible, we discharge the dogs the same day (not bulldogs) and we are often feeding 4-6 hours post recovery with a low-fat diet (chicken works well).

Post-operative Care

- Keep the ET tube in as long as possible at the end of surgery- until the dogs can swallow
- Use supplemental oxygen until fully recovered
- BOAS cases require close monitoring for the first few hours as post-operative swelling or haemorrhage can cause upper airway obstruction.
- Post-operative tracheostomy is occasionally required
- Advise soft food for the next couple of weeks
- Monitor

Surgical Results

Conventional surgical results are, however, usually encouraging. Colin Harvey reported a 70–85% success rate in the 1980s depending on extent of surgery and since then most papers have reported a good outcome for the majority of patients with about a 10% risk of post-operative complications, including death (Harvey 1982, Fasanella et al 2010, Findji et al 2008, Riecks et al 2007, Poncet et al 2006). Torrez in 2006 was a little more guarded with regard to prognosis, with 56% of 73 dogs much improved after surgery, 32% having some improvement and 10% of patients showing no improvement. Signs that persisted after surgery were snoring during sleep, stertor/stridor while conscious, excessive panting and dyspnoea. The authors concluded that very few patients would have resolution of clinical signs post-surgery, even though owner satisfaction was high.

This paper agrees with our objective assessment of improvement post-surgery, where about 50% of dogs improve to an extent that we would consider them clinically unaffected. The remainder of dogs remain clinically affected though usually improved (Liu et al 2017). LATE surgery for dogs that have little response to the first surgery results in a further 80% being classified as clinically unaffected after LATE (Liu unpublished data).

Gastro-intestinal signs such as reflux, nausea and hypersalivation will also frequently respond to upper airway surgery, particularly in French Bulldogs (Poncet et al 2006, Haimel et al 2015).

Dogs that present early, are in a slim body condition on presentation or have laryngeal collapse, will generally have a worse prognosis and it is worth discussing this with owners before starting on an expensive and invasive treatment (Liu et al 2017). On CT, medialisation of the pterygoids in French bulldogs is associated with a poorer prognosis.

If these procedures fail to improve the airway, the options that remain are a laryngeal arytenoid lateralization (White 2012), permanent tracheostomy or euthanasia. In 2012, White reported good success with laryngeal lateralization and we have found it a very useful technique in the most severe cases. Although some extreme brachycephalics may tolerate a permanent tracheostomy, we try to avoid this technique as the hypoplastic trachea and thick (often fat) neck with prominent skin folds is a poor combination.

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https://www.vet.cam.ac.uk/boas/about-boas

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AVA president

Unlocking the fear of brachycephalic anaesthesia

General anaesthesia carries an inherent but relatively low risk of death in dogs; this risk can be influenced by several factors including the reason for anaesthesia (e.g. emergency vs. elective surgery), pre-existing disease and other patient-related factors. An anaesthetic-related death rate in low-risk dogs (ASA 1–2) was reported as 0.05% (1 in 2000) compared with 1.33% (1 in 75) in higher risk (ASA 3–5) patients (Brodbelt et al. 2008). Brachycephalic dogs are at greater risk of experiencing complications in the perioperative period compared to non-brachycephalic breeds (Gruenheid et al. 2018). Complications in the postoperative/recovery period include regurgitation, vomiting, prolonged time to extubation, aspiration pneumonia and stertorous breathing (Gruenheid et al. 2018). Familiarity with the common pre-existing conditions in brachycephalic dogs will help our understanding of the best way to approach them and the need to alter anaesthetic management.

Brachycephalic Obstructive Airway Syndrome (BOAS) is characterised by dynamic and/or static URT obstruction with clinical signs including snoring, dyspnoea, cyanosis, exercise intolerance, and syncope. BOAS is the result of a combination of abnormalities including stenotic nares, elongated soft palate and a hypoplastic trachea. Aberrant turbinates can cause intranasal airway obstruction. Studies have shown that brachycephaly predisposes to (but does not guarantee) the development of BOAS, with French Bulldogs, Pugs and English Bulldogs being overrepresented (Liu et al., 2017b, O'Neill et al., 2015). Surgical intervention may improve the flow of air through the upper airways in up to 90% of dogs with clinical signs of BOAS and may also reduce subsequent anaesthetic risk.

Oesophageal and gastrointestinal abnormalities are common in brachycephalic dogs including gastro-oesophageal reflux (GOR), hiatal hernia, cardial atony, gastritis, distal oesophagitis, and pyloric hyperplasia. The presence of either respiratory or gastrointestinal disease could worsen the other. Improved outcomes after upper airway surgery have been associated with the diagnosis and treatment of gastrointestinal problems. Treatment may include gastroprotectants (omeprazole and sucralfate), prokinetics (cisapride or metoclopramide) and anti-emetics (maropitant).

Corneal ulcerative disease is common in brachycephalic breeds due to their prominent eyes, reduced corneal sensitivity and secondary corneal drying.

General anaesthesia increases the risk of corneal damage by temporarily ablating the normal blink response, increasing the chance of desiccation and ulceration. Hospitalisation, critical illness, and certain anaesthetic drugs, including some opioids, inhalation agents and medetomidine, further reduce tear production, thus prolonged ocular lubrication is necessary.

Anaesthetic management

Brachycephalic dogs may present for anaesthesia for elective or emergency upper airway surgery, or for unrelated routine and emergency procedures. High-risk periods include post sedation/premedication, induction of anaesthesia, recovery from anaesthesia and the initial post-operative period.

Pre-anaesthetic assessment and stabilisation

History and clinical examination prior to anaesthesia is mandatory. Stertor/stridor, snoring, length of time sleeping in the day, exercise and heat tolerance, gagging/retching, cyanosis, and syncope can help identify underlying significant comorbidities. An exercise tolerance test may help identify high risk patients prior to anaesthesia. Risk mitigation may include postponing elective procedures until the dog's anaesthetic risk can be reduced through weight loss, improving fitness and giving pre-operative gastroprotectants.

Withholding food and water prior to anaesthesia is associated with a reduced risk of mortality, however, prolonged fasting (>10 hours) may be associated with decreased gastric pH and increased incidence of reflux. Prior to elective anaesthesia, whilst common to advise withdrawal of food after midnight (practical approach), it may be more appropriate to reduce the fasting period in healthy patients to allow a small meal of easily-digested, wet food, 3–6 hours before anaesthesia. Generally, water intake is permitted until the time of premedication.

Pre-anaesthetic medication and Induction

Pre- anaesthetic medication provides sedation, anxiolysis, facilitates handling and lowers the dose of induction agent required and therefore the risk of post-induction apnoea, and provides analgesia. Whilst most cases benefit from it, there is no 'perfect' pre-anaesthetic medication and induction technique for brachycephalic dogs, with no robust evidence to support the blanket use of one technique above another.
Temperament, the presence and severity of concurrent disease, the procedure to be carried out, the availability different drugs and the clinician's familiarity with a particular protocol will all influence choices.

Sedated brachycephalic patients must be closely monitored at all times for signs of URT obstruction including increased stertor/stridor, increased inspiratory effort and exaggerated movement of the chest wall. Temperature should be kept within the normal range from the time of admittance until discharge. Active warming or cooling might be required.

Drug	Dose	Route
Acepromazine	**Premedication:** 5-20 µg/kg*	IM or IV
	Post-operative sedation: 5-10 µg/kg q. 3-6 hours	IV
Medetomidine	**Premedication:** 1-10 µg/kg	IM or IV
	Post-operative sedation: 1-3 µg/kg IV up to q. 1 hour if needed +/- CRI 1-2 µg/kg/hr	IV
Dexmedetomidine	**Premedication:** 1-5 µg/kg mcg/kg	IV or IM
	Post-operative sedation: 0.5-1 µg /kg IV up to q. 1 hour +/- CRI 0.5-1 µg/kg/hr	IV
Methadone	0.1-0.3 mg/kg	IM or IV
Buprenorphine	0.01-0.02 mg/kg	IM or IV
Butorphanol	0.1-0.3 mg/kg	IM or IV
Propofol	1-4 mg/kg (given slowly to effect)	IV
Alfaxalone	2 mg/kg (given slowly to effect)	IV
Trazodone	5-10 mg/kg	PO or rectal
Gabapentin	10-20 mg/kg	PO

Common drugs available for use for premedication, induction of anaesthesia and post-operative sedation in dogs. The doses specified are commonly used and lie within licensed ranges in the UK. In dogs with clinical signs of BOAS lower doses of sedative drugs are genrally recommended. * Lower doses of acepromazine (10 µg/kg or less) are recommended in Boxer breeds (Murrell, 2016)

Endotracheal intubation may be difficult so consider:

- A wide range of sizes of cuffed endotracheal tubes (ETT), which may need to be long with a narrow diameter (4 mm ID upwards)
- A laryngoscope, or at least a good light source
- A rigid stylet – this can be useful to aid visualisation of the larynx
- A suction catheter to allow suction of regurgitation or saliva if required

- A wet swab to hold and manipulate the tongue (macroglossia is problematic)

In cases of severe tracheal hypoplasia, a urinary catheter may be needed for intubation, either as a stylet, or modified to connect to a breathing system. Ideally assess the presence and severity of BOAS and laryngeal collapse in all brachycephalic dogs at induction of anaesthesia, to help identify patients at higher risk of developing problems during the recovery period.

Appropriate multimodal analgesia is of particular importance in animals with underlying respiratory compromise. Options for the management of acute pain in dogs, include non-steroidal anti-inflammatory drugs (NSAIDs), opioids, paracetamol, ketamine and local anaesthetic drugs. Whilst analgesia should never be denied if required, drugs must always be used appropriately and based on the individual's clinical need to avoid unwanted side effects including sedation, behavioural changes and postoperative nausea/vomiting (PONV). The indiscriminate use of methadone postoperatively in dogs after orthopaedic surgery (4-hourly administration irrespective of clinical need or pain score), dramatically increased the risk of vomiting and vocalisation compared with those receiving the drug post-operatively based on individual pain scoring (CMPS-SF) (Bini et al., 2018). The use of validated pain assessment tools will aid appropriate administration.

Incorporation of locoregional anaesthetic techniques is recommended. They provide excellent perioperative analgesia in orthopaedic, soft tissue and dental surgical patients.

If GOR occurs during anaesthesia, there is a risk of developing aspiration pneumonia, oesophagitis, irritation of pharyngeal tissues and oesophageal stricture formation. If regurgitation is observed and it is feasible to do so you can:

1. Check the ETT cuff is inflated and secure - this protects the airway and reduce the risk of aspiration.
2. Position the patient with the head elevated slightly and nose angled downwards to facilitate drainage of fluid away from the larynx without encouraging further regurgitation. *This may not be feasible if regurgitation occurs and the patient cannot be moved, e.g. during surgery*
3. Remove any fluid from the oesophagus and pharyngeal area by gentle suction as soon as possible.

4. Lavage the oesophagus with water and repeating suctioning until the fluid retrieved is clear. *N.B. some also advise instilling dilute bicarbonate solution into the oesophagus but this is still controversial .*
5. Consider administration of antacids +/- maropitant if not already given.

Recovery and postoperative management

Recovery is a high-risk period for URT obstruction, particularly following airway surgery. There may be post-operative mucosal oedema, active bleeding or blood clots causing obstruction as well as reflux, regurgitation and subsequent aspiration of debris and fluid. In addition, respiratory depression may result from the residual effects of anaesthetic drugs, hypothermia, or the increased work of breathing.

Plan for the following during recovery:

- Multimodal analgesia
- Administration of corticosteroids – criteria for intervention /drug/dose
- Use of sedation
- Use of topical vasopressors and/or nebulised adrenaline (epinephrine)
    - 0.05ml/kg of 1:1000 adrenaline diluted up to 5ml with 0.9% saline and nebulised for 5-10 minutes.
- Mannitol-soaked swabs - 10ml of 10% mannitol per 10x10 cm swab, wrung out, placed in pharynx for 30 mins and removed before recovery
- Temporary tracheostomy tube
- Need for emergency re-intubation at any point

During the recovery period, it is essential to help patients maintain airway patency until they have sufficient muscle tone and are alert enough to do this themselves. To help maintain an open airway, they should ideally be positioned in sternal recumbency with the head elevated, neck extended and the tongue drawn rostrally out of the mouth. Brachycephalic dogs will often tolerate endotracheal tubes for longer during the recovery period (Grubb, 2016), supporting airway management, maintaining a patent airway, and protecting against aspiration of gastric fluid and debris.

There are numerous considerations when anaesthetising brachycephalic patients and no one perfect way to achieve it. However, evidence suggests that implementing a structured way of assessing patients and then standardising the way complications and comorbidities are handled can improve outcomes.

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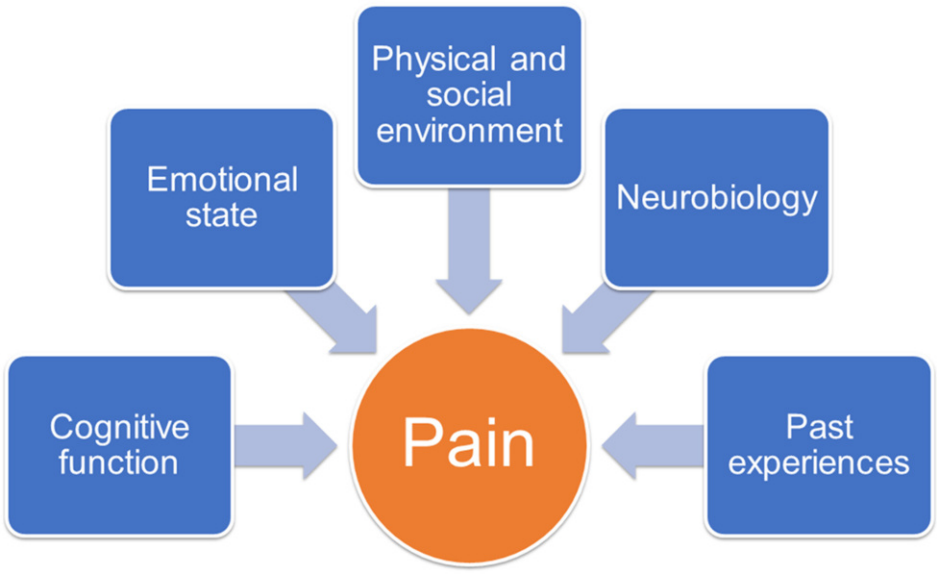
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Updates on acute clinical pain management

There has been a considerable shift in our understanding of pain perception and our ability to manage this pain in domestic animals over the last few years. Global, well publicised pain management guidance publications (Gruen et al 2022, Monteiro et al. 2022, Steagall et al. 2022) have disseminated contemporary information and improved clinician knowledge.

Understanding of pain contributors:

Acute pain is the result of a traumatic, surgical, medical or infectious event that often begins abruptly and should resolve with appropriate and timely management. Pain is a conscious multi-dimensional and individual experience. It is influenced by complex interactions between numerous internal and external factors. These influences can result in increased or decreased pain perception (from Monteiro et al. 2022)



Pain assessment.

Expert consensus emphasizes proactive, pre-emptive pain management rather than a reactive, "damage control" approach (Gruen et al. 2022). Where the aetiology of pain is traumatic, treatment should be initiated as soon as possible. This management strategy must include routine pain assessment. Inadequate analgesia is a likely outcome of not proactively assessing pain. Equally, the use of analgesia without appropriate pain assessment can have negative

consequences and not benefit the patient (Bini et al. 2018). The use of physiological parameters is no longer recommended as sole method of assessing pain. Observations that take behaviour, body posture and facial expressions into account are most likely to capture the animal`s experience pain. Multi-dimensional composite pain scales for assessing postoperative pain are available and their utility is assessed by Lee and Steagall (2026). This is a universally accepted validated feline pain assessment tool http://www.felinegrimacescale.com. Validation for the canine acute pain scales is less robust but detailed by Lee and Steagall (2026).

Pain management principles:

Pre-emptive analgesia refers to the administration of analgesic drugs before tissue damage/insult (i.e. preoperatively). Preventive analgesia is a more appropriate clinical approach as it refers to the administration of analgesics pre-, intra- and post-operatively.

Non-pharmacological therapy:

Non-pharmacological techniques have not previously been emphasised but should be included in pain management strategies. These include cold therapy, acupuncture, passive range-of-motion exercises, massage, therapeutic exercise, hydrotherapy, ultrasound and electrical stimulation. Nursing care including considerations of the physical environment, wound care, bladder emptying and human–animal interactions is important for patient welfare. Peri-operative pain can be influenced by the surgical technique and location. Gentle tissue handling and techniques that minimise trauma are preferred (*e.g*. small incisions; minimally invasive surgery such as arthroscopy and laparoscopy).

Pharmacological therapy:

Drugs frequently used for peri-operative analgesia include NSAIDs, local anaesthetics, opioids, alpha2-adrenocetor agonists & NMDA antagonists (e.g. ketamine). There is extensive detail in the aforementioned pain management guidance.

Some contemporary or developing pharmacological strategies (not comprehensive):

Paracetamol (acetaminophen) is an NSAID with multiple mechanisms of action including on COX-1 present in the CNS. It has analgesic and antipyretic effects but little anti-inflammatory activity. In the peri-operative period, both oral and IV paracetamol have been found to be non-inferior to licensed NSAIDs in dogs undergoing soft tissue or orthopaedic surgery. Recent work documents the pharmacokinetics of IV paracetamol administration and shows non-inferiority to buprenorphine in dogs undergoing laparoscopic ovariohysterectomy. Paracetamol is widely used by veterinarians working in the UK. Most veterinarians have changed their perception on the use of paracetamol over the last decade and consider it to be associated with fewer side effects and with more effective analgesic and antipyretic effects than they had previously believed.

Local anaesthetic techniques: Use of a local technique can often reduce the dose of other anaesthetic drugs required for maintenance of anaesthesia and contributes to a multi-modal analgesic technique. They are cheap and often easy to learn – the following are options for their administration:

- **Topical anaesthesia (transmucosal).** This can produce analgesia rapidly (within 5 minutes). Locations for the topical administration include the cornea for ocular examinations, the nasal passages (*e.g*. before placement of a nasal oxygen cannula and the larynx during intubation). The depth of analgesia produced in tissues is usually small (1 to 2 mm).
- **Infiltration anaesthesia** This is safe, reliable and does not require extensive experience. Sterile needles should be used. For example, local anaesthetics can be infiltrated along abdominal and hemilaminectomy incisions (*i.e*. incisional anaesthesia). This technique may be applied before and/or after surgery.
- **Local and Regional anaesthesia.** Nerve blocks for dental procedures based on anatomical landmarks are now perceived to be “standard of care” The first regional blocks to be described were based on analgesia of large motor nerves or nerve trunks, localised by nerve stimulators or ultrasound guidance. More recently fascial plane blocks (e.g. erector spinus plane block) using ultrasound guidance have broadened options for local anaesthetic use.

- **Epidural anaesthesia** Lumbosacral epidural anaesthesia may be used to provide analgesia for all procedures caudal to the diaphragm and sacrococcygeal epidural for caudal urogenital surgery.

A note on gabapentin

Gabapentin is primarily a calcium channel antagonist, binding to α-2-δ-1 subunit of pre-synaptic Ca2+ channels. It was developed to treat seizures in humans and subsequently in animals and later licensed for the treatment of chronic pain syndromes such as postherpetic neuralgia, fibromyalgia and neuropathic pain in humans. It ↓neuronal Ca2+ influx; ↓excitatory neurotransmitters. Its primary indication is neuropathic pain. It is not effective for nociceptive pain. Sedation, ataxia and somnolence are major side effects. Sedation side effect can be utilised clinically. It is important to correctly identify the animals in which gabapentin might be useful. Not to be considered substitutes of NSAIDs. Can be used in the perioperative period for sedation and anxiolysis but NOT as an analgesic. Whilst veterinary data is poor, recent human data confirms a lack of efficacy for peri-operative pain management.

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Louise Clark

Anaesthesia for hepatic, renal & paediatric patients

Effective management of these differing cases is based around the physiological differences present in these populations of patients, and the impact that these will have on the management and monitoring of anaesthesia. Drug pharmacology in each species and in patients of different age and physical status varies. There are differences between adults, paediatrics (puppies and kittens <12 weeks of age), seniors, and patients with comorbidities.

Paediatric patients:

The physiological differences in **paediatric patients (less than 12 weeks)** compared to adults are summarised below based on da Cunha (2022).

Respiratory system	
↑	Rib cage compliance
↓	Intercostal muscle strength
↑	Inspiratory pressure
↑	Work of breathing
↑	Sensitivity to hypercarbia
↓	Sensitivity to hypoxemia
↑	Resting respiratory rate
↑	Minute volume
↑	Tendency to develop atelectasis
↑	Oxygen demand
↓	Functional residual capacity (FRC)
Cardiovascular system	
↓	Blood pressure
↑	Heart rate
↑	Cardiac output
↓	Systemic vascular resistance
↓	Ability to vasoconstrict

↓	Baroreceptor maturity
↓	Central venous pressure
↑	Plasma volume
↓	Blood volume
Hepatic system	
↓	Cytochrome P-450 enzyme system maturity
↓	Albumin levels
↓	Drug metabolism
↓	Gluconeogenic ability
Central nervous system	
↓	CNS maturity
↓	Neuromuscular junction maturity
↓	Thermoregulatory centre responsiveness
↑	Susceptibility to hypothermia
Renal system	
↓	Nephrogenesis
↓	Clearance rate
↓	Glomerular filtration rate
↓	Renal plasma flow
↓	Concentrating ability
↓	Filtration fraction
↓	Amino acids and phosphate reabsorption
↑	Proximal tubule natriuresis
↑	Phosphorous concentrations
↓	Creatinine concentrations
↓	Blood urea nitrogen concentrations
Pharmacokinetics	
↑	Drug elimination time
↓	Albumin–drug affinity
↓	Protein-bound
↑	Blood–brain barrier permeability
↑	Body water content

↓	Fat content
↑	Initial volume of distribution for water-soluble drugs
↓	Smaller volume of distribution for lipid-soluble drugs

↑ = Increase, ↓ = Decrease, CNS = central nervous system.

Successful anaesthetic management of the paediatric patient primarily relies on following anaesthetic principles whilst being cognisant pf physiological differences For example: The alveolar rate of rise of inhaled anaesthetic concentrations is faster in infants and children in comparison to adults due to the larger ratio of alveolar ventilation to FRC, greater fraction of the cardiac output to vessel-rich organs, and a greater cardiac output per kilogram. Age also can significantly affect inhalant blood–gas partition coefficients. In young patients the lower blood–gas partition coefficients in children may help explain the more rapid rise of alveolar anaesthetic partial pressure. Despite these differences, inhaled anaesthetic drugs are minimally metabolized and primarily eliminated by the lungs, and are ideal for maintenance of anaesthesia in animals with immature hepatic or renal function with appropriate physiologic support and vigilant monitoring to minimize patient risk.

There is strong evidence that human neonates experience pain and may be more sensitive to pain if left untreated. Based on the similarities of neuroanatomy between mammals it must be assumed that neonatal dogs and cats can experience pain although it may differ from the adult experience (da Cunha 2022)

<u>Practical approach to the paediatric patient:</u>

- Individualise plans.
- Comprehensive history especially compared to siblings and familial history of congenital disease.

- Systematic physical evaluation with focus on the cardiovascular system and ensuring no congenital abnormalities.
- Supporting laboratory data based on assessment.
- Understand the physiological reserves of the various organ systems.
- Patients with abnormalities should be stabilized prior to anaesthesia
- Close monitoring of various organ systems should be performed beginning at the preinduction period and maintained until the patient regains consciousness, prioritising body temperature & normoglycemia in addition to usual standard of care.

Pre-anaesthetic medication:

- In true neonates, mask induction may be performed without stress. Atropine or glycopyrrolate (anticholinergics) should be available for any neonatal and paediatric to maintain a higher heart rate, which is necessary to maintain their cardiac output. Low-dose opioids and/or benzodiazepines are "not" contraindicated in neonatal and paediatric patients.
- The use of alpha-2-adrenergic agonists and phenothiazines (acepromazine) should be avoided in neonates carefully considered in paediatric patients. Alpha-2-adrenergic agonists cause significant vasoconstriction, bradycardia and decreased cardiac output. Acepromazine administration, even in low doses, can produce hypotension that may be challenging to manage.

Anaesthetic maintenance:

- Many problems relate to size related difficulties including management and monitoring strategies. Due to possible drug effects on the respiratory control system (such as hypoventilation) and the potential for difficulties in airway management of small patients, preoxygenation with 100%

oxygen via tight face mask is recommended for all neonatal and paediatric patients prior to induction of anaesthesia and intubation. In addition, tracheal intubation can be difficult, care should be taken for an atraumatic intubation. Use of regular monitoring equipment can be problematic; intravenous catheter placement can be difficult.

- IPPV is generally needed in neonatal puppies and kittens, since neonatal patients tend to fatigue easily with increased work of breathing.
- Rebreathing systems are not recommended for patients smaller than 3 kg unless specially designed for extremely small tidal volume ventilation. A non-rebreathing system, with low dead-space such as a modified Bain system or Ayres T piece can also be used to reduce breathing resistance and to provide manual IPPV.
- Young cats and dogs are frequently anesthetized for numerous procedures including ovariohysterectomy or castration with minimal complications. Propofol, alfaxalone, and etomidate are usually considered safe. Dissociative agents (ketamine, tiletamine) should be avoided in the first 2–3 weeks of life because of an association with high mortality rates.
- Intravenous administration of warm isotonic fluids with 2.5% dextrose/glucose is often recommended. Monitoring of core temperature and blood glucose throughout the procedure should be performed and deviations corrected.
- For any small patient, fluid overload and heparin overdose associated with frequent intravenous catheter flushing should be avoided. Flushing with nonheparinized solutions is recommended,
- Hypothermia occurs readily in these patients because of the greater body surface area, minimum amounts of subcutaneous fat, and their reduced ability to shiver. Providing a warm environment and specific active warming are essential.

- Independent of the coexisting conditions, electrocardiography, capnography, pulse oximetry, core body temperature, and blood pressure should always be monitored. Blood pressure is expected to be slightly lower in cases under 12 weeks. There is a paucity of studies performed regarding the accuracy of the non-invasive blood pressure monitoring in younger cats and dogs. Urinary output and blood gas analysis may also be recommended in specific circumstances.
- Local anaesthetic agents can be used and are very effective in all ages; dose requirements and techniques are similar in neonatal, paediatric, and adults.

Renal disease:

Renal disease is a common pathology - estimated prevalence of chronic kidney disease (CKD) is 0.5–1.0% in dogs and 1.0–3.0% in cats, increasing to 80% among older cats. Renal disease is broadly classified as acute kidney injury (AKI) or chronic kidney disease (CKD).
The kidneys receive and require 20–25% of cardiac output to function adequately. Renal disease and functional impairment occur frequently in veterinary patients, particularly in geriatric populations. General anaesthesia alters haemodynamics and organ perfusion, thus, whilst renal effects of drugs should be considered, the maintenance of normotension, euvolaemia (normovolaemia), and adequate cardiac output sufficient to maintain renal perfusion have the greatest effect on the prevention of renal insult.

Physiological abnormalities commonly associated with acute kidney injury (AKI) from Schroeder (2022)

Abnormalities commonly associated with acute renal injury
↑ BUN
↑ Creatinine
↑, ↓, or – Na^+
↑ K^+
↑ P
↑, ↓, or – urine output
Metabolic acidosis
↑ Respiratory rate
Hypertension or hypotension
Cardiac dysrhythmias
Nausea and vomiting

↑ and ↓ indicate an increase and decrease, respectfully, in a parameter, – indicates no change.

Physiological abnormalities commonly associated with chronic renal disease.

Abnormalities commonly associated with chronic renal disease
↑ BUN
↑ Creatinine
↑ SDMA
↑ or ↓ K^+
↑ Mg
Ca^{2+}
↑ P
Metabolic acidosis
↑ Respiratory rate
Hypertension
Anaemia (chronic)
Uraemic coagulopathy
Uraemic encephalopathy (rare)
Hypoalbuminemia
Dehydration
Nausea, vomiting

↑ and ↓ indicate an increase and a decrease, respectively, in a parameter.

Interventions that decrease cardiac output can similarly affect renal blood flow (RBF) and glomerular filtration rate (GFR). Interventions include general anaesthesia, positive pressure ventilation (PPV), and positive end-expiratory

pressure (PEEP). It is important to monitor and manage blood pressure and cardiorespiratory parameters as surrogate measures of cardiac output to maintain adequate RBF. Optimizing hydration status and achieving euvolemia should attenuate the haemodynamic responses associated with positive pressure ventilation such as decreased venous return and pulse pressure variation, but increased airway and pleural pressures associated with PPV and PEEP can lead to renal vasoconstriction, antidiuresis, and anti-natriuresis. Increasing levels of PEEP can lead to increases in renin and aldosterone and depression of cardiac output, RBF, GFR, and urine volume. When utilizing PPV and/or PEEP, more conservative ventilator settings are recommended.

Pre-anaesthetic stabilization of the renal patient

Ideally AKI and CKD patients should be optimally medically managed prior to anaesthesia and the case delayed or rescheduled until the patient presents less of a risk. If stabilization cannot be achieved, the risk: benefit ratio of the procedure to the patient should be closely considered. Unfortunately, stabilization is often not a possibility, and general anaesthesia may be required in unstable patients, increasing the risk of significant morbidity and potentially mortality.

Preanesthetic stabilization should be individualized; however, attaining normovolaemia optimizes cardiac output and RBF while helping to attenuate variations in blood pressure that may accompany anaesthetic induction and maintenance. Ideally, a patient with CKD and concomitant dehydration presenting for an elective procedure should be admitted to the hospital for 12–24 h to allow for volume repletion. Patients presenting in an emergency with either AKI or CKD should be administered fluids to, at minimum, replace fluid deficits. Fluid choice should be made based on the patient's electrolyte status; typically, a polyionic balanced crystalloid, such as lactated Ringer’s (Hartmann`s) solution, is an excellent choice. Saline (0.9%) may be chosen if

the patient is hyperkalaemic, but care should be taken to avoid worsening any preexisting metabolic acidosis with a large chloride load. Fluid administration should be carefully monitored to overload volume overload avoiding especially in patients with concurrent cardiovascular disease (e.g. B2 HCM in older cats)

Metabolic abnormalities in renal disease.

Metabolic problems that are most relevant to anaesthetic management are anaemia, azotaemia, hyperkalemia and metabolic acidosis

- Patients with CKD may have moderate to severe anaemia owing to increased red blood cell fragility and decreased production of erythropoietin Chronic anaemia is well-tolerated, but with severe anaemia (Hgb <7.0 g dl^{-1}), oxygen-carrying capacity can be critically impaired. Oxygen should be supplemented throughout the entire case: before induction through recovery. Very low Hb concentrations are consistent with significant decreases in oxygen-carrying capacity and may require transfusion of red blood cells to support this. Ideally, anaesthesia should be delayed until the haemoglobin is >7.0 g dl^{-1}.

- Azotaemia may be due to dehydration (i.e., prerenal), intrinsic kidney damage or disease (i.e., renal), or due to urinary obstruction (i.e., postrenal).
    - BUN/urea level is a nonspecific test that may be altered by several nonrenal processes. Creatinine is a biomarker that is more specific to renal disease but may be falsely decreased in animals with cachexia or muscle wasting reference ranges for serum are quite

wide and mild elevations that fall within the reference range may be falsely interpreted as normal renal function.

- Kidney function often needs substantial decline before elevations are detected. There is high individual, age, and breed variability; therefore the trend of serum creatinine in individual patients may be of more value if available.
- SDMA is a biomarker that is a more kidney-specific diagnostic test and is a direct reflection of GFR, elevating earlier in CKD than creatinine. Serum creatinine and SDMA are tools that are used to stage CKD by IRIS guidelines. Patients may then be substaged based on systemic blood pressure and urine protein to creatinine ratio (UP/C).
- Azotaemia can also alter blood–brain barrier - anaesthetic agents which cross the blood–brain barrier can have more profound effects in the presence of azotaemia and impact drug–protein binding.

- Patients with AKI or CKD should have electrolytes assessed prior to anaesthesia.
  - Hyperkalemia is the most life-threatening and may require immediate correction. Comprehensive management discussion is beyond the scope of this presentation but may involve the use of soluble (regular) insulin and dextrose (insulin (0.5–1.0 U kg^{-1}) and dextrose (1–2 g per unit of administered insulin, diluted at least 1:3 due to hyperosmolarity)).
  - Acidosis worsens hyperkalemia by shifting potassium extracellularly - thus during anaesthesia, mechanical hyperventilation should be initiated to avoid the addition of respiratory acidosis through respiratory depression-induced

hypercapnia, or a slight compensatory respiratory alkalosis can be induced through mild hyperventilation. Intravenous administration of sodium bicarbonate can also be used but evidence is very limited.

  - Calcium administration does not directly affect serum potassium concentration, the presence of hypocalcemia can potentiate the cardiovascular effects of hyperkalemia, such as bradycardia or ECG changes; if cardiovascular effects are present, this should be the first line of therapy. To counteract adverse cardiac effects, 10% calcium gluconate can be slowly administered (0.5–1.5 ml kg^{-1} IV over 20–30 minutes)
  - Induction of anesthesia should not be attempted in patients with serum potassium concentrations exceeding 6.0 mEq l^{-1}.

- Acid–base disorders and dehydration should be corrected before general anaesthesia and ventilation controlled to prevent a respiratory acidosis worsening acidaemia during anaesthesia. End-tidal carbon dioxide should be monitored and maintained at approximately 30–35 mmHg, and in cases of moderate to severe metabolic acidosis, arterial blood gas measurements should be taken.

- Hypertension is common in CKD patients and cause shifts in individual organ autoregulatory ranges - the patient with chronic hypertension may have autoregulatory ranges shifted to a higher range from chronic compensation. These patients may not tolerate hypotension to the degree of a healthy patient and may require earlier intervention in the event of hypotension. Attempts should be made to maintain mean arterial blood pressure within approximately 20% less than baseline (ideally at least 70–80 mmHg). Many patients with underlying renal disease are treated

with angiotensin-converting enzyme inhibitors (ACEIs), such as enalapril or benazepril, or an angiotensin receptor blocker such as telmisartan. Refractory hypotension may occur under general anaesthesia if these agents are not discontinued 12-24 hours prior to anaesthesia. Even with appropriate withdrawal of these agents, significant hypotension can occur, and vigilant monitoring of blood pressure is required.

Pharmacological approach:

- Plans should be based on the individual
- Temperament must be considered
- Opioid + benzodiazepine in premedication or at induction in very sick patients.
- Low doses of alpha 2 agonists can be utilized depending upon the pathology (not if arrhythmic), co-morbidities and temperament.
- Following sedation and intravenous catheter placement, 100% oxygen should be supplemented by face mask, especially in anaemic animals, where oxygen-carrying capacity is compromised.
- Intravenous propofol (2–6 mg kg^{-1} IV) or alfaxalone (0.5–2 mg kg^{-1}) utilised to facilitate intubation and transition to volatile agent.
- Intravenous fluids should be administered on an individual basis, but rates may need to be higher than usual, dependent upon co-morbidities.
- Anaesthetic monitoring depends on the severity of the patient's condition, the procedure and any coexisting disease.
    - Capnography, pulse oximetry, and non-invasive blood pressure monitors should be placed. Where hyperkalaemia is or has been present, an ECG should be closely monitored for dysrhythmias.
    - Invasive monitoring may be appropriate if available.

Hepatic disease

Liver dysfunction can range from an elevation in liver enzymes with no clinical disease to fulminant liver failure. Anaesthesia is less commonly required in cases with severe liver disorders leading to hepatic failure such as cirrhosis or hepatitis. However, many liver conditions require surgical interventions such as liver biopsy or portosystemic venous shunt attenuation, require sedation or general anaesthesia. Blood flow to the liver comprises 25–30% of total cardiac output. The portal vein accounts for 75% of the blood flow, but, owing to the relatively low oxygen saturation of portal venous blood, provides only 50–55% of hepatic oxygen requirements. The hepatic artery provides only 25% of blood flow, but 45–50% of oxygen requirements. Owing to the constitutively high hepatic blood flow, increases in hepatic oxygen demand are met with increased oxygen extraction from the blood rather than further increases in blood flow to the organ itself.

Liver functions:

- bile formation and excretion
- metabolic functions
- plasma protein synthesis including albumin and coagulation factors
    - All coagulation factors except III, IV, and VIII (thromboplastin, calcium, and von Willebrand factor) are synthesized by hepatocytes and have relatively short half-lives
        - Coagulation factors may be affected by acute liver disease
    - Liver disease can cause hypercoagulable state
    - Hypoalbuminemia, azotaemia, and acidosis reduce drug binding to albumin. This subsequently increases the free drug.
        - Albumin has a half-life of approximately 21 days; due to this prolonged half-life, acute liver disease typically will not manifest with hypoalbuminemia
- glycogen storage and gluconeogenesis

Although the liver serves a wide array of functions, it has substantial reserve, and significant disease may be present before clinical abnormalities are evident.

Biochemical markers and commonly encountered abnormalities associated with hepatic dysfunction.

Biochemical markers	**Clinical signs and abnormalities**
↑ Ammonia	Hepatic encephalopathy
↑ Bile acids	Ascites
↓ BUN	Hypotension
↓ Cholesterol	Impaired drug biotransformation
↓ Blood glucose	Coagulopathy
↓ Serum albumin	
↑ Coagulation times (PT/PTT)	
↓ K	
↑ AST, ALT, ALP, GGT (nonspecific and often do not reflect true liver function)	

Hepatic enzyme elevations suggest hepatocellular damage rather than true dysfunction. The absolute values of these enzymes can be related to the extent of hepatocellular injury but not the reversibility of injury; Serum alkaline phosphatase (ALP) is produced by the liver, bone, small intestine, and kidney. Mild elevations in this enzyme are consistent with hepatocellular injury, but elevations may also be present in paediatric patients, patients with bone disease, or patients chronically administered drugs such as phenobarbital, phenytoin, and corticosteroids. GGT is generally associated with cholestasis or biliary hyperplasia and is a more sensitive biomarker for hepatobiliary disease than ALP in cats. Hepatobiliary dysfunction can be assessed by pre- and post-prandial measurement of bile acids. Elevations in pre-prandial and postprandial bile acids can be present in portal venous shunts, cirrhosis, hepatic necrosis, and inflammation, steroid hepatopathy, cholestasis, and intestinal obstruction

<u>Hepatic Dysfunction and Pharmacological Agents</u>

- A comprehensive review is beyond the scope of this presentation. Nearly all injectable anaesthetics and analgesics rely on varying degrees of hepatic biotransformation for drug elimination, but most do not cause

hepatotoxicity. Hepatic injury may not adversely impact the enzymatic processes of the liver, disease states that impact hepatic function such as portosystemic shunt (PSS) may result in altered drug pharmacokinetics and exaggerated or prolonged effects.

- Alterations in hepatic blood flow are associated with general anaesthesia. Decreases in cardiac output and mean arterial pressure by volatile anaesthetics subsequently decrease portal blood flow in a dose-dependent manner
- Drugs with high intrinsic clearance relative to hepatic blood flow, such as morphine and ketamine, may have exaggerated or prolonged effects in the perioperative period. Factors, including extrahepatic clearance of drugs, protein binding, and autoregulation of local blood flow, can profoundly affect the overall impact on drug metabolism.

General comments on pre-anaesthetic medication (not detailed)

Acepromazine metabolism is via hepatic biotransformation, and no antagonist is available. Caution should be exercised when administering to patients with hepatobiliary disease. The direct effects of acepromazine on hepatic blood flow and hepatic function have not been well characterized. Vasodilation may lead to systemic hypotension. Caution should also be exercised in acepromazine administration prior to laparoscopic procedures, as systemic vasodilation may lead to splenic engorgement.

Alpha-2-adrenergic receptor agonists such as dexmedetomidine offer excellent sedation but with significant cardiovascular depression. Alpha-2-adrenergic receptor agonists significantly decrease heart rate and cardiac output while increasing systemic vascular resistance, but most studies suggest that hepatic blood flow may be well-preserved. Careful dosage titration to the desired clinical effect and close monitoring of sedated patients are advised.

Benzodiazepines, such as midazolam and diazepam, are gamma-aminobutyric acid (GABA) agonists. These agents have minimal cardiovascular effects and are generally well tolerated in patients with many disease states. Fulminant liver failure has been associated with oral administration of diazepam in cats. Metabolism occurs through hepatic biotransformation and a GABA receptor antagonist, flumazenil, is available if sedation is prolonged or excessive. Care must be exercised in patients with HE. Neurons from animals with HE have increased sensitivity to benzodiazepines and increased “GABAergic tone” due

to the presence of endogenous benzodiazepines, and the administration of exogenous benzodiazepines can aggravate HE.

Opioids rely on hepatic biotransformation, and the duration of effect may be prolonged in cases of liver dysfunction and patients with HE may demonstrate an increased sensitivity to opioids, owing to increased opioid receptor density in the brain Naloxone, a nonselective opioid receptor antagonist, may be given if drug duration is prolonged or opioid agonist effects are profound.

Nonsteroidal anti-inflammatory drugs (NSAIDs) have the potential to cause idiosyncratic hepatotoxicity with clinical signs of hepatic dysfunction and liver enzyme elevation, largely due to oxidative stress. Caution should be used when administering NSAIDs to animals with liver disease owing to the potential for this hepatotoxicity, their potential to impact coagulation (via platelet function) and the potential for toxicity in the face of hypalbuminaemia.

Surgical Effects

- Stress associated with surgery as well as mechanical factors can greatly impact hepatic blood flow.
- Hepatic vasculature is richly innervated with alpha-1-adrenergic receptors; activation of the sympathetic nervous system associated with surgical stress and pain may lead to catecholamine release resulting in an initial vasoconstriction, thereby reducing hepatic arterial flow
- Intra-abdominal surgery can result in decreased hepatic blood flow due to the direct compression of hepatic vasculature by surgical manipulation; direct compression of vasculature leading to the liver either by manipulation of large tumours or by surgical instruments or hands may temporarily decrease arterial supply, portal blood flow, or venous return.
- Abdominal insufflation, utilized with laparoscopic techniques, may induce temporary increases in intra-abdominal pressure (IAP) that may compromise splanchnic and abdominal organ blood flow.

Practical Management of Patients with Hepatic Dysfunction

- Ensure adequate history, clinical examination and relevant laboratory date considering risks of hypoglycaemia, hypoalbuminaema, and coagulopathy.

- Assess the degree of hepatic dysfunction
- Duration of effect of many drugs may be prolonged if liver function is impaired.
- Prepare for management of hypotension (i.e., positive inotropes, vasopressors)
- Choose reversible drugs for premedication/sedation
- Use adjuncts to decrease inhalant requirements
- Invasive blood pressure measurement should be utilized for surgical procedures
- Watch for hypoglyaemia and hypothermia
- Positive pressure ventilation when surgery is performed on the liver
- Manage coagulopathy with plasma, vitamin K1, blood transfusion, as necessary

Further reading

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PROCEEDINGS 1st ESAVA CONGRESS

Canine lymphoma- from the basics to beyond

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Canine lymphoma is the most common hematopoietic malignancy in dogs and a defining feature of this disease is its marked heterogeneity, encompassing a wide spectrum of morphological, immunophenotypic, molecular, and clinical entities:

- Morphological diversity: Tumours range from small, indolent lymphocytic proliferations to aggressive large cell lymphomas.
- Immunophenotypic variation: The majority are B-cell lymphomas, but T-cell and rarer null or biphenotypic lymphomas also occur.
- Molecular complexity: Genetic alterations, clonality patterns, and gene expression profiles vary widely, including cases with dual B- and T-cell marker expression suggesting aberrant differentiation.
- Clinical behaviour: Indolent forms (e.g., T-zone lymphoma) contrast with aggressive subtypes such as diffuse large B-cell lymphoma (DLBCL), leading to markedly different survival outcomes.

This heterogeneity leads to differences in biological behaviour, therapeutic response, and prognosis.

Classification systems

The World Health Organization (WHO) classification, adapted from the Revised European-American Lymphoma (REAL) system, is the current gold standard for categorising canine lymphomas. It integrates histomorphology, immunophenotype (B vs T lineage) and clinical features.

Application of this system in veterinary medicine has improved diagnostic precision and prognostic stratification. Common WHO-recognized subtypes in dogs include: diffuse large B-cell lymphoma (DLBCL), marginal zone lymphoma, follicular lymphoma, peripheral T-cell lymphoma (PTCL) and T-zone lymphoma (TZL). Despite its utility, limitations remain, particularly in cases with atypical or overlapping immunophenotypes.

Clinically, canine lymphoma can also be categorised based on anatomical presentation. The most common form is peripheral nodal (or multicentric)

followed by gastrointestinal, mediastinal (thymic or mediastinal lymph nodes) and extranodal (e.g., cutaneous, CNS, ocular).

Each anatomical subtype correlates variably with immunophenotype and prognosis. For example, peripheral nodal lymphoma is often B-cell in origin, whereas mediastinal forms are frequently T-cell.

Diagnostic modalities

Accurate diagnosis requires integration of cytology, histopathology, immunophenotyping, and molecular techniques.

Cytology remains the diagnostic cornerstone with high sensitivity for the diagnosis of high-grade lymphoma. Histology is more often used when low-grade lymphomas are suspected, enabling evaluation of tissue architecture and mitotic activity.

Immunohistochemistry (IHC) involves antigen detection using antibodies such as CD3 (T-cell marker), CD79a, CD20, PAX5 (B-cell markers). IHC allows for lineage determination but also spatial localisation of antigen expression within tissue, which can help distinguishing neoplastic from reactive processes.

Flow cytometry (FC) has emerged as a rapid and highly informative diagnostic tool, particularly using fine-needle aspirates. It enables quantitative immunophenotyping, assessment of cell size and complexity and detection of aberrant antigen expression. It is especially useful for determining lineage (B vs T cell) and identifying prognostic markers such as Ki-67 and CD45 (lost in T-zone lymphomas). However, FC has limitations in distinguishing WHO histological subtypes and must be interpreted alongside cellular morphology.

PCR for antigen rearrangement (PARR) is a molecular assay used to detect clonality by identifying rearrangements in immunoglobulin (IgH) or T-cell receptor (TCR) genes. The main utilities include: differentiation of reactive vs neoplastic lymphoid populations and confirmation of lineage in ambiguous cases.

Treatment modalities

Multi-agent chemotherapy remains the standard of care for high-grade lymphomas, with CHOP-based protocols (cyclophosphamide, doxorubicin, vincristine, prednisone) widely used. This protocol is associated with high response rates (up to ~80–90%) in B-cell lymphoma and median survival times often 10–14 months for high-grade disease. T-cell lymphomas generally exhibit

poorer responses and shorter survival. Recent advances have introduced targeted agents such as rabacfosadine (Tanovea®) a nucleotide analog selectively targeting lymphoid cells, showing efficacy in relapsed and first-line settings.

Verdinexor (Laverdia®) is a selective inhibitor of nuclear export (SINE) targeting XPO1, disrupting oncogenic signalling pathways. This drug provides an alternative to conventional chemotherapy, particularly in chemoresistant cases. Response rates are in the region of 35% in the relapse setting.

Half-body radiotherapy (HBRT) is used as an adjunct or alternative in selected cases, particularly as consolidation therapy or in the relapse setting. HBRT can provide systemic disease control while minimizing toxicity compared to total body irradiation.

Low-grade (indolent) lymphomas are typically treated with a combination of chlorambucil-prednisolone.

Prognostic factors

Prognosis in canine lymphoma is multifactorial, influenced by:

- Immunophenotype: B-cell lymphomas generally have better outcomes than T-cell lymphomas.
- Histologic grade: High-grade lymphomas are more aggressive but often more chemosensitive.
- Clinical stage and substage (presence of systemic signs)
- Molecular and proliferative markers: e.g., Ki-67, CD25 expression via flow cytometry
- Response to therapy: complete remission is associated with improved survival

Conclusions

Canine lymphoma represents a heterogeneous group of malignancies requiring an integrated diagnostic approach. The WHO–REAL classification provides a framework, but accurate characterisation depends on combining histopathology, immunohistochemistry, flow cytometry, and molecular diagnostics such as PARR. Advances in therapeutics, including novel agents like rabacfosadine, verdinexor and HBRT provide new treatment options for these patients.

6th MAY 5pm – Urinary tumours in dogs- an update on diagnosis and treatment

Urothelial carcinoma (UC), also referred to as transitional cell carcinoma, is the most common malignant tumour in the lower urinary tract, accounting for 80-85% of tumours in this location. Other tumours such as squamous cell carcinoma, leiomyoma/sarcoma, rhabdomyosarcomas and lymphomas can occur but are less common. UC arises from the urothelial lining and demonstrates a locally invasive behaviour with metastatic disease seen in less than 35% of dogs.

Clinically, urothelial carcinoma often presents with nonspecific lower urinary tract signs that may mimic benign conditions such as urinary tract infection or urolithiasis. Common presenting signs include haematuria, dysuria, pollakiuria, and stranguria. In more advanced cases, patients may exhibit urinary obstruction, weight loss, lethargy, and signs associated with metastatic disease, such as respiratory distress or lameness. Because of this overlap with inflammatory conditions, diagnosis is frequently delayed until the disease is at an advance stage.

Diagnostic approach and clinical staging

Diagnosis of urothelial carcinoma relies on a combination of imaging, cytology or histopathology. Abdominal ultrasound is often the first-line imaging modality, allowing visualization of bladder wall thickening, masses, or urethral involvement. However, definitive diagnosis requires tissue sampling. Several techniques are available, each with specific advantages and limitations.

Traumatic catheterisation has historically been used to obtain cytology samples from urinary masses. This technique involves passing a catheter to intentionally disrupt the mucosa and collect exfoliated cells for cytological examination. While it can yield diagnostic material, it is less specific than histopathology and may cause discomfort or complications. Ultrasound-guided endoscopy forceps biopsy provides a good minimally invasive alternative technique; it allows for a biopsy to be obtained and decreases costs (compared to cystoscopy).

Cystoscopy is a valuable minimally invasive diagnostic tool that enables direct visualization of the bladder mucosa and targeted biopsy. It is particularly useful for identifying small or early lesions that may not be apparent on imaging.

Fine-needle aspiration can be performed in selected patients, however, the possible risk of tumour seeding along the needle tract needs to be taken in consideration, although this risk is generally low.

Molecular diagnostics have become increasingly important in the evaluation of urothelial carcinoma. One of the most significant findings, is the presence of the BRAF mutation in 65-85% of dogs with UC. This mutation can be detected in urine samples using non-invasive assays and serves as a highly sensitive and specific biomarker for UC.

Accurate staging is critical for determining prognosis and guiding treatment decisions. The tumour-node-metastasis (TNM) staging system is widely used. The primary tumour (T) is assessed based on depth of invasion into the bladder

wall and surrounding tissues. Tumours confined to the mucosa or submucosa are considered lower stage, while invasion into the muscularis or beyond indicates more advanced disease. Regional lymph node involvement (N) and distant metastasis (M), commonly affecting lungs, liver, or bones, further define disease stage.

Treatment options

Treatment of UC depends on tumour stage, location, and patient factors. Complete surgical can be an option for apical bladder masses, however this is often not an option particularly when the trigone is involved. Therefore, medical management plays an important role.

Chemotherapy remains a cornerstone of treatment. Agents such as vinblastine, carboplatin and mitoxantrone are commonly used, often in combination with nonsteroidal anti-inflammatory drugs (NSAIDs), which have demonstrated antitumour effects. Chemotherapy can lead to partial remission or disease stabilisation, improving quality of life and survival times (MST: 7-11months).

Targeted therapy has emerged as a promising approach in recent years. Lapatinib, a tyrosine kinase inhibitor targeting epidermal growth factor receptor (EGFR) and HER2, has shown efficacy in some dogs with urothelial carcinoma. Its use is particularly relevant in tumours expressing these receptors, offering a more tailored therapeutic strategy. While not universally effective, it represents an important advancement in precision oncology.

Radiotherapy plays a palliative and sometimes definitive role in managing UC in dogs by reducing tumour size, alleviating urinary obstruction, controlling pain, and improving quality of life, often combined with chemotherapy or NSAIDs, though outcomes vary due to tumour invasiveness, location, and risk of radiation induced side effects.

Placement of urethral stents and cystostomy tubes can be considered in dogs experiencing severe urinary obstructions as a result of the tumour with the aim to improve signs of severe dysuria and stranguria.

Prognosis

Tumour stage at diagnosis is one of the most significant determinants of outcome, with advanced-stage disease associated with poorer prognosis. Tumour location, particularly involvement of the trigone or urethra, is also associated with reduced

survival due to increased likelihood of obstruction and limited treatment options. Histological grade and response to therapy further influence prognosis.

Conclusions

Urothelial carcinoma is a complex malignancy requiring a multimodal diagnostic and therapeutic approach. Advances in imaging, minimally invasive biopsy techniques, and molecular diagnostics have improved early detection and characterisation of the disease. While chemotherapy remains the mainstay of treatment, targeted therapies such as lapatinib and radiotherapy offer new opportunities for improved outcomes.

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Fracture management - don't panic! From presentation to surgery

Initial triage & management

An initial *major body systems* assessment should be prioritised to identify and treat any potentially life-threatening injuries which includes a cardiovascular, respiratory, abdominal and neurological assessment. Any non-ambulatory limb should have a quick *deep pain assessment prior to administration of opioids.* Large wounds, grazes, or open fractures should be covered with sterile dressings/support bandages during triage, and then be fully assessed and treated when stable. A minimum database including PCV/TS should be checked at presentation and usually repeated at 24 hours. *What next? Typical scenarios are:*

- Stable animal, with fracture or multiple fractures.
- Soft-tissue surgery is suspected (diaphragmatic rupture, urinary tract trauma significant wounds etc) and appropriate diagnostics such as thoracic radiographs, abdominal ultrasound, retrograde studies are prioritised once stable. Fracture planning can be delayed by up to 5 days.
- Stable animal, open fracture requires management prior to fracture surgery – you need to urgently decontaminate the wound.
- Fracture repair pointless – there is significant damage to the nerve supply to the limb; there are overwhelming soft-tissue injuries; a blood supply to the distal limb can't be found via doppler or pin prick test.
- It is an 'urgent fracture'; young animals, articular fractures and suspected nerve entrapment require surgery as soon as possible.

Diagnostic Imaging

Most fractures can be accurately evaluated plain radiography:

1. Radiographs under GA so you can fully examine the animal prior to taking the radiographs – there may be additional injuries to find, and you have move time under GA to ensure you get good radiographs for fracture planning.
2. Always obtain orthogonal views of the region of interest.
3. Centre your radiograph on the area of trauma/fracture but collimate widely to get a better survey.
4. Always take the contralateral limb – this helps you compare for breed variation, growth plates lines, and shows you what the bone was like before it was fractured –plate contouring or making measurements for implants.
5. Have micropore tape and a range of sand-bags and sponges for positioning.

6. Obtain diagnostic quality radiographs
7. Trauma or unobserved cause of the fracture – take thoracic radiographs as well.
8. Do I need a CT - probably not.

Wounds and Open Fractures
Protect the exposed tissues (including bone) with sterile lube, clip and prep the surrounding skin, copious lavage with sterile saline, bacteriology swab at the end of flushing and then application of a sterile dressing. The urgency is the decontamination of the site and wound management; you can take your time over planning the fracture repair.

What to do whilst you wait for orthopaedic surgery
A supportive dressing (*splinted modified Robert-Jones*, or Robert Jones) works for fractures below the elbow and the stifle. Providing a splinted support is particularly important in the distal limb as there is little soft tissue and fractures can puncture through the skin, or the distal segment can rotate and cut-off the blood supply. For all other fractures, a bandage simply makes the limb into a pendulum and worsens the situation. It is better to provide padded cage rest and good analgesia.

Would I manage this fracture with external coaptation or rest alone?
Most of the time the answer is no. Due to the need to immobilize the joint above and below, only tibial and radial fractures can be managed with external coaptation without hugely incapacitating the animal. The ideal scenario is a relatively young animal with rapid healing potential, a minimally displaced ideally interdigitating fracture and an intact paired bone. Fractures of the metacarpus/metatarsus can be easily stabilized with a modified Robert-Jones and a green gutter splint. Fractures that are non-displaced with a paired bone intact in a very young animal could be amenable to VERY strict cage rest alone. Likewise ‘green-stick’ and selected pelvic fractures particularly if animals are starting to mobilise and the pelvic canal is not significantly narrowed.

Would my approach be internal fixation – plates, screws, pins and wire.
In referral orthopaedics this tends to be default as most animals start walking again within 24 hours after internal fixation, the fractures heal well, and there are few complications. For articular fractures internal fixation is a must. The issue with internal fixation and in particular plates and screws is the big range of equipment tailored to the size of the implants. For instance, when considering the most widely used plating systems, a toy breed dog or cat may need a 1.5mm or 2.0mm screws and appropriate plate, but large dogs may need 3.5mm, and there are 2 further sizes between. Each size requires different tooling and sets of screws, and not to mention more advanced locking screw systems now available. Some

growth plate / Salter-Harris fractures are very amenable to simple fixation often with only crossing pins/Kirschner wires, examples including distal tibial, distal femur, femoral head physes, or the proximal tibial crest physis/apophysis. These growth plate fractures will heal quickly and don't want large damaging implants placed. It is important to radiograph at an early stage, often 3-4 weeks and consider removal to allow ongoing growth.

Would my approach be external fixation – external fixators

External fixators are very adaptable but prone to frequent minor complications – usually associated with pin tract infections or loose pins. To improve their stability, they can be combined with an intramedullary pin which is bent into and bolted to the fixator (femoral and humeral fractures). The equipment outlay is minimal compared with internal fixation and most of the implants are either re-usable (clamps and bars) or used and disposed (pins and putty). They have 4 key advantages:

- Implants are not permanent therefore they are good for open/contaminated fractures
- The pins can be placed and then the fracture alignment can be checked with radiographs and adjustments can be made immediately
- They can be altered over the duration of healing to accelerate healing by removing some of the apparatus
- They don't significantly damage the surface of the bone so may have more rapid healing.

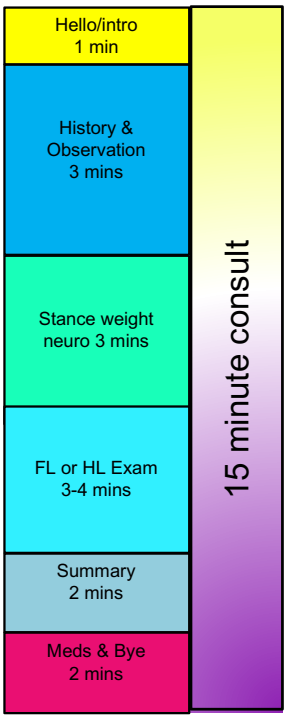
I would consider a simple external fixator for an immature animal with rapid growth in preference to external coaptation or internal plating, as if there is significant growth to occur and the implant could be in place for many years. I would consider external fixators for open fractures and for situations where adjustment is desirable, and they are also useful in the mandible and for operative metatarsal/metacarpal fractures.

<u>The orthopaedic examination - how to be efficient and effective</u>

The typical veterinary consultation ranges from 5-15minutes long. A ***Screening Orthopaedic Examination*** aims to incorporate meaningful orthopaedic assessment within this time frame.

Introductions 1 minute; History and patient observation; 4 minutes
Keep an eye on the dog or cat in your consult room as they acclimatise to the room and you start to take a history from the owner. Dogs and cats with orthopaedic problems may be obviously lame, or show other signs of a problem, such as hovering their leg when standing, or sitting unevenly. The necessary questions for a capsule history is captured in the following mnemonic: LIMB

- L – Location: Which limb or joint is affected?
- I – Initiation: How and when did it start?
- M – Movement pattern: How is gait affected?
- B – Background: Any relevant history (previous injuries, workload, changes)?
- If a re-visit appointment, then apply LIMB**S** – **Sensitivity** to treatment



Owners can also usually correctly identify a fore vs hindlimb lameness, however it is not uncommon for them to assign the lameness to the non-lame limb. Owners may have noticed clicks or clunks – which could be due to a dysplasia hip or an unstable meniscal tear. It is of course important to be wary of 'pattern-recognition', however certain orthopaedic common orthopaedic conditions are seen more commonly in certain signalments or presentation characteristics.

A comment on *Visual Gait Assessment.* Often considered an essential part of the orthopaedic exam and it certainly has value, it has significant time costs associated. Interestingly, there is a body of research evidence indicating that it is an inconsistent and unreliable method to measure and grade severity of lameness.

Stance, weight distribution, neuro screen; 3 minutes
I tend to do most of my examinations with the dog or cat standing and usually with the dog on the floor – even the small ones. Typically, I stand behind the dog or cat, with the owners at the 'head-end'. I tend to avoid the lying down

exam, as it can be time consuming to get the dog or cat on its side, and keep it there. Although a lying-down exam allows easier assessment of each limb, it severely hinders the ability to compare the *symmetry between right and left limbs simultaneously*. Whilst standing, *stance* can be assessed by carefully pushing the paw away and determining how much resistance there is on each leg, or rocking the dog from side to side. Now is also the time to feel the musculature for muscle atrophy. Likewise, it can be an ideal time to palpate the musculature between the right and left limbs simultaneously. Muscle atrophy on the forelimbs is most obviously appreciable over the scapula – a more prominent scapular spine is felt on the atrophied side. For the hindlimbs, orthopaedic muscle atrophy is most obvious by comparing the girth of musculature over the thigh – comparison of hamstring and quadricep muscle volumes with your hands. (Red-Flag is extreme by focal muscle atrophy – this is less likely to be 'disuse wastage' from lameness and could indicate a neurologically driven muscle atrophy). At this point, a brief neurological exam is performed. Conscious proprioception (paw knuckling test) is a sensitive if not specific means to identify a problem with the neurological system. If there is delay in paw repositioning, check again but support the dog's weight. This is followed by spinal palpation, which should include head-neck and tail range of motion.

Forelimbs examination only 3 minutes ; OR Hindlimbs examination only ; 3 minutes

By this point you will most likely know whether you are most interested in the forelimbs or the hindlimbs. To keep within the 15-minute consult window, you are better-off being thorough on either fore OR hindlimbs IF you have a clear indication of whether a fore or hindlimb issue. A complete examination is always preferable, however in a pragmatic screening scenario, this is most likely sufficient for this stage. It is recommended to evaluate the contralateral good limb first. … I start distally at the digits and work proximally – and don't forget to use your eyes – some causes of lameness you can see, such as corns!

For commonly affected joints of the carpus, elbow, tarsus and stifle, detecting a joint effusion is normally non-painful for the animal, and a very specific means to identify the disease location – the lecture will indicate where to palpate joint effusions and gives tips on palpation. For instance, for dogs with cruciate disease, most conscious dogs will NOT tolerate a correctly performed cranial draw test, although many will tolerate a tibial thrust test and therefore this is the most useful test in the consult room. I often find I can get a good tibial thrust with larger dogs when they are standing upright. If they are unwilling, I will not force the issue – these tests are most reliable and accurate when performed under sedation, however other clues can significantly help you increase your suspicion of cruciate rupture. Therefore, with the conscious animal in the consult room, palpation alone

may be the best you can get, but can often give you a clear indication of the likely diagnosis. Check for stifle effusion by feeling either side of the patella ligament. A normal dog will have a clear, clean border and you should feel a depression on either side. Patellar luxation rarely creates a large effusion, and won't create a medial buttress (typical of the more chronic cruciate disease stifle), which will feel like a hard, almost bony circular prominence over the medial collateral region, and is best assessed by comparing to the contralateral limb. Red Flag – pain reaction to a joint or to focal bone pressure – should consider radiographs.

Change in joint range of motion and understanding what is normal is also very helpful, as are specific manipulation tests including: Cruciate instability – cranial draw/tibial thrust; Shoulder disease – biceps tendon test, should abduction; Hip dysplasia – in young dogs identifying an Ortolani sign/clunk; Patella luxation manipulation and grading the luxation.

Summary for Client ; 2 minutes
It is important to finish the consult with a clear plan based on your findings; which leg you think is the problem, and the location or type of issue; muscular, joint, bone. Clarify your confidence in your screening exam and the subsequent plan:

- Presumptive diagnosis
    - Book back for confirmation work-up/radiographs etc
    - Treatment trial with follow-up
- Unclear diagnosis
    - No Red Flags – re-assurance treatment trial; rest, NSAIDs etc, follow-up
    - No Red Flags but owner wants full work-up
    - You have concerns – return for full work-up
- Normal – or no indication
    - Reassurance ruled out common/concerning
    - Benign neglect
    - Follow-up if concerns remain
- Suspect neuro
    - Book back for full neuro-ortho exam

Medication and goodbyes ; 2 minutes
<u>Osteoarthritis management in 2026 - what do you need to know?</u>

Osteoarthritis (OA) isn't just wear and tear – it is a chronic inflammatory disease and should be managed accordingly. Inflammation is the key and earliest driver of OA. Cytokines and inflammatory cells orchestrate tissue damage and structural dysfunction; damage-associated molecular patterns (DAMPs) signal via Toll-like receptors to engage innate immunity. These

processes directly activate nociceptors while up-regulating central and peripheral sensitisation pathways. OA pain is a combination of nociception but also a chronic inflammatory pain state of "wind-up." Reduction of inflammation should therefore be a key objective in OA management wherever possible. Primary options are non-steroidal anti-inflammatory drugs (NSAIDs), prostaglandin-receptor antagonists (piprants such as grapiprant/Galliprant), and controversially and usually only in end-stage cases, intra-articular corticosteroids. Paracetamol is not an anti-inflammatory, so has no impact on the inflammation driven processes in OA. Helpful adjuncts include omega-3 supplementation and adipose reduction (weight loss), which influences the degree of inflammatory response. NSAIDs remain first-line for chronic, inflammatory OA pain with unequivocal evidence of efficacy in OA (including force-plate studies). There is no robust evidence that one class is categorically superior; however a common mistake is the short course of NSAID, whereas evidence suggests 4-6 weeks are needed for best effects. Anti-NGF monoclonal antibodies (bedinvetmab/Librela; frunevetmab/Solensia) are not anti-inflammatories per se. OA up-regulates nerve growth factor (NGF) within joint tissues (including mast cells); NGF binds TrkA to amplify pain signalling and drive peripheral and central sensitisation. There is evidence of good efficacy of clinical improvement in canine OA trials equivalent to that of NSAID therapy, however, there have been recent concerns for rare adverse events.

Exercise and weight reduction is beneficial and necessary for joint health: movement supports cartilage nutrition, maintains flexibility, and strengthens periarticular muscles; it also promotes anti-inflammatory myokines, offers cognitive benefits, and facilitates weight reduction. Human NICE guidance endorses therapeutic exercise for OA. Prolonged lack of activity is harmful to joints: less than six weeks of immobility leads to mild, reversible cartilage atrophy, whereas beyond 6–12 weeks degeneration can become irreversible. High-quality human evidence shows that exercise increases joint motion and decreases pain, without proof that moderate exercise accelerates OA, or that one exercise type is categorically superior. Dogs with OA should have multiple, regular, shorter walks rather than a single long outing (e.g., ~20 minutes three times daily), keep routines consistent, favour soft terrain, warm up gently, and limit off-lead sprints or sharp twists/turns. Do not prescribe complete rest other than during a severe flare-ups. Hydrotherapy provides low-impact resistance and strengthens while improving range of motion; physiotherapy can help, although owner adherence varies. Passive range-of-motion performed for more than three weeks has been associated with improved ROM.

Whilst excess weight does lead to excess joint loading, it is now thought that excess adipose tissue increases adipokines and systemic "metabolic body" inflammation—an inflammatory driver which drives OA and its severity.

Reducing adipose mass lowers these drivers. Clinically, >12–18% body-weight loss significantly improves clinical OA signs, and even >8% loss produces measurable force-plate changes; human studies mirror these effects, with an "effect size" similar to NSAIDs. Achieving and sustaining loss is challenging, however prescription low-calorie diets can deliver ~11% loss when used correctly.

Multimodal but not polypharmacy Multimodal analgesia targets distinct elements of the OA pain syndrome to enhance efficacy and spare side-effects, and hence would include a medication targeting each element:

- Inflammation control (NSAIDs, piprants, omega-3);
- Chronic pain de-escalation (amantadine, gabapentin) to reduce central sensitisation;
- Direct analgesics (paracetamol, tramadol, cannabinoids).

This is different from polypharmacy—i.e., adding more and more medications without clear rationale, including multiple drugs targeting the same element of OA, which risks interactions, adverse effects, and cost without benefit. A sensible plan might include "one from each group," adjusted to response and tolerance.

Treat the dog, not the radiograph, and clinical disease is highly variable and prone to flare-ups

Clinical severity does not track linearly with radiographic changes. Many OA joints are structurally abnormal yet clinically variable, and radiographs are a poor surrogate for pain because they may not display active, painful disease. In people, early bone marrow lesions, which are not seen on radiography, but seen on MRI are highly painful and clinically important, but we do not routinely look for those in our veterinary patients. For day-to-day practice this means focus on pain and function, not the radiographic severity. A radiographically osteoarthritic joint is certainly not normal, but may not be clinically sufficiently important to warrant treatment at that stage. While OA is a pathologically progressive and ultimately a non-reversible disease, the clinical course commonly fluctuates as a series of flare-ups. Historically, practitioners often equated clinical deterioration with inexorably worsening structure, which encourages permanent drug escalation ("set and forget") and polypharmacy. Human studies, including ACT-FLARE, describe flares triggered within <24 hours (e.g., over-activity or "buckle" injury) and lasting more than five days with stiffness and pain. A better model is to treat the flare effectively (which may take several weeks), then titrate therapy down again, recognising that as joint "organ reserve" diminishes over time, a new baseline may require a slightly different or more intensive regimen. Owners should be made aware of

managing flare-up triggers proactively – such as rough play with other dogs, or excessive ball play. In many cases treat for one to three months, address instigators, step down, and repeat at the next flare.

A role for joint supplements? Although glucosamine and chondroitin have a plausible rationale in OA treatment as matrix precursors, high-quality human evidence finds them equivalent to placebo; in dogs. In contrast, omega-3 fatty acids (EPA/DHA) shift arachidonic metabolism towards anti-inflammatory pathways and increase anti-inflammatory cytokines. Several canine RCTs (including force-plate outcomes) demonstrate benefit; use fish-oil sources rather than plant oils, typically around 70–100 mg/kg/day. Human meta-analyses also support an effect. Green-lipped mussel is an omega-3 source.

Can Stem cell therapy regenerate the joint? The notion that stem cells "regenerate" OA joints is highlighted here as a myth requiring critical appraisal. Whilst experimental studies have shown cartilage regeneration when the joint is acutely damaged followed by immediate stem cell administration, this has not been shown in chronically diseased clinical OA joints. However, there is good evidence of a clinically significant benefit from some stem cell therapies in clinical patients. This effect is thought to be the release of a wide range of cytokines and factors that suppress inflammation and immunomodulate the inflammatory OA joint environment. Based on our current understanding, stem cells are better considered as 'cell therapies' or 'biological therapies' and not 'regenerative per se.

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1. POCUS Made Practical – From Probe to Diagnosis (Part 1)

General Concepts of POCUS, Abdominal POCUS, and Vascular POCUS

Point-of-care ultrasound (POCUS) has become a transformative tool in veterinary practice by allowing clinicians to perform rapid, targeted diagnostic assessments directly at the patient's side. The core principle of POCUS is its **focused, problem-oriented nature**. Unlike a full diagnostic ultrasound, which aims for comprehensive imaging, POCUS is designed to answer specific, binary clinical questions such as "Is there free fluid?", "Is the stomach dilated?", "Is the bladder obstructed?", or "Is this patient hypovolemic?". The method is structured, repeatable, and immediately linked to clinical decision-making, making it extremely valuable in emergency and critical care settings but also in general practice.

Understanding the **general concepts** involves recognizing how ultrasound beams interact with tissues and how artifacts form. For practitioners, the most important implication is that many life-threatening conditions generate **high-yield ultrasound findings** that are easy to identify even without extensive specialist training. Indeed, the emergency clinician should be able to answer the **clinical question** with a **high degree of sensitivity** and ideally also specificity. Seeking free fluid is a great example, as this question has clinical relevance (i.e. identifying this in an unstable emergency patient will have an impact on decision making), but moreover the question can be answered with a high degree of sensitivity by practitioners (i.e. if there is fluid, the clinician will typically *see* it). Even more so, identifying free fluid also has high specificity (i.e. if the clinician sees fluid, that typically means there actually *is* fluid). Free fluid, severe organ enlargement, obstructive patterns, and major vascular changes all have unmistakable ultrasound signatures that can be recognized with minimal experience. Equally essential is the understanding that POCUS is intended to **complement—not replace**—other diagnostic modalities. Its strength lies in its **immediacy** and its ability to refine a clinician's differential diagnosis in minutes.

Abdominal POCUS provides rapid information about the presence of effusion, gross organ changes, gastrointestinal abnormalities, urinary obstruction, and major surgical emergencies. Effusion, which appears as black (anechoic) pockets surrounding organs or filling body compartments, is one of the first findings

clinicians learn to identify. Recognizing effusion can indicate trauma, ruptured masses, septic peritonitis, or internal hemorrhage. If not evident from the clinical scenario, the nature of the fluid should be investigated via abdominocentesis (why not POCUS guided 😊) and fluid analysis.

Gall bladder wall edema has been demonstrated to be suggestive of increased hydrostatic pressure (e.g. pericardial tamponade, pulmonary thromboembolism), or increased vascular permeability (e.g. anaphylactic or septic shock).

Stasis of the gastrointestinal tract evidenced by decreased contractions of the duodenum or stomach, or a fluid filled stomach are common findings in the perioperative period and in critical care patients. Identifying dilated, fluid-filled loops with decreased motility may raise concern for obstruction, but this is exactly where POCUS evolves into formal and comprehensive ultrasound, requiring a more advanced skill set to answer this question with a sufficient degree of certainty.

The urinary system is similarly amenable to quick evaluation. The bladder volume can bes assessed and monitored with sequential POCUS studies. This allows to screen at risk patients for the development of oligo or anuria, preventing the use of indwelling catheters which have been associated with high risks of bacterial colonization of the urinary tract. The kidneys can also be quickly screened for pyelectasia, hydronephrosis as well as a set of changes suggestive of acute rather than chronic renal disease in cats. Although this does not replace full abdominal imaging, it allows practitioners to rapidly categorize the severity of disease, prioritize stabilization, and decide whether urgent referral or surgical intervention is needed.

Vascular POCUS focuses on the large abdominal vessels—primarily the caudal vena cava (CVC) and the abdominal aorta—to evaluate volume status and fluid responsiveness or intolerance. The CVC is a dynamic indicator of intravascular volume. A small, collapsing CVC suggests hypovolemia or distributive shock, while a large, non-collapsing vessel may indicate fluid overload, right sided cardiac disease, or obstructive shock such as pericardial tamponade. Although not perfectly sensitive, this method provides information unavailable with other non-invasive and easily accessible techniques, which moreover can be repeated frequently to guide fluid therapy.

By integrating abdominal and vascular findings, practitioners can categorize patients into volume-responsive versus volume-intolerant groups. For example, in a dog with suspected sepsis, a very small CVC and empty cardiac chambers suggest that additional fluids may be beneficial. In contrast, if the CVC is full and

the patient has crackles or B-lines on lung ultrasound, fluids should be limited. This dynamic, individualized approach marks one of the major advantages of POCUS in emergency stabilization.

The main take-home message from Part 1 is that POCUS dramatically shortens the time from clinical suspicion to diagnostic clarity. By providing immediate insight into the abdomen and vasculature, it increases clinician confidence, guides fluid therapy, helps identify surgical emergencies quickly, and improves the overall quality of patient care. It is one of the most impactful skills general practitioners can integrate into everyday practice.

2. POCUS Made Practical – From Probe to Diagnosis (Part 2)

Pleural Space, Lung, and Cardiac POCUS

The second part of POCUS training focuses on the pleural space, lungs, and heart—structures that often threaten the immediate survival of acutely dyspneic or unstable patients. Because radiography can be difficult or dangerous in severely compromised animals, due to the necessity to manipulate the position of the pet which can negatively impact its oxygenation and venous return, POCUS provides a safer and faster alternative for diagnosis.

Lung ultrasound relies heavily on interpreting artifacts rather than anatomic detail. Horizontal A-lines represent normal air-filled lung or a pneumothorax, as they represent reverberation artifacts of the pleural line. B-lines signify interstitial or alveolar fluid or tissue, and are caused by the close proximity of air and fluid or tissue at the level of the pleura. The pattern and distribution of B-lines help distinguish between cardiogenic pulmonary edema, non-cardiogenic edema, pneumonia, or contusions. Consolidation appears as tissue-like echotexture ("hepatization"), allowing differentiation between atelectasis, pneumonia, or mass-like lesions. Shred signs refer to an irregular distal border of the consolidation, often seen in pulmonary infection, contusion or hemorrhage; a ,nodule sign refers to a smooth and round structure, which is suggestive of neoplastic or granulomatous disease; finally the wedge sign refers to a wedge shaped consolidation as is often seen in humans (but rarely in dogs) with thromembolism. The ability to perform these assessments in seconds makes lung POCUS indispensable for triage and stabilization.

Evaluation of the **pleural space** is among the most rewarding aspects of POCUS because pleural effusion and pneumothorax produce unmistakable ultrasound signs. In pleural effusion, the clinician typically sees anechoic or hypoechoic fluid separating the lung from the body wall, and the lung typically appears as a floating, consolidated structure. (Sidenite: in cats with a pyothorax, the cellularity can result in a hyperechoic fluid) This allows rapid differentiation between pulmonary disease and pleural space disease and helps guide thoracocentesis. In contrast, pneumothorax is characterized by the absence of **lung sliding**, a shimmering movement representing the visceral pleura gliding against the parietal pleura. When lung sliding is absent and A-lines dominate the screen, pneumothorax is likely, and identification of a **lung point**—the location where sliding lung meets detached pleura—is considered highly specific for the diagnosis. In the majority of patients with a pneumothorax, the collapsed lung will demonstrate abundant B-lines due to the decreased amount of air present in it, which should allow for easier identification of the lung point. Finally, when

one follows the curtain sign (i.e. the transition of the thoracic in the abdominal cavity), a double curtain sign is a highly specific finding to diagnose a pneumothorax.

Cardiac POCUS, based mostly on the short axis right parasternal views, though not a full echocardiogram, provides vital information on pericardial fluid, chamber size, and contractility. Identifying a pericardial effusion is often life-saving because tamponade can rapidly lead to death if unrecognized. The pericardial sac appears as a dark halo surrounding the heart, and right atrial or right ventricular collapse indicates hemodynamically significant tamponade requiring immediate drainage.

Assessing gross chamber size gives invaluable clues: a small, “empty” heart beating rapidly suggests hypovolemia, while a massively enlarged left atrium points toward congestive heart failure or severe hypervolemia. Contractility can be judged visually; poor contractility may result from cardiomyopathy, sepsis, or severe shock. Integrating this information helps clinicians make immediate decisions about whether fluids, diuretics, positive inotropes, or pericardiocentesis are appropriate.

The synergy of lung, pleural, and cardiac POCUS allows clinicians to rapidly classify respiratory distress into obstructive, pulmonary parenchymal, pleural space, or cardiac origin—even in unstable patients. Similarly, the combination of POCUS findings allows for rapid ultrasound identification of the underlying mechanisms leading to a state of cardiovascular shock. This rapid categorization speeds up treatment decisions and reduces morbidity.

The overarching lesson of Part 2 is that even basic training in lung and cardiac POCUS dramatically enhances diagnostic accuracy and reduces time to intervention, making it an essential skill for modern veterinary general practice.

3. Recognizing and Managing SIRS, Sepsis, and Septic Shock

SIRS, sepsis, and septic shock remain major causes of morbidity and mortality in small-animal patients, making early recognition and intervention critical for improving outcomes. Understanding these syndromes requires a clear distinction between them. **SIRS** (Systemic Inflammatory Response Syndrome) is a non-specific, dysregulated inflammatory response to various triggers, such as trauma, heatstroke, pancreatitis, or infection. **Sepsis** occurs when this response is triggered by infection and leads to life-threatening organ dysfunction. **Septic shock** represents the most severe stage: a state of circulatory and metabolic collapse characterized by persistent hypotension despite fluid resuscitation, often accompanied by elevated lactate and signs of severe perfusion failure.

Clinically, these conditions overlap significantly, and no single parameter confirms the diagnosis. Instead, clinicians must rely on pattern recognition. Typical findings include tachycardia, tachypnea, hypo- or hyperthermia, altered mentation, prolonged capillary refill time, hyperlactatemia, leukocytosis or leukopenia, and hypotension. Not all animals will display the full syndrome, so awareness and suspicion are essential. Early diagnosis is one of the most important determinants of survival.

A major point emphasized in the lecture is the importance of locating the **source of infection or inflammation**. Abdominal sources such as septic peritonitis, pyometra, biliary rupture, or pancreatitis are common in dogs and cats. Respiratory infections and urogenital infections are also major contributors. This is where **POCUS** becomes a critical adjunct. It allows clinicians to detect effusions, guide sampling, identify obstructive processes, and assist in triage, without delaying stabilization. The combination of clinical signs, laboratory data, and POCUS findings greatly enhances diagnostic confidence.

Management hinges on three pillars: **hemodynamic stabilization**, **source control**, and **appropriate antimicrobial therapy**. Fluid resuscitation remains a cornerstone of initial stabilization, but the approach has become more nuanced. Excessive fluid therapy has been shown to worsen outcomes by contributing to edema, including in the lungs and gastrointestinal tract. Therefore, clinicians must tailor fluid administration using perfusion parameters, lactate trends, mentation, and, ideally, POCUS assessment of the CVC, cardiac chambers, and lung fields. Patients with a collapsing CVC and small ventricles are more likely to benefit from additional fluids, whereas those developing B-lines, gall bladder wall edema, pleural or abdominal effusion or exhibiting enlarged atria or stretched ventricles are fluid-intolerant and require more conservative management.

Vasopressors may be required when hypotension persists despite adequate fluid resuscitation. Noradrenaline is typically the first-line agent. In septic shock, early initiation of vasopressors can be life-saving. When the clinician has confirmed the vasodilatory nature of the cardiovascular shock, vasopressors may already be administered as fluid is still being administered. However, administering vasopressors implies the use of syringe pumps for these potent molecules. **Antibiotics** should be administered as soon as sepsis is suspected—ideally within the first hour in unstable patients—after obtaining appropriate cultures whenever possible, without delaying treatment in emergencies. Source control involves surgical exploration, drainage of abscesses or effusions, removal of infected tissue or foreign bodies, and appropriate wound or urinary tract management. Prompt identification and control of the source significantly improve survival.

Finally, these intensive care patients often have multiple organs affected by the systemic effects of the inflammation and/or infection. Hence the clinician should perform regular assessment of organ damage and function, and provide an appropriate treatment plan to prevent or palliate any complications.

Overall, the key lesson is that early recognition, aggressive but controlled stabilization, and rapid source identification dramatically improve outcomes. By integrating clinical assessment with modern tools such as POCUS, general practitioners can identify sepsis sooner, intervene more effectively, and significantly increase survival chances in affected patients.

Transapical Edge to Edge Repair (TEER) for the treatment of mitral valve disease: patient selection, technique, and follow-up

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Chronic mitral regurgitation (MR) is the most common acquired cardiac disease in adult dogs. It's caused by the progressive myxomatous degeneration of the atrioventricular valves, which leads to regurgitation from the mitral valve. Studies on mortality from heart disease in the general canine population show that approximately 7% of all dogs die or are euthanized due to heart failure before the age of 10. It primarily affects small to medium-sized dogs (usually under 20 kg) and those over 8 years of age. In 60% of cases, the degenerative process only affects the mitral valve, while in 30% it affects both the mitral and tricuspid valves. Only 10-15% of cases involve the tricuspid valve alone. The phenomenon of valve incompetence and regurgitation results in prolapse and myxomatous valve degeneration.

The structural and functional changes are progressive and advance as the heart disease worsens. Degenerative mitral valve disease is characterized by a thickening of the valve leaflets, which eventually leads to a failure of proper valve alignment and closure. The lesions are generally smooth and small, creating a small, club-shaped appearance on the tips of the leaflets in the early stages of the disease. However, the lesions can become large and irregular as the disease progresses. Larger lesions are generally associated with more severe insufficiency, but this is not always the case.

Mild mitral regurgitation is well tolerated and does not result in a compensatory response from the cardiovascular system. The worsening of the disease and the increased amount of regurgitated blood lead to a reduction in the systolic anterograde aortic flow, activation of the body's neuroendocrine response, hypertrophy of the heart muscle and severe left atrial and ventricular dilatation.

In mitral regurgitation, the "target organs" are the left atrium and the left ventricle. We can therefore schematically divide the evolution of the disease into three phases:

- **Phase 1 or Initial Phase:** Regurgitation is observed (generally mild or moderate) but left atrial and ventricular dimensions are normal (without substantial volumetric and pressure variations). A decrease in afterload can cause "supernormal" ventricular function and a consequent apparent increase in ejection and shortening fraction. This phase corresponds to the **ACVIM Consensus Phase B1**.
- **Phase 2:** Left atrial dilation and left ventricular volume overload appear. The contractile capacity of the myocardium is preserved thanks to the involvement of intrinsic mechanisms and Starling's Law. Initially, atrial pressure remains unchanged due to compensation mechanisms according to the postulates of Laplace's Law (IB ISACHC or B2 ACVIM Consensus), or the size of the atrium increases without large pressure variations. In this phase, the lymphatic circulation also enhances pulmonary drainage. With disease progression, however, there is a slow and gradual increase in left atrial pressure, an increase in pulmonary blood volume, and a consequent increase in pulmonary venous pressure and pulmonary capillary wedge pressure. This phase coincides with the **ISACHC Phase II** or **ACVIM Consensus Phase C**. Chronic left ventricular volume overload, associated with valve insufficiency, causes eccentric hypertrophy with a progressive tendency in the more advanced phases to shift to the right of the Starling curve with loss of myocardial contractile efficiency.

- **Phase 3:** We find echocardiographic signs of systolic dysfunction with further deterioration of diastolic function.

The evolution and progression of mitral disease are therefore conditioned by the extent of the valve damage, the amount of blood regurgitated into the left atrium, and atrial compliance. These factors influence both the dilation of the left atrial chamber and the increase in left atrial and pulmonary pressure. The progression of the disease accompanies the gradual increase in left atrial pressure, an increase in pulmonary venous pressure, pulmonary capillary wedge pressure, and finally pulmonary arteriolar pressure.

Surgical Therapy

The concept of "edge-to-edge" mitral valve repair was first proposed by cardiac surgeon Ottavio Alfieri as a surgical solution to address the problem of anterior leaflet prolapse. In this repair, the prolapsing leaflet is sutured to the adjacent leaflet to create a double-orifice mitral valve. The success of this procedure as a surgical repair led to the development of transcatheter "edge-to-edge" mitral valve repair (TEER) in humans. The EVEREST study on the feasibility of the MitraClip device in humans was followed by two other trials, EVEREST II and COAPT, which established it as a widely accepted and successful option for the treatment of severe mitral regurgitation (MR) in humans. The success of TEER in humans led to the development of a TEER device specifically for dogs called the **Canine Mitral V-Clamp**. A preliminary feasibility study demonstrated the feasibility of the approach and the device in eight dogs with stage B1 degenerative mitral valve disease. Based on these results, a prospective clinical feasibility study of the V-Clamp device in dogs with severe degenerative mitral regurgitation was undertaken.

Patient Selection

Dogs undergoing mitral valve repair must meet the criteria that identify severe mitral regurgitation (MR), adapted from the American Society of Echocardiography guidelines for severe mitral regurgitation in humans. This could include dogs in **ACVIM stage B2** or **stage C**. Dogs in advanced **stage D** or with **atrial fibrillation** are considered poor candidates for mitral surgery. The determination of the severity of the regurgitation should be based on the consideration of multiple factors, which include:

- Holosystolic regurgitation with an eccentric direction on color-Doppler or central jet area studied with color-Doppler ≥ 50%.
- Peak velocity of the mitral antegrade flow E-wave ≥ 1.0 m/s.
- Dense regurgitant jet with a triangular profile on CW Doppler.
- Regurgitant fraction (RF) ≥ 50% (PISA, volumetric, Doppler).
- Regurgitant volume (RVol) ≥ 1.0 ml/kg.

The study of the regurgitant jet with color-Doppler must be carried out on multiple systolic images with appropriate gain and settings. Evaluation based on a single image can overestimate the severity of MR. The measurement of RF using PISA, Simpson volumetric, and/or Doppler methods provides a quantitative assessment of the severity of the regurgitation and therefore represents an important objective criterion. Measuring RF with multiple methods increases confidence in the evaluation. The width of the vena contracta (VC), RVol, and the effective regurgitant orifice area (ERO) can also be considered, but they depend on the patient's body size, and the limits for severity have not been fully established in dogs. Left ventricular and left atrial dilation (LVIDdN > 1.9, LA:Ao > 2.0) can be considered, but these are secondary variations to MR and depend on other factors such as chronicity and therapy. Left ventricular systolic function indices can be considered, as systolic dysfunction (e.g., LVEF < 50%, LVIDsN > 1.0) can be a negative predictive factor.

Mitral functional anatomy is an important assessment that allows for establishing the possibility of success and the outcome of the procedure in dogs undergoing TEER. Based on the size of the currently available canine device (14 mm, 16 mm, 18 mm, 20 mm<), the antero-posterior (A-P) mesosystolic mitral annulus diameter should be between 14 mm and 20 mm in the standard 2 echocardiographic projection, with visualization of the left outflow. The guidelines for optimal functional anatomy are not absolute but represent a continuum from favourable to less favourable to unfavourable. Dogs with the most favourable functional anatomy can be expected to achieve the best results in terms of reducing MR severity and extending life expectancy with the lowest procedural risk. Dogs with less favourable functional anatomy may benefit from TEER with some associated increase in procedural risk. Dogs with unfavourable functional anatomy carry a higher procedural risk than the potential benefit and are considered inadequate candidates for the TEER procedure. The final evaluation of functional anatomy for repair should be based on pre-procedural transesophageal echocardiography (TEE).

TABLE 1. Factors determining transcatheter edge-to-edge mitral valve repair risks and outcome.

Center experience

Expectation for mitral regurgitation reduction

Non-complex Ideal for TEER	Complex Suitable for TEER	Very complex Challenging TEER	TEER Difficult or impossible
Benefit >>> risk	**Benefit >> risk**	**Benefit ≥ risk**	**Risk > benefit**
■ Focal A2 leaflet prolapse ■ Posterior wall-hugging regurgitant jet ■ Coaptation gap ≤2 mm ■ Commissural vena contracta (VC) width <33% of commissural mitral diameter ■ Preserved coaptation reserve (depth)	■ A2 and P2 prolapse ■ Two-segment anterior leaflet prolapse ■ Eccentric posterior to central regurgitant jet ■ Coaptation gap 2–4 mm ■ commissural VC width 33% to 66% commissural mitral diameter ■ Coaptation reserve (depth) <3 mm	■ A2 or P2 leaflet flail ■ Three-segment anterior leaflet prolapse ■ Two-segment prolapse of posterior leaflet prolapse ■ Central regurgitant jet ■ Coaptation gap >4 mm ■ Commissural VC width >66% commissural mitral diameter ■ Limited coaptation reserve with wide coaptation gap ■ Systolic anterior–posterior (A–P) diameter >20 mm ■ Body weight <4 kg	■ A2 and P2 leaflet flail ■ Severe multisegment leaflet flail ■ Multiple disparate and wide regurgitant jets ■ Deep leaflet cleft(s) or perforation ■ Leaflet thickness >5 mm ■ Coaptation gap >6 mm ■ Posterior leaflet length <4 mm ■ Systolic A–P mitral diameter >22 mm ■ Body weight <2.8 kg

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Procedure Description

TEER in dogs is performed under general anaesthesia via a mini-thoracotomy and a transapical cardiac approach in right lateral recumbency. The optimal intercostal space is typically the 7th intercostal space, confirmed by a lateral fluoroscopic view of the cardiac silhouette and TEE echocardiography. A 3-4 cm intercostal thoracotomy is performed just dorsal to the sternum. The pericardium is opened and sutured to the incision to elevate the cardiac apex. The cardiac puncture site is determined by external compression of the heart during biplane TEE of the left ventricle and mitral valve. The optimal puncture site is determined as being perpendicular to the plane of the mitral valve in both inflow-outflow and commissural views. Two

mattress sutures reinforced with 4-0 polypropylene pledgets are placed at the optimal puncture site and passed through haemostatics loops.

The procedure is performed under fluoroscopic and TEE visualization. The typical fluoroscopic view is 10° cranial and 0° to 10° left anterior oblique on fluoroscopy (90° to 100° on the patient). The simultaneous TEE views are biplane commissural and inflow-outflow views and a three-dimensional "en face" view of the mitral valve. The TEE and fluoroscopic views are oriented with the aorta at 9 o'clock to provide synchronous cranial-caudal movement of the device between the TEE and fluoroscopic views.

Before entering the heart, an intravenous bolus of heparin (50 U/kg) is administered. The cardiac puncture is performed with an 18 g catheter over a needle. A 0.035-inch, 50 cm J-tipped guidewire is passed through the catheter into the left ventricle, and the catheter is removed. A 14F introducer sheath is advanced over the guidewire into the left ventricle. The J-tipped guidewire and the dilator are replaced with a specialized mitral crossing guide with a soft tip and a Nitinol structure. The mitral crossing guide is passed retrograde through the mitral valve under TEE guidance, and the introducer sheath is passed into the left atrium. The V-Clamp delivery system is passed into the left atrium via the introducer sheath. The upper portion of the V-Clamp device is manipulated to obtain optimal medial-lateral, A-P, and rotational orientation through TEE guidance and fluoroscopic visualizations within the left atrium before being positioned on the mitral valve. The lower portion of the device is brought through the mitral valve while maintaining the appropriate orientation. The lower portion is positioned under the mitral valve. The valve leaflets are captured by bringing the upper portion toward the valve. Once initial leaflet capture is confirmed based on TEE and fluoroscopy, the device is locked. The uncaptured portion of the anterior and posterior valve leaflets is measured and subtracted from the pre-closure lengths to ensure adequate leaflet capture (> 4 mm for each leaflet). The device is then released. In some cases, the placement of a second V-clamp can be considered to reduce residual valve regurgitation by following the same basic procedure and positioning the second V-clamp as close as possible to the first.

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Canine cardiac tumors - diagnosis and therapeutic options

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Introduction

Cardiac tumors in dogs and cats were considered rare diseases for many years. Recently, more sophisticated diagnostic tools, the spread of certain breeds predisposed to specific neoplasms, and the availability of new surgical and medical treatments have stimulated greater attention toward these pathologies, which in the past were mostly diagnosed incidentally or during necroscopic examination. Furthermore, the increase in the average age of the canine population, due to a longer life expectancy, may have contributed to the increased incidence of these tumors. In fact, in the daily practice of veterinary clinics, the diagnosis of cardiac tumors is becoming increasingly common, but in a consistent percentage of cases, the diagnosis is still an incidental finding on chest radiography or an echocardiographic study performed for other indications. Cardiac tumors can be classified according to their origin: primary or secondary, and their histological characteristics. Nowadays, diagnostic imaging and particularly echocardiography allow for an accurate in vivo description of most of the macroscopic aspects of this tumor, such as echostructure, size, shape, location, mobility, and site of insertion.

Primary and metastatic cardiac tumors are relatively rare in our patients, presenting an incidence equal to 0.19% in dogs and less than 0.03% in cats. The relative frequency of the various tumors depends on the population studied. The most frequently reported neoplasm in dogs is hemangiosarcoma (69% of all cardiac neoplasms), followed by tumors of the heart base (chemodectoma 8%, lymphoma 4%, and ectopic thyroid carcinoma 1%). In cats, the most often diagnosed neoplasm is lymphosarcoma, while carcinomas, hemangiosarcomas, chemodectomas, and fibrosarcomas are found more rarely.

All the studies present in the literature highlight a specific predisposition for hemangiosarcoma in the German Shepherd and the Golden Retriever; in our experience, we have found a higher incidence in the Labrador than in the Golden Retriever. Brachycephalic dogs—Boxers, English Bulldogs, and French Bulldogs—are predisposed to tumors of the heart base. The physiological function of chemoreceptor cells has suggested a hypothesis according to which the onset of chemodectoma in brachycephalics is secondary to chronic overstimulation of the chemoreceptors caused by the common respiratory difficulties in these subjects. This hypothesis is an alternative to a perhaps more probable genetic predisposition, but both have not yet been demonstrated by specific studies.

PHENOTYPES OF CARDIAC TUMORS

Hemangiosarcoma

Hemangiosarcomas are tumors that originate from the vascular endothelium and are localized at the level of the right auricle and the right atrium. It seems that their incidence is higher for some canine breeds (e.g., German Shepherd, Golden Retriever), while cats are rarely affected.

They are tumors with a tendency to bleed and are frequently the cause of pericardial effusion. In the case of slow bleeding and chronic tamponade, dogs present the classic signs of right-sided heart failure, while in the case of acute tamponade, symptoms of cardiogenic shock are present. Often at the time of diagnosis, the tumor has already given origin to metastases (liver, lungs, spleen, kidneys).

The echocardiographic appearance of this type of tumor is that of a non-homogeneous formation, with regular margins, which develops at the level of the wall of the right auricle or the right atrium and extends into the pericardial sac. Sometimes it can involve the right ventricle, the pericardial sac, or the left ventricle. The identification of the lesion can be difficult, especially in the case of small formations, and is easier in the case of pericardial effusion.

Hemangiosarcoma presents a poor short-term prognosis (<2 months from diagnosis). Palliative pericardiectomy and surgical removal of the tumour associated with chemotherapy represent therapeutic attempts with poor results in terms of survival (<2 months and <6 months respectively). Currently, the association of specific immunotherapies associated with surgery and standard chemotherapy have been shown to be able to offer improved survival even up to one year after surgery.

The morphological characteristics and localization of primary hemangiosarcoma in most cases allow for a reliable presumptive diagnosis. It usually consists of a single mass that originates from the myocardial wall of the right atrium and/or the right auricle and protrudes inside the pericardial or atrial cavity and sometimes involves the atrioventricular junction.

Heart Base Tumors

Heart base tumors are usually classified as chemoreceptor neoplasms (chemodectomas, non-chromaffin paragangliomas) and ectopic thyroid carcinomas. They are found with relative frequency in dogs and only rarely in cats. They often represent incidental findings in asymptomatic animals and can lead to the onset of symptoms if they cause the formation of pericardial effusion, compression of vascular structures, or invasion of adjacent structures.

Cardiac neoplasms of the heart base are very common. Generally, these are chemodectomas, in differential diagnosis with other tumors originating from the aortic body, such as paragangliomas. In the same location, it is possible to find lymphomas and ectopic thyroid tumors.

Echographically, heart base tumours are usually rounded masses, sometimes multilobed, homogeneous in appearance and with well-defined margins, located between the aorta and the pulmonary artery. Large lesions present poorly defined margins and tend to invade adjacent structures. More rarely, they may appear as small nodular areas distributed along the aortic wall, and in these cases, their visualization can be difficult.

Chemodectoma

Tumours originating from chemoreceptors, also known as aortic body neoplasms, develop around the aortic root (aortic bodies) and the carotid bifurcation (carotid bodies). Brachycephalic breeds show a greater tendency to develop this type of neoplasia, likely due to the conformation of the upper airways which favors a state of hypoxia. Males appear to be more affected than females. Chemodectomas, in most cases, are slow-growing, rarely metastasize, and rarely invade the lumen of the cardiac chambers or great vessels. Surgical tumour removal is possible only in very early cases. In the rare cases where tumour compression on the base of the heart causes recurrent cardiac tamponade, subphrenic percardiectomy is curative for this complication. In some cases, very large chemodectoma can compress the vessels at the base of the heart, with a more evident effect in vessels with lower pressure, such as the vena cava or pulmonary arteries (most commonly the right). In these cases, endovascular stents can maintain circulation.

Toceranib is currently used in other canine cancers, such as insulinomas, pheochromocytomas, thyroid carcinomas, and nasal carcinomas. It has antiangiogenic effects on the signalling pathways of several growth factors. This achieves stabilization or partial remission of tumour growth. Currently, Toceranib is indicated in the treatment of HBTs and despite its limited effectiveness in reducing tumour size, it is useful to alleviate the clinical signs and slow down tumour growth and, therefore, to improve survival rates. Treatment is based on oral administration every other day at the label dose of 3.25 mg/kg.

Chemodectomas develop around the wall of the aortic root and usually surround the main pulmonary artery. These tumors are non-functional and have very limited metastatic potential. Paragangliomas, like chemodectomas, originate from the aortic bodies, but unlike the latter, they are functional and can secrete catecholamines.

In cats, chemodectomas are particularly invasive, and affected animals usually have a poor prognosis, However, chemodectoma are very rare in cats and are often confused with cardiac basal lymphomas, which are much more common in this species and have similar characteristics.

Ectopic Thyroid Tumors

Ectopic thyroid carcinomas represent 5-10% of heart base tumors. They tend to develop at the base of the heart, then expanding into the cranial mediastinum, near the pericardium. They can sometimes present as intracardiac masses. These are tumors that tend to metastasize and are usually non-functional. The treatment of choice is surgical, but non-operable cases can be treated with radiotherapy. The prognosis is influenced by local invasiveness and the possibility of surgical resection. The average survival reported in the literature varies between 9 and 17 months.

Ectopic parathyroid tumours and ectopic thyroid tumours can appear as huge masses located at the base of the heart and, in this condition, represent a differential diagnosis for chemodectoma; in other cases, ectopic thyroid tumours originate from the interventricular septum and invade the right ventricle. Ectopic thyroid tissue can be found throughout the area

embryologically derived from the thyroglossal duct region of the canine embryo, therefore from the base of the tongue to the diaphragm. The migratory tissue that leads to the formation of the thyroid gland during embryonic development is in contact with the tissues that lead to the formation of the aortic root and the ventricular myocardium; the ectopic cells remaining in the mediastinum and those associated with these structures can lead to ectopic thyroid carcinomas and adenocarcinomas.

Lymphoma

Primary cardiac lymphoma can affect the myocardium and pericardium; in this case, according to the WHO classification of canine lymphoma, it is considered stage V (extranodal and in an organ other than the spleen or liver). In some other cases, it is formed by a homogeneous hyperechoic mass located at the base of the heart. It usually presents with an emergence in a more caudal position than a chemodectoma.

Mesothelioma

Mesotheliomas can affect the serous membranes; in addition to invading the pleura and peritoneum, they can also primarily involve the pericardium. Neoplastic invasion of the serous membranes leads to their thickening, the proliferation of characteristic nodules, and severe cavitary effusion. Histopathological diagnosis can be quite challenging, as these neoplasms can resemble other epithelial or mesenchymal neoplasms. However, in most cases, immunohistochemical investigation is decisive.

Metastatic Cardiac Tumours

Cardiac metastases reach the heart through anatomical continuity, via pulmonary or systemic venous return, through lymphatic circulation, or via the coronary arteries, causing intramural metastases. In dogs and cats, cardiac metastases secondary to hemangiosarcoma, lymphoma, and melanoma have been described mainly at the level of the left ventricular free wall, while metastatic carcinomas can cause pericarditis with or without pericardial effusion. Intramural metastases appear as multiple disseminated nodules. The echocardiographic tissue appears more heterogeneous compared to the surrounding normal myocardium; conversely, in some rare cases, the echostructure is normal; therefore, the suspicion of neoplastic infiltration can be raised by the absence of systolic contraction in the segment.

Intracardiac Neoplasms

Lipoma

Although lipomas are tumors frequently found in subcutaneous sites, they can develop in visceral sites and can involve various organs, including the heart. They originate from the epicardium, myocardium, or endocardium and develop in intracardiac or pericardial locations. They are slow-growing tumors and often do not give rise to symptoms for long periods of time. When the neoplasm infiltrates the cardiac walls or causes obstruction to cardiac inflow or outflow, clinical signs appear. Surgical excision of the neoplastic mass represents the therapeutic treatment of choice.

Rhabdomyosarcoma

Rhabdomyosarcomas are rare tumours that often have an infiltrative character and, more rarely, a polypoid appearance protruding into the cardiac chambers. In the literature, the right atrium and right ventricle are the cardiac chambers most often involved by this type of neoplasia, and clinical symptoms are caused by the obstruction of blood inflow and outflow.

Myxoma and Myxosarcoma

The myxoma is a benign tumour of the endocardium that originates from multipotent mesenchymal cells located in the subendocardial space. It represents one of the most common primary cardiac tumours in humans but is extremely rare in veterinary medicine. The localization of myxomas varies. The right heart chambers, the right outflow tract, the tricuspid valve, the left atrium, and the mitral valve can be involved. Echographically, myxomas appear as rounded, polypoid, and pedunculated formations, homogeneous and with smooth margins. The clinical signs of affected animals depend on the location, size, and mobility of the mass. Cases of tumour thromboembolism are not rare, reaching approximately 30-45% of cases in human medicine. The growth rate of myxomas has not been determined, and cases in the literature are discordant. The treatment of choice is surgical removal of the tumour.

Myxosarcomas are rare malignant tumours of mesenchymal origin. They often present in an infiltrative form and can involve the pulmonary artery, the right atrium and ventricle, the right ventricular outflow tract, the left ventricle, and the pericardial sac. The echocardiographic appearance of the mixosarcoma is that of a non-homogeneous and multilobulated mass. The treatment of choice is surgical removal of the tumour, although there is a risk of local recurrence and metastasis to other sites.

Diagnostic Imaging

Radiology

Radiographic study in cases of pericardial effusion identifies typical alterations, such as an increase in the transverse diameter of the cardiac silhouette, which takes on a rounded appearance and masks the profiles that allow for the recognition of the posterior atrioventricular junction and the great vessels. In this condition, the hemodynamic effects of tamponade can be recognized by the reduced size of the pulmonary vessels and the dilation of the caudal vena cava. However, the radiograph does not differ from those of pericardial tamponade caused by other conditions, such as idiopathic pericarditis.

After pericardiocentesis, in some cases, it is possible to recognize morphological alterations attributable to neoplastic masses. As previously mentioned, the first sign indicating the possible presence of a cardiac tumour may be observed on a chest radiograph performed for other reasons. This occurs especially in e early stage chemodectoma; the most common and evident sign in this condition is the dorsal displacement of the trachea cranially at its bifurcation, resulting from the dorsal expansion of the mass originating from the aortic body.

Radiographic study should always be included in the routine for cases with cardiac masses to identify thoracic metastases; for this purpose, CT offers even greater precision. Today, the latest generation of CT and high-field MRI offer accurate visualization of cardiac structures and provide further diagnostic support, especially in studying the relationship between masses and great vessels—information that can be useful for planning surgical interventions or palliative interventional procedures. However, while CT should be recommended for the study of pulmonary vessels and the search for metastases, for the study of cardiac and pericardial masses, neither CT nor cardiac MRI offers superior information to that obtainable via echocardiography.

The prognosis of cardiac tumours is primarily linked to the histological type and secondarily to operability. Due to the location of the tumours, surgical intervention is rarely feasible. Nowadays, greater surgical experience and the availability of new technologies have increased feasibility and success rates. The first choice diagnostic technique is echocardiography, including supplemental technologies such as Real-Time 3D and the use of ultrasound contrast agents; secondarily, CT allows for additional views of the mass and its relationships with surrounding tissue; as it is however essential for the staging of the patient by identifying the presence of any metastases or excluding their presence. The experience of the operator, the temporal resolution of the machine, and the choice of the correct probes strongly influence diagnostic accuracy of echocardiography.

Echocardiographic Examination

Nowadays, the diagnosis of cardiac tumours is becoming much more frequent than in the past. Pericardial effusion creates a better acoustic window that allows for the visualization of cardiac structures; in this condition, the specificity and sensitivity of echocardiography for observing and characterizing masses are very high, significantly superior to any other method. In many cases, the localization, extension, and echocardiographic characteristics of these lesions can help suggest the histological type.

The echocardiographic study defines the extension of the mass and the infiltration of the myocardial tissue, helps distinguish whether the lesion is monofocal or multifocal, and documents the hemodynamic impact of tumours invading the cardiac cavities. Most cardiac tumours can be easily visualized using transthoracic echocardiography (TTE), while transesophageal echocardiography (TEE) should be used if the acoustic window is inadequate, to identify small masses, or to study the relationship between the tumour and other structures at the base of the heart. Recent technical evolutions in both TTE and TEE, along with the introduction of real-time three-dimensional echocardiography (RT3DE), offer a more accurate view of cardiac tumours, allowing for the definition of the mass's boundaries for a better understanding of local invasiveness and operability in cases where the mass can be surgically removed. Furthermore, contrast imaging performed using specific ultrasound media and dedicated software can help differentiate a cardiac mass from the myocardium or adjacent thrombi: malignant and vascular tumours are hyper-enhanced, while stromal tumours and thrombi are hypo-enhanced.

The echocardiographic examination of these patients is essentially observational; however, it should still follow a complete, systematic, and sequential approach, obtaining all standard projections and exploring less common ones to examine the entire cardiac anatomy and identify any anomalies.

When the examination begins with a sequential approach from the right parasternal view, the right atrium and auricle are the first anatomical regions studied, as they are the most frequent locations for neoplasms. Hemangiosarcomas are most located here, appearing as heterogeneous masses with small hypoechoic cavities. The presence of pericardial effusion can be useful for identifying even small, early lesions located on the apex of the right auricle. Hemangiosarcoma can develop invasively toward the right atrial cavity, partially invading it. Rarely are the tricuspid leaflets and the atrioventricular junction involved; more frequently, the hemangiosarcoma grows into the pericardial space, creating a mass that extends ventrally and cranially to the right ventricular outflow tract. This mass can also extend dorsally, touching the aortic root and the main pulmonary artery. The external part of the hemangiosarcoma facing the pericardial cavity frequently features a soft portion, composed mainly of fibrin overlying the bleeding site. Some hemangiosarcoma expand toward the right atrial chamber as thin, pedunculated structures that move with the blood flow.

A complete view of the right auricle can only be obtained in an optimized off-axis view; from the right parasternal long-axis view, the probe must be moved laterally and slightly counter-clockwise to obtain a dedicated off-axis view where the right auricle, right atrium, part of the tricuspid valve, and both venae cavae are all visualized on the same plane. Subsequently, it is necessary to tilt the probe slightly dorsally and cranially to visualize the typical non-homogeneous structure of hemangiosarcoma and its degree of invasion into the atrial myocardium. Three-dimensional echocardiography is the gold standard technique for defining the extent of many tumours, particularly in cases where the invasion of auricular hemangiosarcoma into surrounding anatomical areas, such as the atrioventricular junction, is unclear. The greatest advantage of 3D eco consists in the possibility of acquiring the full volume of the area to be explored and subsequently performing multi-plane sections in real time. These tomographic sections performed during the heart's movement constitute a significant advantage of 3D eco over CT.

To study the extent of hemangiosarcoma and the contiguity of these masses with the atrioventricular junction the infiltration of which defines absolute inoperability—this anatomical zone can be studied using apical 4- and 5-chamber views in addition to right parasternal views. By visualizing the aorta in the 5-chamber view, the latter can be used as an anatomical reference; by directing the ultrasonic beam in front of it, another view of the right auricle can be obtained to move toward an off-axis view, which is useful for defining the precise localization of the mass. This topographic detail, combined with all other observations obtained in the complete echocardiographic examination, can be crucial for a surgical plan.

The study of the neoplastic mass's limits and the relationships of contiguity or infiltration of the myocardial tissue, as previously mentioned, should be carried out with maximum accuracy to define the tumor's operability. This applies not only to the most common condition, hemangiosarcoma, but also to other intracavitary neoplasms; for example, in advanced-stage adrenal tumors invading the caudal vena cava, the mass may progress inside the right atrium

and right ventricle without any myocardial infiltration, but this needs to be confirmed with accurate observation of the mass's entire extent. An accurate study of the acoustic interface between intracavitary masses and the endocardium is also part of the differential diagnosis process between endocavitary neoplasms and thrombi. Thrombi are usually located in slow-flow areas such as the right atrium and right auricle, and even when the thrombus is strongly attached to the endocardium, it is possible to identify the thin anechoic boundary between the thrombus and the endocardium. Furthermore, fresh thrombi are homogeneously hyperechoic, while in old thrombi, the centre becomes less echogenic, and the border becomes definitively hyperechoic. The optimization of this diagnostic process depends on the adjustment of echocardiographic instruments and the choice of probes with adequate spatial resolution to define acoustic interfaces.

Valvular neoplasms have a rounded and homogeneously hyperechoic appearance, though this can vary with the histological type; the mass causes functional stenosis by occluding the valvular orifice. These functional stenoses cause severe asthenia and syncope. Frequently, these valvular masses seem to affect only the valve, but upon closer examination, in some cases, it is possible to identify the infiltration of the contiguous myocardium with the same echostructure. In these cases, it is difficult, if not impossible, to define whether the neoplasm is primary myocardial or endocardial with extension to adjacent tissue.

In our experience, myocardial neoplasms of the pulmonary and tricuspid valves were hemangiosarcoma, while in the left atrium, myxosarcoma were more frequently identified. The differential diagnosis between valvular neoplasms and endocarditis is relatively simple. Valvular masses are large compared to vegetative neoformations, which may present with a few nodules and very irregular echostructure and borders; furthermore, endocarditis causes erosion of the valvular structure and therefore almost always massive valvular insufficiency. However, the differential diagnosis should not be based on echocardiographic observation alone but should be contextualized within the patient's clinical presentation. Thrombi in the hearts of dogs are observed mainly in patients suffering from coagulation disorders, while patients with acute endocarditis present all the clinical characteristics of systemic infection. Other neoplasms protruding into the cavities are the rarer rhabdomyosarcomas, which originate from myocardial tissue, altering its architecture and thus its echostructure, rapidly invading the ventricular cavity and sometimes infiltrating surrounding tissues.

The echocardiographic study of heart base tumours should be aimed at identifying the echostructure of the mass—which in chemodectoma is slightly hyperechoic and partially homogeneous—and recognizing the site of origin, which is still possible at least in early cases when masses are small. Subsequently, the extension of the mass and the compression of chambers and vessels must be described. In chemodectoma, its origin is recognizable between the aorta and the pulmonary artery at the base of both; then, the mass progressively surrounds the aortic root and the pulmonary artery. Most frequently, the right pulmonary artery, which is closest to the aortic root, is compressed. For the recognition of vascular compressions, as in obstructive forms, the severity of these effects can be studied using Doppler. Color Doppler can identify the extent of flow interference, while its severity can be quantified with continuous Doppler based on the pressure gradient determined.

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PDA in dogs and cat: assessment, management and interventional options

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Patent Ductus Arteriosus (PDA) is one of the most common congenital heart diseases in dogs. It is almost always found in an isolated form, though it can sometimes occur in association with pulmonary stenosis or subaortic stenosis; the latter association is frequently found in Newfoundlands.

During intrauterine life, the Ductus Arteriosus diverts blood flow directed toward the lungs toward the aorta. At birth, with the first breaths, the lungs expand and pulmonary resistance decreases; the increase in oxygen concentration in the arterial blood is responsible for the vasoconstrictive effect on the ductus and is decisive for the start of the closure process, which is generally completed within the first days of life. Additional factors contributing to the closure of the ductus in the first days of life are: the degenerative process of the ductal tissue, the formation of thrombi within it, and the local vasoconstrictive effect of prostaglandins. If the patency of the ductus persists, pulmonary hyperperfusion results in left ventricular volume overload. In the perinatal period, the heart's adaptation mechanisms to volume overload determine an important remodeling , characterized by a widespread hyperplastic response This postnatal cell proliferation phenomenon allows animals affected by PDA an extraordinary adaptation not found in adult subjects lacking such a mechanism for adapting to volume overload. For this reason, in dogs with patent ductus arteriosus, we observe a unique relationship between clinical symptoms and the degree of ventricular dilation: the left ventricle of dogs with PDA can, in fact, dilate to sizes not otherwise observable in any other heart disease.

Symptoms vary depending on the amount of blood passing through the ductus and the volume overload of the pulmonary circulation and the left sectors of the heart,and are directly proportional to the magnitude of this blood volume crossing the patent ductus. The shunt amount is conditioned by the size of the communication at the pulmonary side of the ductus and by the pressure gradient between the aorta and pulmonary artery, which in turn is predominantly determined by pulmonary resistance.

Therefore, the clinical picture can be extremely variable, with a wide spectrum of clinical presentations ranging from nearly asymptomatic subjects with very small ducti who can remain asymptomatic for years, presenting only clinico-instrumental signs such as a murmur and very slight dilation of the left side of the heart (which in very rare cases can even be absent) to subjects who, since the first months of life, can present symptoms of pulmonary circulation congestion and left heart failure.

Upon palpation of the thorax, the apex beat is perceptible over a wider area, and a precordial thrill is sometimes detectable right under the left axilla. The arterial pulse is "pounding," characterized by a wide rise during systole and a rapid fall in diastole. The auscultatory finding is that of a typical continuous systolic-diastolic murmur (Gibson murmur), with maximum audibility cranially and dorsally to the aortic area, in the region located beneath the axilla (it is

necessary to move the left leg slightly forward to be certain of identifying it). The murmur can have different intensities (4-6/6); in small ducti, it is usually well-localized, whereas in large ones, it can radiate cranially. In those cases in which the disease progresses with the dilation of the left ventricle, this also causes dilation of the annulus; therefore, in addition to the systo-diastolic murmur of ductal origin, a systolic murmur due to mitral regurgitation is evident at the mitral area.

In addition to various degrees of left atrioventricular insufficiency, the clinical and pathophysiological picture can take on very different characteristics. In fact, in cases of very large PDA, as with other congenital pathologies with a high left-to-right shunt volume, Eisenmenger syndrome with shunt reversal can develop. The remodeling of the pulmonary arterioles leading to pre-capillary pulmonary hypertension occurs mostly in the first months of life; this is followed by cardiac remodeling with a prevalence of right ventricular hypertrophy signs and little or no evidence of left volume overload. This described scenario differs from late post-capillary pulmonary hypertension observed in adult subjects with large ducti, which depends on severe volume overload. In this situation, the dominant symptoms and cardiac remodeling aspects are still predominantly those of left volume overload, and shunt reversal is rarely observed.

In young dogs with a reversed PDA due to Eisenmenger syndrome, one of the first symptoms reported by owners often confused with a neurological deficit—is the loss of control of the hind limbs after even mild exertion. This is due to local hypoxia: because the ductus connects the aorta to the pulmonary artery caudally to the epiaortic vessels that perfuse the front of the body, the anterior part receives oxygenated blood while the caudal aorta carries blood with low oxygen content. This is also the basis for the clinical sign of differential cyanosis, where cyanosis is observed only in the posterior mucous membranes (generally the genitals), while the anterior mucous membranes remain normal in color. These animals may present a very slight murmur or none. Sometimes, a splitting of the second heart sound or a systolic murmur over the tricuspid area due to valvular incontinence can be found.

The role of echocardiography in managing a patient with patent ductus arteriosus is not only to establish a precise etiological diagnosis (the actual patency of the ductus), but also to identify other associated cardiovascular pathologies, establish different levels of severity of alterations resulting from volume and pressure overload, and perform a comprehensive evaluation to guide the most correct choices for ductal closure (surgical vs. interventional) and the actual need for medical therapies.

Parameters to be considered in the echocardiographic examination of a subject with PDA are: the dimensions of the left ventricle (systolic and diastolic ventricular diameters and volumes) and the measurement of the maximum diameter of the communication point between the ductus and the pulmonary artery. Flow through the ductus is identified with Color Doppler, while Continuous Doppler must be used to measure the maximum flow velocity, which expresses the transductal pressure gradient. This velocity, measured with correct alignment, must be at least 4.5 m/sec. This reflects a pressure difference between the descending aorta and the pulmonary artery of approximately 80 mmHg; if pulmonary pressures increase or systemic pressure decreases due to severe left ventricular dysfunction, the gradient tends to decrease.

With Doppler, it is possible to approximate the ratio between pulmonary flow (Qp) and systemic flow (Qs); the difference between these represents the shunt volume. However, in the

case of PDA, the shunt occurs distally to the aortic and pulmonary valves. Therefore, we should consider the flow measured at the aortic annulus as the pulmonary flow (Qp): the flow volume measured at this level corresponds to the blood entering the lungs distally to the ductus plus the systemic flow. The systemic flow (Qs) must be measured at the [pulmonary] annulus, where the blood volume corresponds to the flow carried into circulation by the post-ductal descending aorta, i.e., the blood crossing the pulmonary orifice.

The most suitable scan to measure the pulmonary side of the ductus is obtained from the left cranial parasternal short-axis view, orienting the ultrasound beam slightly dorsally relative to the pulmonary bifurcation. Before measuring, it is necessary to ensure that the minimum diameter at the outlet point has been identified; it is very easy to measure the ampullary diameter instead, leading to an overestimation of the actual orifice size. The ductal ampulla may present an aneurysmal dilation, which must be considered for therapeutic procedures but has no diagnostic or prognostic value in staging the severity of the disease.

In subjects with small ducti—with a pulmonary side diameter of less than 3 mm in small dogs and less than 5 mm in large dogs a various degree of remodeling of the left ventricle is nonetheless evident, depending on the subject's age. We might identify an increase in left ventricular volumes compared to reference values; it is indeed very rare to find dogs affected by PDA that present normal ventricular dimensions.

Usually, there are no signs of systolic and/or diastolic dysfunction, nor signs of valvular insufficiency. The Qp/Qs value is generally < 1.4.

Subjects presenting moderate hemodynamic, pathophysiological, and cardiostructural adaptations represent the majority of diagnosed PDA cases. Puppies with medium-sized ducti (approximately 4 mm for small breeds, 5-6 mm for large breeds) or adult dogs with small ducti not operated on in their youth fall into this severity class. The dimensions of the left ventricle are frankly increased, and the ventricular morphology also begins to change; the ventricle takes on a spherical shape with a clear increase in transverse diameters, and volumetric indices measured in M-mode reach frankly elevated values: EDVI > 150 ml/m^2 and ESVI > 60 ml/m^2. The dilation also affects the mitral annulus, causing central mitral regurgitation that leads to further volume overload with an increase in end-diastolic pressure and left atrial dilation. The dilation of the pulmonary artery is even more evident. The Qp/Qs ratio assumes values between 1.4 and 1.8.

In what we could define as severe echocardiographic patterns of PDA, a wide spectrum of morphological and functional modifications of the cardiovascular structures is observed. The dimensions of the ductal pulmonary side are almost always very significant: ≥ 5 mm in small dogs and ≥ 6/7 mm in large dogs. The signs of left atrioventricular dilation are massive, with indexed volume values measured in M-mode rarely found in other congenital or acquired heart diseases: EDVI > 200, ESVI > 100, significant reduction in the volume/mass ratio and shortening fraction, and a decrease in parietal and septal thickening indices. Frequently, the Qp/Qs ratio is greater than two and, in some cases, the transmitral flow is restrictive.

In subjects with particularly wide ducti, alongside the signs of left volume overload, it is possible to identify echocardiographic signs of pulmonary hypertension, such as flattening of the interventricular septum with systolic paradoxical movement and high-velocity tricuspid regurgitation, which expresses the increased pressure in the right ventricle. In these conditions, the shunt remains directed from left to right despite the decrease in transductal velocity due to the increase in pulmonary pressure and the decrease in left ventricular systolic

function. In some subjects, the massive left ventricular volume overload causes the rightward displacement of the interventricular septum which, by reducing the size of the right ventricular cavity, slows right venous return, causing ascites even in the absence of tricuspid regurgitation and pulmonary hypertension.
In PDA with reversed shunt due to Eisenmenger syndrome, the echocardiographic picture is completely different: right ventricular hypertrophy, pulmonary artery dilation, and high-velocity tricuspid and pulmonary regurgitation prevail, while the ductal flow, although reversed, is low-velocity and difficult to identify.
As in most congenital heart diseases, the ideal therapy for PDA is the surgical or interventional correction of the defect. These therapies, performed in the appropriate manner and timing, can lead to the complete resolution of the problem with extremely low surgical risk percentages and extremely high therapeutic success rates, allowing operated subjects normal quality and life expectancy.

Treatment
Medical therapy in these cases is palliative, used to stabilize symptomatic patients before intervention or postoperatively for severe dysfunction. Depending on the pathology, it may include diuretics, vasodilators, positive inotropes, and antiarrhythmics. The curative treatment for PDA is duct closure, indicated for all animals with a hemodynamically significant left-to-right shunt.

- Surgical Ligation: Historically the only option, it remains a high-success technique used in centres lacking percutaneous equipment, or for very small dogs, cats, and patients with Type 3 PDA (where a lack of pulmonary narrowing prevents stable device positioning).
- Interventional Closure: The Amplatz canine duct occluder (ACDO) is currently the first-choice method worldwide, characterized as quick, effective, and safe when performed by experts.

Limitations and Alternatives: Percutaneous procedures are limited by patient size; in dogs under 2 kg, the femoral artery is often too small for the vascular introducer. In very small dogs and cats where surgery is not indicated, "off-label" alternatives include small vascular plugs or controlled-release coils delivered via a 4 Fr catheter.
Supportive medical therapy must be maintained in those cases operated on late, in which a state of congestive heart failure persists despite the surgical correction of the defect.
Over the years, various types of coils have been employed which, after introduction into the ductal lumen, lead to its progressive occlusion. In the case of large ducti, these devices present certain limits regarding safety and efficacy. Devices originally designed for other purposes but adapted for this type of ductal occlusion in dogs have also been used, with results that are not always satisfactory.
The closure of the ductus in cases of pulmonary hypertension is still indicated when it is of the post-capillary type induced by volume overload. It is contraindicated in the case of Eisenmenger syndrome, although in some cases identified when the pathophysiological mechanism is still developing and the shunt is still bidirectional, closing the ductus can bring benefits by interrupting the ongoing pathophysiological mechanism. In these cases, closure must be performed in a specialized centre where an occlusion test is performed, simultaneously measuring pulmonary and systemic arterial pressure. If temporary occlusion

of the duct causes systemic pressure to increase and pulmonary pressure to remain unchanged or decrease, permanent occlusion can be performed.

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Right-sided obstructive congenital heart disease: interventional treatment and follow-up

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Introduction

Congenital heart diseases (CHD) can manifest from the early to the late stages of cardiac development. The development of the heart is regulated by intricate networks of transcription factors. The decisions on cell fate at the origin of all cardiac cell lines are influenced by both genetic (DNA sequence) and epigenetic processes (regulating genetic expression without altering the sequence, for example DNA methylation, histone modifications, higher-order chromatin structure). However, the exact genetic, epigenetic, or environmental basis of CHDs remains poorly understood, although the exact mechanism is likely multifactorial, both in humans and in animals. This suggests that for the majority of CHDs a more complex dysregulation of multiple genes and transcriptional pathways occurs, resulting from changes in the long-range regulation of transcription.
The first references in the literature on genetic evidence as a cause of congenital heart diseases in dogs were published by Patterson at the end of the years '68. Subsequently, genealogical studies in specific breeds on various CHDs revealed a polygenic transmission such as PDA, PS, SAS, VSD, TF, and persistent aortic arch. Each of these defects is inherited in a complex way consistent with a polygenic basis.
Since populations of purebred dogs and cats are much less genetically mixed and were established more recently compared to human ones, the genetic model of CHDs in dogs or cats may be different from that of humans. However, knowledge of human genetic aetiologies can be a good starting point for fundamental studies on CHDs in small animals using new genetic sequencing techniques.

Pulmonic stenosis (PS) includes all those structural malformations that cause obstruction of blood flow from the right ventricular outflow tract (RVOT) to the pulmonary artery (PA). Pulmonic valve stenosis is one of the most common congenital heart diseases in dogs, representing from 20% to 32% of all CHDs, and brachycephalic breeds are commonly affected. In addition to alterations of the valvular apparatus, forms of subvalvular stenosis can be observed, located below the valvular plane at the infundibular level, while supravalvular malformations are rarer and are caused by segmental or diffuse narrowing of the main pulmonary artery (MPA).
In some breeds, pulmonic is associated to an anomaly of the coronary arteries which emerge from a single right coronary ostium. Buchanan and Patterson have described an anomaly of the coronary arteries called “type R2A,” particularly widespread in the English Bulldog. This anomaly involves a single right coronary artery from which the left circumflex coronary artery and the left anterior descending coronary artery originate. This aberrant coronary artery surrounds and narrows the ventricular outflow tract near the level of the pulmonary valve, contributing therefore to the pulmonic stenosis. The diagnosis of the presence of this condition is essential for the purposes of interventional therapeutic treatment (balloon catheter valvuloplasty), as in this specific case it can cause death due to myocardial ischemia and coronary avulsion.

Echocardiographic Examination

The two-dimensional echocardiographic study of the apparatus allows for an initial distinction between the two main phenotypes of pulmonic stenosis.

Type A Pulmonic Stenosis:
It is characterized by the lack of division of the valvular leaflets, which thus remain fused together. The valvular apparatus behaves like a diaphragm that in systole assumes a dome-like appearance, moving upwards ("systolic doming" effect). The annulus is of normal dimensions, meaning the ratio between the aortic and pulmonic annular diameters is less than 1.2. Often the pulmonary trunk and the main branches appear dilated.

Type B Pulmonic Stenosis:
The characteristic of type B is the hypoplasia of the pulmonary annulus with an aortic annulus/pulmonic annulus ratio > 1.5. The valvular leaflets appear thickened (rudimentary leaflets) and sometimes fused together. Annular hypoplasia is often accompanied by hypoplasia of the main pulmonary artery.

It is important to remember that nowadays the subdivision of pulmonic stenoses into only two groups is quite simplistic. The characteristics described above can in fact present themselves in various combinations, and it is therefore important to always try to perform as complete an echocardiographic examination as possible, identifying all the various characteristics of the subject under examination to best define its CHD and thus identify the best available therapeutic options.

The minimal data set that should be always collect includes:

- Pulmonary and Aortic annulus measurement
- Leaflets morphology
- Coronary anatomy
- Main pulmonary artery and outflow tract evaluation
- Doppler Echocardiography
- Assessment RV function
- Evaluate associated pathologies

In subvalvular or infundibular stenosis, the obstruction is determined by the presence of a fibro-muscular ring located below the valvular ostium. It is also necessary to distinguish between isolated forms, resulting from the narrowing of the valvular ostium, from hypertrophic forms of the right ventricular outflow tract resulting from the valvular stenosis. In mid-ventricular obstructions, the excess fibro-muscular tissue originates from the marginal trabecula and divides the right ventricle into a high-pressure chamber corresponding to the inflow area between the tricuspid and the obstruction, and a low-pressure chamber approximately corresponding to the outflow tract and included between the obstruction and the pulmonary valve.

The severity criterion for pulmonic stenosis based on the peak pulmonary gradient classifies the stenoses into three classes:

- **Mild pulmonic stenosis:** peak gradient up to 50mmHg

- **Moderate pulmonic stenosis:** peak gradient between 50 and 80mmHg
- **Severe pulmonic stenosis:** peak gradient over 80mmHg

One-dimensional and two-dimensional scans are used in the evaluation of the degree of ventricular hypertrophy and right ventricular functionality. In the presence of pulmonic stenosis, the wall of the inflow tract (optimal view in right and left parasternal short-axis scans) and that of the outflow tract appear thickened and with a more or less evident degree of hypokinesia. In advanced cases, the persistence of the increased wall stress produced by the systolic overload results in systolic dysfunction with reduction of the kinesis and thickening of the right ventricular wall and in the progressive dilation of the right ventricular cavity.
The morphological evaluation of the pulmonary valve apparatus and the main pulmonary trunk begin from the right parasternal short-axis scan, in which the pulmonary annulus, the valve leaflets, the right outflow tract, and the main pulmonary trunk are highlighted. These structures can also be studied in the left cranial parasternal short-axis scan and in its mirror image, which is obtained by rotating the transducer 180° counterclockwise and which in some cases offers better images of the valve and the outflow tract. The diameters of the pulmonary annulus and the aortic annulus must be measured at the inner endocardial borders immediately below the insertion of the valve leaflets at the end of the systolic event both on their long axis. The study of valve morphology includes the evaluation of the degree of thickening, fusion, and mobility of the valve leaflets in addition to the identification of any (very rare) fixed subvalvular obstructions. The left parasternal short-axis scan allows for a better visualization of the main pulmonary trunk and its bifurcation into the right and left pulmonary branches and of the right outflow tract for the evaluation of possible dynamic stenoses.

The spectral Doppler analysis performed with Continuous Wave Doppler serves to measure the mean and peak gradients of the pulmonary flow. In addition to this numerical data, from the morphological analysis of the curve, it is possible to identify cases in which a dynamic stenosis due to infundibular hypertrophy is superimposed on the fixed stenosis. The flow due to the dynamic component presents an asymmetrical morphology, so-called "knife-blade," with slowed acceleration and rapid deceleration that superimposes on the symmetrical morphology of the fixed component, whose peak velocity will therefore be earlier compared to that of the dynamic component.
Always with continuous Doppler, any tricuspid regurgitation must also be investigated, the peak velocity of which must obviously confirm the measurement of the right ventricular pressure estimated with the pulmonary peak gradient, while the amount of regurgitant flow estimated in a semi-quantitative way with color Doppler will be proportional to the dilation of the tricuspid annulus and therefore to the worsening of the pathology.

Association with Other Congenital Heart Diseases
In most cases, Pulmonic Stenosis (PS) occurs as an isolated malformation or as a component of complex diseases like tetralogy of Fallot. However, it can also be associated with simple defects such as subaortic stenosis (frequent in Boxers), tricuspid dysplasia, patent ductus arteriosus, and septal defects.
The association with tricuspid dysplasia often involves hypoplasia of the pulmonary annulus and the main pulmonary artery. When PS occurs with left-to-right shunts, therapeutic decisions depend on which malformation is hemodynamically dominant. It is advisable to treat both or at least the most significant one; if this is not possible, it may be better to perform no

procedure. For instance, in large, unclosable VSDs, PS may actually protect the pulmonary circulation.

Treatment options

In patients with a transpulmonary gradient lower than 60 mmHg no procedures are indicated
When applicable, PBV percutaneous balloon valvuloplasty is considered the first-choice treatment for PS. This procedure usually has a high success rate and is fast and safe when performed by experienced cardiologist. Restenosis occurs in less than 20% of treated cases; in these patients, a second BVP has proved to be safe and efficacious in most cases. Generally, a reduction of the peak pulmonary gradient by at least 50% can be considered satisfactory. The patients that may obtain greater gradient reduction are those with type A PS, with a normal annulus and fused and mildly thickened valve leaflets. Annular hypoplasia and thickening of the valve leaflets are not or only mildly improved by balloon dilation, so the pulmonary gradient of patients with the typical type B PS may not be greatly reduced.
In dogs with large pulmonary annulus (>18 mm) double balloon technique is recommended to achieve a better stability during the procedure e to reduce the drop in flow rate.
When thick and narrowly fused leaflets are present, consider combined PBV (cutting + High pressure ballon catheters).
When type B with extreme morphology (P. Annulus hypoplasia) is present, with PA/Ao ≤ 0,7 the simple PBV would be ineffective and it is important to advise pulmonary stenting. This can be performed with hybrid or percutaneous procedure, depending on the remodeling of the heart.
In case of severe RA dilation, severe tricuspid regurgitation, severe RV hypertrophy, the hybrid technique is to prefer.
In cases of severe annular hypoplasia, the transannular patch graft is another option.

In the double-chambered right ventricle (DCRV), a fixed obstruction located at the mid-ventricular level is identified, which divides the ventricle into a high-pressure chamber and a low-pressure chamber. This division occurs between the obstruction itself and the pulmonary valve plane. In most cases, the obstruction goes from the anterior interventricular septum and the anterior leaflet of the tricuspid valve to the anterior wall of the right ventricular outflow tract. The obstruction can be a continuous muscular band between the septum and the free wall or a muscular hypertrophy of both the septal and parietal portions of the outflow tract and is delimited by fibrous tissue of endocardial origin.
The hemodynamics of DCRV are influenced by the pressure overload in the right ventricle, and clinical progression leads to severe right heart failure like pulmonic stenosis. Surgery has a high success rate in humans and is generally recommended if the systolic pressure gradient across the lesion is greater than 40 mmHg. Surgical correction has also been reported in small animals with survival rates beyond 30 days post-intervention of 71% in dogs (14 cases) and 67% in cats (3 cases). Balloon catheter dilation of DCRV has had positive outcomes only in rare cases in humans, with mixed results in cats and rather discouraging results in dogs.

Tricuspid valve dysplasia includes various valvular and subvalvular anomalies and malformations of the chordae tendineae and papillary muscles. In dogs, the reduction in length or the absence of chordae tendineae is frequent, while the valve leaflets may be thickened, shorter or longer, sometimes fused together. The most common tricuspid dysplasia consists

of varying degrees of valvular insufficiency. Furthermore, various degrees of stenosis are reported, which are often associated with severe forms, while valvular atresia is quite rare in animals. In some dogs and in cats, tricuspid dysplasia shows anatomical aspects like Ebstein's anomaly, a congenital cardiac malformation characterized by the apical displacement of the functional annulus, redundancy and shortening of the leaflets and chordae tendineae, and dilation of the right atrioventricular junction. In Labrador Retrievers, tricuspid dysplasia is a hereditary disease.

Tricuspid dysplasia is characterized by the pattern of valvular insufficiency with right atrio-ventricular volumetric overload. The two-dimensional scans used for the study of the tricuspid valve apparatus are the right parasternal long-axis and the left apical four-chamber scan. From the right parasternal short-axis, it is difficult to obtain reliable information due to the systo-diastolic displacement of the valve plane. Under normal conditions, the septal insertion of the tricuspid is slightly lower than the mitral one (together they form the "crus cordis"). In the dysplastic valve, this distance can be more accentuated. The anatomical valvular alterations can involve, in addition to the valve leaflets (thickened, fenestrated, with reduced excursion), also the chordae tendineae (usually shorter than normal), an anomaly particularly evident on the septal leaflet.

Treatment

In Tricuspid Dysplasia (TD), medical therapy provides palliation. While patients with mild TD and no remodeling do not require treatment, those with atrioventricular dilation benefit from ACE inhibitors and diuretics to reduce volume overload and delay right-sided congestive heart failure. Spironolactone should be included to manage water retention caused by chronic hepatic congestion. If ascites develops, maximum doses of loop diuretics are used, though paracentesis is often necessary. Tachyarrhythmias are managed through ablation or antiarrhythmic drugs, while digoxin and diltiazem are used for heart rate control in chronic atrial fibrillation.

For tricuspid stenosis, percutaneous valvuloplasty can be highly effective but is influenced by the underlying anatomy. Pre-procedural 3D echocardiography is essential. In cases like double orifice stenosis, or absence of chordae tendineae the dilation may cause severe insufficiency. Negative prognostic factors include right ventricular hypoplasia and excessive thickening of the chordae tendineae or moderator band.

In humans, percutaneous devices for tricuspid insufficiency are under investigation, but cost and size currently prohibit their use in dogs. In selected cases, surgical annuloplasty on cardiopulmonary bypass with valve repair or biological prosthesis placement is an effective option.

Cor triatriatum dexter (CTD) is a rare cardiac malformation that involves the persistence of the valve of the right sinus venosus, In the most common form the persistence of the Eustachian Valve forms a membrane that divides the right atrium into two chambers: a proximal chamber that receives blood from the venae cavae and the coronary sinus, and a distal chamber where the right auricle and the tricuspid valve are located.

From a therapeutic point of view, surgery is indicated in cases of severe obstruction and aims to reduce the mean gradient between proximal and distal right atrium. The interventional dilation procedure with a single balloon catheter has given poor results. In cases where the obstruction consists predominantly of fibrous tissue, the percutaneous interventional

procedure must be performed with an initial dilation using a “cutting balloon” followed by a second dilation with a high-pressure balloon catheter.

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Cloudy Eyes, Clear Solutions:
Understanding and Treating Canine Glaucoma

Speaker: Serena Maini

Glaucoma is a progressive, neurodegenerative disease of the eye that results in irreversible blindness if untreated. It is characterised by optic nerve damage and retinal ganglion cell death, most often associated with increased intraocular pressure (IOP).

In general practice, prompt recognition and immediate management are essential. Acute or uncontrolled glaucoma constitutes an ophthalmic emergency, requiring rapid action to preserve vision and relieve pain.

These notes outline the practical approach to diagnosis, management, and referral of glaucoma in dogs for the general practitioner.

Definition and Pathophysiology

Glaucoma refers to a group of diseases characterised by elevated IOP that exceeds the eye's capacity to maintain normal optic nerve function. The elevation in IOP occurs when aqueous humour production and outflow are imbalanced. Chronic elevation leads to retinal ganglion cell death, optic nerve damage, and progressive, irreversible vision loss.

Aqueous Humour Dynamics

- Produced by the ciliary body epithelium.
- Flows from the posterior chamber through the pupil and into the anterior chamber.
- Exits via:
- Conventional pathway: iridocorneal angle and trabecular meshwork.
- Unconventional (uveoscleral) pathway: through the ciliary muscle and suprachoroidal space.
- Normal canine IOP ranges between 10–25 mmHg

Measuring Intraocular Pressure

Reliable measurement of IOP is key to diagnosis.

Practical Tips:

- Avoid neck pressure when restraining; jugular pressure can artificially elevate readings.
- Interpret results alongside clinical signs, particularly if elevation is mild.
- A single reading is a snapshot in time – a 24-hour intraocular pressure curve will increase chances to identifying an IOP spike.

Methods of Tonometry:

1. Indentation Tonometry (e.g. Schiøtz tonometer) uses a plunger to indent the cornea to a degree that is inversely proportional to the intraocular pressure (IOP) (e.g., Schiøtz tonometer). The reading from the indicator is converted to an IOP value using a calibration chart—the closer the scale is to zero, the higher the IOP. This method can be cumbersome as the instrument must be assembled and the corneal surface kept horizontal, often requiring anaesthesia (which itself will affect accuracy of IOP readings). It should not be used on fragile eyes and is slightly less accurate than newer techniques, though still useful in some settings.

2. Applanation Tonometry (e.g. Tono-Pen®) uses a small, magnetised probe that briefly contacts the cornea; the probe's deceleration speed correlates with IOP (higher IOP = faster rebound). Devices such as the TonoVet® and TonoVet Plus® are easy to use, require no topical anaesthetic, and are well tolerated, particularly in cats. Although relatively expensive, they are increasingly common in general practice.

3. Rebound Tonometry (e.g. TonoVet®) measures the force needed to flatten a defined area of the cornea and converts this into a digital IOP reading. It is durable and easy to operate. The Tono-Pen® is widely used in clinics. Topical anaesthesia is required prior to measurement. This method may slightly underestimate IOP in cats.

Gonioscopy

Gonioscopy assesses the iridocorneal angle and aqueous outflow structures, identifying primary anatomical abnormalities or secondary causes of closure.

- Open angle: Normal appearance with visible pectinate ligament fibres.
- Closed angle: Narrowed or obliterated drainage pathway.
- Pectinate ligament dysplasia (PLD): Thickened, fused fibres reducing outflow.

Interpretation requires experience and is best performed by an ophthalmologist. Most cases will require referral for formal assessment.

Clinical Signs of Glaucoma

Acute Glaucoma:

A true emergency. Rapid increase in IOP causes sudden, painful vision loss.

Key findings:

- Ocular pain: blepharospasm, lacrimation, globe retraction, third eyelid protrusion.
- Corneal oedema and diffuse haze (IOP >40–50 mmHg).
- Episcleral congestion.
- Fixed, mid-dilated pupil (mydriasis) unresponsive to light.
- Visual loss, lethargy, reduced appetite.

Chronic Glaucoma:

Typical features:

- Buphthalmos (enlarged globe).
- Corneal vascularisation and Haab's striae.
- Iris atrophy, lens subluxation/luxation.
- Optic disc cupping and retinal atrophy.
- Phthisis bulbi (end-stage globe shrinkage).

Differential Diagnoses

Several ocular diseases can mimic or accompany glaucoma. Always rule out these differentials when interpreting elevated IOP:

- Uveitis
- Endophthalmitis or Panophthalmitis
- Episcleritis
- Retrobulbar disease or abscess
- Lens-induced uveitis or luxation

Management of Glaucoma

Treatment decisions should consider:

- Presence or absence of vision
- Underlying cause (primary vs secondary)
- Owner and patient compliance
- Long-term cost and monitoring requirements
- Availability of equipment and expertise

Client Communication

- Glaucoma is lifelong.
- Medications control but do not cure.

- Determine if client and patient can be compliant for lifelong medication and monitoring.
- Discuss realistic prognosis and focus on comfort and quality of life.

Medical Management

Medical therapy is the mainstay of glaucoma control in general practice. The goals are to lower IOP, preserve remaining vision, and reduce pain.

1. Carbonic Anhydrase Inhibitors (CAIs): Aqueous humour is formed by the ciliary processes. Its formation depends on the production of bicarbonate (HCO_3). The enzyme carbonic anhydrase catalyses carbon dioxide (CO_2) hydration to carbonic acid (H_2CO_3) which dissociates spontaneously into bicarbonate and hydrogen ions. Inhibition of carbonic anhydrase reduces aqueous humour formation. Topical CAIs available in the UK include brinzolamide (Azopt®), and dorzolamide (Trusopt® / Cosopt®).

2. Prostaglandin Analogues: Prostaglandin analogues increase aqueous drainage mainly via the unconventional uveoscleral outflow. Topical prostaglandin analogues available in the UK include latanoprost and travoprost (generic or branded e.g. Xalatan®, Travatan®)

3. β-Blockers: Used topically to reduce aqueous production (also increase outflow). Limited effect so usually used in combination with a carbonic anhydrase inhibitor e.g. Cosopt® (timolol/dorzolamide). May cause cardiac side effects (bradycardia); use with care and only in patients over 10kg.

4. α-Agonists: Apraclonidine is used in humans and has been shown to have a moderately beneficial effect in dogs; can cause bradycardia and miosis.

5. Cholinergic Agents e.g., pilocarpine: no longer typically used in practice, due to local irritation and the wide range of topical CAIs and prostaglandins available. Not recommended in general practice.

6. Osmotic Agents e.g. intravenous mannitol, oral glycerol: achieve a rapid reduction in IOP, however may cause a rapid loss of body fluid; can induce pre-renal azotaemia and even renal failure. Not recommended in general practice.

Surgical Management

When medical therapy fails or vision remains at risk, referral for surgical options may be considered.

- Gonioimplant surgery

- Cyclodestructive techniques

Indications for Surgery:
- IOP uncontrolled despite maximal medical therapy.
- Vision still present and salvageable.
- Owner able to commit to cost and follow-up.
- Owner compliance high and patient manageable for ongoing care.

Monitoring and Long-Term Care
- Monitor frequently in early stages; extend intervals later.
- Educate owners on clinical signs of recurrence or pain.
- Ensure compliance with drops and revisit schedules.

When to Seek Advice / Refer
- No tonometer
- No appropriate ocular anti-hypertensives in stock
- If you are not sure of the underlying cause or other concurrent conditions (gonioscopy required in most cases)
- Unable to perform a 24-hour intraocular pressure curve
- Considering implant surgery
- Acute or uncontrolled glaucoma is an emergency.

Summary
Glaucoma in dogs is complex but manageable when recognised early and treated appropriately. Success in general practice relies on:
1. Identify clinical signs and measure IOP accurately.
2. Communicate with client and ensure compliance for life-long management.
3. Initiate prompt therapy to reduce pressure and pain.
4. Monitor life-long; focus on comfort and quality of life.
5. Refer when needed.

Early detection and a confident, informed response can make the difference between vision preservation and blindness.

Mastering the Art of Funduscopy

Speaker: Serena Maini

Funduscopy is a valuable but often underutilised skill in general practice. It allows direct assessment of the posterior segment of the eye and can provide important information about both ocular and systemic disease.

One of the main challenges is distinguishing normal from abnormal, as the appearance of the fundus varies widely between patients. Developing confidence in funduscopy requires an understanding of basic anatomy, recognition of normal variation, and a structured approach to interpretation.

These notes provide a practical guide to performing funduscopy and interpreting common findings in dogs and cats.

Anatomy Revision – Thinking in Layers

A helpful way to understand the fundus is to think of it in layers, like a pizza built from the base upwards. This approach makes it easier to relate anatomy to what is seen clinically.

From outermost to innermost:

- Sclera: the white, fibrous outer layer of the eye
- Choroid: a vascular layer containing blood vessels and pigment
- Tapetum: a reflective layer (present in the dorsal fundus only)
- Retinal pigment epithelium (RPE): a pigmented layer, which lacks pigment over the tapetum
- Neurosensory retina: a transparent layer containing photoreceptors and retinal vessels. Has a ‘dulling’ effect on the reflectivity of the tapetum.

Understanding these layers helps explain why the fundus appears the way it does and how disease alters its appearance.

What is Normal?

A key challenge in funduscopy is recognising that normal can look very different between patients. Building a mental picture of normal variation is essential before abnormalities can be identified.

Species Differences

Dog

- Tapetum: Bright and reflective, located dorsally
- Non-tapetal fundus: Darker, ventral region. Pigment dilution in the RPE and choroid lead to variations ranging from reddish-tan (common in dogs with pale, yellow irises) to a red striped appearance in blue-eyed, dogs.
- Optic nerve head (aka optic disc): White–pink, round/oval to triangular, located at the junction of tapetal and non-tapetal areas.
- Retinal vessels: 3–5 main veins converge at the optic nerve head; arteries are thinner and more numerous.

Cat

- Tapetum: Larger and brighter (often yellow–green)
- Non-tapetal fundus: Less variation than dogs
- Optic nerve head (aka optic disc): Smaller, darker (due to less myeline), located within the tapetum.
- Retinal vessels: Fewer vessels, no obvious convergence or anastomosis over the optic nerve head.

Normal Variation

Normal fundi can vary significantly between individuals:

- Tapetal colour may range from blue to green, yellow, or orange
- The size and shape of the tapetum vary between individuals
- Pigmentation may be reduced in light-coloured or blue-eyed animals (subalbinotic / albinotic)
- Choroidal vessels +/- sclera may be visible in subalbinotic / albinotic fundi

Recognising this variation is essential to avoid over-interpreting normal findings as disease.

Fundus Abnormalities – A Structured Approach

When assessing the fundus, it is helpful to focus on three key areas:

- Tapetal reflectivity
- Retinal vasculature
- Optic nerve head

Tapetal Reflectivity – The “Dusty Mirror”

A useful way to think about tapetal reflectivity is as a mirror covered by dust:

- The tapetum is the mirror
- The retina is the layer of dust

Changes in reflectivity reflect changes in retinal thickness:

- Increased reflectivity (hyperreflective) indicates retinal thinning or loss. Causes include:
    - Retinal atrophy (e.g., progressive retinal atrophy)
    - Retinal detachment
    - Taurine deficiency in cats
    - Chronic post-inflammatory retinal degeneration / atrophy

- Decreased reflectivity (hyporeflective) indicates retinal thickening or subretinal accumulations. Causes include:
    - Retinal oedema / inflammation (e.g., chorioretinitis)
    - Retinal folds
    - Subretinal effusion / granulomas (e.g., blastomycosis)
    - Cellular infiltration

Retinal Vasculature

Changes in the retinal vessels can indicate both ocular and systemic disease. Careful assessment of vessel size, shape, and distribution is essential.

Vascular patterns include:

- Vascular attenuation (narrowing of vessels) e.g., retinal degeneration, retinal atrophy
- Vascular tortuosity e.g., systemic hypertension
- Haemorrhage e.g., hypertension, anaemia, coagulopathy
- Lipaemia retinalis (vessels appear pale pink due to lipid accumulation) e.g., hyperlipidaemia
- Perivascular cuffing e.g., vasculitis due to feline infectious peritonitis

Optic Nerve Head (ONH)

The optic nerve head should be assessed for size, shape, colour, and position.

Important changes include:

- Swelling (raised ONH) may be seen with optic neuritis or papilloedema
- Retinal vessels appear to bend over the disc margin
- Pallor suggests optic nerve degeneration
- Hyperaemia suggests inflammation / hypertension
- ONH cupping is seen in glaucoma due to loss of optic nerve fibres and posterior displacement of optic nerve tissue, due to high pressure.

Comparing the focus of the optic disc and surrounding retina can help identify elevation or depression.

How to Perform Funduscopy

Funduscopy can be performed using either direct or indirect techniques. Both have advantages and are often complementary.

Direct Funduscopy

- Real, direct image
- High magnification
- Narrow field of view
- Useful for detailed assessment

Indirect Funduscopy

- Virtual, inverted image
- Lower magnification
- Wide field of view
- Better for screening and peripheral examination

<u>Performing close direct funduscopy</u>

- Instrumentation: Direct ophthalmoscope
- Set dioptre wheel to '0'
- Adjust for your refractive error if necessary (positive lenses=black or green, negative lenses=red)
    - E.g., if your glasses prescription is -4.0 (short-sighted), your neutral setting will be - 4
    - E.g., if your glasses prescription is +2.0 (long-sighted), your neutral setting will be + 2
- Position yourself close to the patient
- Use brow rest
- Allows detailed assessment of intraocular structures
    - Retina will be in focus at 0 D
    - Lens will be in focus at +8 to +12 D
    - Cornea will be in focus at + 20 D

<u>Performing Indirect Funduscopy</u>

- Instrumentation: Condensing lens (20 D, 30 D or 2.2 Pan retinal) and focal light source (e.g., bright pen torch, Finhoff transilluminator)
- An assistant will need to restrain the patient
- Align the light source with your visual axis (i.e., place alongside your temple)
- Position yourself at arm's length from the patient

- Hold the condensing lens with with the thumb and forefinger, and balance your hand on the patient's head
- Direct the light into the patient's eye and locate the tapetal reflection
- Lower the condensing lens in front of the patient's eye (approx. 3-5cm away)
- Examine the entire fundus, including the periphery (needs mydriasis)
- Paying particular attention to **tapetal reflectivity, retinal vasculature and the optic nerve head**.

A panoptic ophthalmoscope presents a good compromise between the direct and indirect ophthalmoscopes. The image is real and intermediate in magnification between the two types.

Practical Tips

- Dilate the pupil (e.g. tropicamide)
- Turn off the lights to improve visibility
- Position yourself correctly and stabilise your hand
- Examine systematically, including the periphery
- Focus on: Tapetal reflectivity, Retinal vessels, Optic nerve head

Focal planes

In a normal eye, the fundus lies in one plane and is in focus at the same setting.

If the ONH is:
Raised → requires a more positive lens to focus
Depressed → requires a more negative lens

A difference in focus between the ONH and retina indicates a change in depth.

Summary

Funduscopy is a valuable clinical skill that improves with practice and a structured approach.

Key points:

- Understand the layered anatomy of the fundus
- Recognise the wide range of normal variation
- Pay attention to: tapetal reflectivity, retinal vasculature, optic nerve head
- Use systematic examination techniques
- Small adjustments in technique can greatly improve your view

With time and experience, funduscopy becomes an invaluable tool for identifying both ocular and systemic disease.

INFLAMMATORY FELINE ORAL DISEASES

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CAUDAL STOMATITIS is a severe inflammatory reaction of the oral tissues of cats. It is a **clinical diagnosis** of inflammation and proliferation of the gingiva and oral mucosa. Specifically, it is inflammation associated with the caudal mouth (mucositis), which is the delineating factor between caudal stomatitis and periodontal disease.
Multiple etiologies may exist that, either singularly or combined, create the inflammation. Possible causative agents include an inflammatory response to plaque bacteria, viruses (FCV), *Bartonella Henselae* infection, or altered immune status (FeLV or FIV).
For Type 1 TR the agreed ethology is the inflammatory process surrounding affected teeth.
In cases of caudal stomatitis, the history typically includes: halitosis, dysphagia, pawing at the mouth, reluctance to eat, anorexia (complete or partial), crying out in pain when eating or yawning, weight loss, decreased grooming (which results in an unkempt appearance), and drooling (pseudoptyalism).
The affected gingiva and oral mucosa have varying amounts of inflammation, proliferation, and ulceration. The inflammation is typically, but not always bilaterally symmetrical with friable oral tissues that bleed easily.
Caudal Stomatitis is a clinical syndrome and does not indicate a specific etiology or diagnosis. Diagnosis is made by visual inspection of the oral cavity. Diagnostic tests to further define the disease should minimally include: dental radiographs, a minimum data base (CBC, chemistry panel, t4, U/A) to evaluate for underlying and/or concurrent systemic health problems, and evaluation of FeLV/FIV status. A biopsy should be taken and submitted for histopathology, especially if the inflammation is asymmetrical or otherwise atypical, or if radiographic findings are suspicious for neoplasia.

The goal of therapy for this disease process is to completely eradicate the oral inflammation. However, maintaining a state of decreased inflammation is sometimes the best that can be achieved. Several treatments have been suggested, including: extraction of all premolars and molars, full mouth extraction, chronic immune-suppressive treatment, anti-inflammatory treatment, interferon, and antibiotics.

<u>Surgical therapy</u>

Controlling inflammation is the key to management of this disease process. Therefore, any tooth affected with inflammation from any cause should be extracted. All remaining teeth must receive strict homecare and routine professional cleanings to keep inflammation at bay. However, since the majority of patients have widespread inflammation, the most successful long-term treatment for cats with chronic gingivostomatitis is the COMPLETE extraction of all premolars and molars including the periodontal ligament as well as smoothing the alveolar bone. An additional step

taken by many veterinary dentists is to perform careful aveloplasty to remove periodontal ligament remnants, which has anecdotally improved success rates.
Extraction of the canine and incisor teeth is indicated when the inflammation extends to include the gingiva surrounding them. Therefore, this author will typically perform FULL mouth extractions in cases of significant oral inflammation, while some veterinary dentists prefer to leave the canines and incisors if at all possible. Post-operative dental radiographs **must** be exposed to document complete extraction of all tooth roots.

Medical therapy

In cases where owners are reluctant to have multiple extractions performed early in the course of treatment, medical management may be attempted to reduce bacterial load and inflammation. The majority of the products utilized are oral medications, which require daily to twice daily administration. This is difficult to achieve in cats in general, and the oral pain and inflammation only serves to complicate matters. Finally, many of the products have significant side effects.
Clients should be informed that medical therapy is almost invariably a life-long process with numerous side effects. In addition, there are no medical protocols that have been shown to be completely effective; rather they just temporarily reduce the clinical signs. Therefore, clients should be counseled that delaying extraction therapy often results in a decreased response, possibly yet again necessitating long term medical therapy.

Systemic antibiotics *may* result in some improvement in the amount of oral inflammation. However, this is generally temporary at best, and most patients will relapse even during the course of antibiotic therapy. Rinsing with a. 0.12% Chlorhexidine gluconate solution may also be beneficial in some cases.

Corticosteroids are by far the most commonly used and effective drugs for immune modulation. However, long-term use of corticosteroids may have detrimental effects such as the induction of diabetes mellitus and opportunistic infections. Use the lowest effective dose and monitor biochemical values on a regular basis. Injectable treatment (methylprednisone 10-20 mg SC) is usually recommended initially, due to the degree of oral pain. This typically results in clinical improvement within 24-48 hours, and lasts for 3-6 weeks. However, these intervals tend to progressively shorten the longer the injections are continued. For these many reasons, chronic corticosteroid therapy should only be performed as a last resort when an owner will not allow extractions.

Cyclosporine A has been purported as an immunosuppressive drug for cats with chronic gingivostomatitis. Some have promoted as an alternative to extractions in order to avoid the use of glucocorticoids. However, this author prefers to withhold its use to those cases where additional medical management is necessary post-extractions. There is scant information which supports its use other than one unpublished veterinary study, which showed efficacy in cases refractory to extractions. However, it may provide an alternative to long-term steroid therapy. It must be used with caution in cats with hepatic or renal disease. In addition, it must be noted that cyclosporine is **not**

approved in cats and there are reports of fatal opportunistic infections associated with its use.
The bioavailability of the three available forms of cyclosporine is quite variable, and therefore dosing depends on which form is used. Recently, a veterinary product has been released for canine atopy, and may be of use in cats. Serum cyclosporine levels should be evaluated within 24-48 hours of beginning therapy and then monitored weekly for a month, and monthly thereafter, to maintain concentrations within the therapeutic range and avoid toxic levels. In addition to monitoring cyclosporine levels, standard biochemical tests should be routinely performed as surveillance for deleterious side effects.
Feline interferon. There is currently significant interest in the use of this product for caudal stomatitis. It is reported to not only provide an antiviral effect, but to also provide an immunomodulatory effect and bring about a return to normal local immune response. Several studies have shown efficacy in resistant cases, but as of yet, no evidence exists to show that it works as a primary treatment. There are several options for therapy including intralesional injection and/or oral vs. injectable systemic treatment. The preferred method at this point is to inject 5 MU intralesional (often at the time of extractions) and then follow this up with the remainder of the vial (5 MU) diluted into 100 cc of sterile saline and administered Per Os by the owner at a dose of 1 ml once daily for 100 days. Finally, Lactoferrin (topically @ 40 mg/kg), gold salts (1-2 mg weekly for 8 weeks and then monthly), levamisole (2-5 mg/kg PO three times weekly), Doxycycline (2 mg/kg BID, with decreasing doses if effective), and co-enzyme Q10 (30-60 mg daily) have also been used with occasional success. However, none of these therapies are recommended at this time.
In conclusion, at the time of this writing, surgical extraction therapy is the preferred treatment and should be performed as soon as possible. Medical therapy should be reserved for those cases where clients will not allow (or cannot afford) extraction therapy.

TOOTH RESORPTION (TR)

Completely subgingival TRs (those that have not progressed to the crown of the tooth) likely cause no discomfort for the patient. This presumption is based on the fact that similar lesions in humans are non-painful. Once lesions progress to the crown of the tooth they are typically very painful, however cats rarely show overt clinical signs. It is possible that the tissue filling the defect may provide some protection from sensitivity.
Most TRs are quite large before they become clinically evident. Therefore, it is very important to perform a thorough oral exam on all cats. Visualization of a resorptive defect near the gingival margin is almost diagnostic for a TR. The vast majority of feline patients afflicted with TRs will show no outward clinical signs. However, patients have been presented for oral pain, anorexia, ptyalism, lethargy, depression, dysphagia, and halitosis.

Type 1

The lesions are first clinically evident on the crown at the gingival margin when the internal resorption reaches the enamel. The gingiva surrounding the teeth with type 1 lesions is usually affected with a significant inflammatory problem such as L/P stomatitis or periodontal disease. In cases of periodontal disease, it is very common to have calculus covering the lesion. This calculus must be removed to properly diagnose the lesion. The clinically visible defect typically indicates a much larger subgingival defect (tip of the iceberg). Hyperplastic inflamed gingiva also often conceals the defect.

Type 2

Type 2 TRs are usually associated with only localized gingivitis on oral exam, in contrast to the more severe inflammation due to periodontal disease or gingivostomatitis seen with type 1 lesions. Type 2 TRs often begin just below the gingival surface near the cemento-enamel junction close to the gingival margin, or "neck" of the tooth. Visualization of a defect on the tooth surface or of gingival hyperplasia onto the crown surface is indicative of a TR. The lower third premolar is commonly the first tooth affected in these cases, however canines can also be affected without other teeth being involved. Cats with a type 2 TR will generally have more than one lesion and are at increased risk for developing additional lesions. Early lesions may appear only as localized marginal gingivitis in a mouth that has very little inflammation. TRs can be differentiated from a normal furcation or periodontal pocket with a dental explorer. A dental explorer catches on the edge of the defect which feels rough, as opposed to the smoothness of a furcation. Larger lesions are seen extending above the gingival margin onto the crown of the tooth. Advanced type 2 lesions often have gingiva or inflamed granulation tissue growing up onto the tooth crown. In end-stage lesions, the tooth fractures and the gingiva grows over the area, developing a firm "bump" in the area of the missing tooth. This bump distinguishes these lesions from tooth loss caused by periodontal disease or previously performed extraction.

<u>Diagnosis:</u>

Determination of whether a lesion is type 1 or type 2 requires a dental radiograph. Type 1 lesions have normal root density in some areas (as compared to surrounding teeth) and also a well defined periodontal space in some areas. These teeth often have a definable root canal in the intact part of the tooth. Additionally, type 1 lesions are generally associated with periodontal bone loss (either horizontal or vertical). The radiographic appearance of type 2 lesions is that of teeth which have a different radiographic density (as compared to surrounding teeth) as they have undergone significant replacement resorption. Radiographic findings will include areas with no discernable periodontal ligament space (dentoalveolar ankylosis) or root canal, and in late stages there will be little discernable root structure (ghost roots).

<u>Management</u>

Restoration of any TR carries a very poor prognosis because the odontoclasts remain present under the restoration, and therefore the resorptive process continues. In short

order, usually around 6 months, the restoration will be lost and the pain and inflammation will recur. In addition, the visible lesion normally represents only a small part of the actual pathology (i.e. tip of the iceberg).
Treatment of choice for teeth with TRs is extraction. Recently, crown amputation has been suggested as an acceptable treatment option for advanced type2 lesions. Crown amputation results in significantly less trauma to the patient with faster healing than complete extraction. This procedure, although widely accepted, is still controversial. Most veterinary dentists employ this technique, however in widely varying frequency. Veterinary dentists typically use this treatment option only when there is significant or complete root replacement by bone. Unfortunately, the majority of general practitioners use this technique far too often. Crown amputation can only be performed on teeth with radiographically confirmed type 2 TRs which show no peri-apical or periodontal bone loss, with roots which are being completely resorbed. Crown amputation should not be done for teeth with: type 1 TRs, radiographic or clinical evidence of endodontic or periodontal pathology, inflammation, or infection. It should also not be performed in patients that have any evidence of inflammation in the caudal tissues between the upper and lower molar teeth, or that are known to be positive for retrovirus. Those practitioners without dental radiology capability SHOULD NOT perform crown amputation. In these cases, the teeth should either be fully extracted or the patient referred to a facility with dental radiology.
Crown amputation involves creating a small gingival flap, and then using a high-speed bur to remove the entire crown to the level of the alveolar bone. Following radiographic conformation that the tooth is removed to at least the level of the bone (and that the resulting bone is smooth), the gingiva is sutured over the defect.

EOSINOPHILIC GRANULOMA COMPLEX (EGC) is a group of conditions which share a common etiology, as well as some histopathological features. While these lesions have been reported in dogs (especially Siberian Huskies and Malamutes and Cavaliers), they are much more common in cats. The discussion in this section will relate to cats, although the disease process is similar in either species. The true etiology of these conditions is unknown. Local accumulation of eosinophils (and their release of inflammatory agents) is thought to initiate the inflammation and necrosis seen in most of these lesions. The presence of eosinophils suggests that these lesions are secondary to an immune-mediated or hypersensitivity reaction. This reaction may result from a local (food) or systemic (flea allergy or atopy) allergy, although these lesions have been seen in cases where allergic and infectious disease have been ruled out. Additional causes may include a response to irritation, such as chronic grooming, or a traumatic malocclusion. Finally, there appears to be a genetic predisposition to this Syndrome.

<u>Management</u>

The acute disease process is best treated with corticosteroids. However, corticosteroids should not be used for long-term disease control, due to the significant systemic side-effects. The typical initial protocol is prednisone 2 mg/kg q 12 hours for 3–4 weeks. Other corticosteroid options include intralesional triamcinalone (3 mg weekly) or methyl prednisone injections (20 mg q 2 weeks). Author prefers bethamethasone (diprophos) injections. Antibiotic therapy is required in some cases to induce remission or to treat secondary infection. In addition, there are cases that appear to respond to antibiotic therapy alone as has doxycycline at 10 mg/kg PO q24h.
Therefore, it is routine in our practice to treat mild cases initially with antibiotics alone and more severe cases with a combination of antibiotic and corticosteroid medications. Thorough allergy testing should be performed, especially in nonresponsive or recurrent cases. If an underlying allergic component is found, specific treatment can be instituted with a good rate of success. Many cases remain idiopathic and require lifelong therapy. Options for long cyclosporine. Cyclosporine has recently been introduced as a veterinary labeled product for atopy and appears as effective as corticosteroids for atopic dermatitis in dogs and cats. Cats should be treated for 60 days with 25 mg cat (4.9 to 12.5 mg/kg), given 2 h before a meal. It has also been proven to be an effective medication for longterm therapy of oral eosinophilic diseases. In addition, a lower incidence of severe sideeffects may be expected in comparison to steroids. This is especially valuable in cases requiring long-term therapy. Consequently, many dermatologists use this medication rather than corticosteroids for inflammatory skin diseases, including the EGC. When treating larger patients long term, the addition of ketoconazole may lower the necessary dosage of cyclosporine and subsequent cost to the client. It is important to note that cyclosporine is currently not approved in cats and there are reports of opportunistic fungal and fatal protozoal infections associated with its chronic use. Therefore, using the lowest effective dose (by tapering), and performing regular therapeutic levels as well as routine blood testing, are recommended. Hormonal therapy is another medical option for treating these lesions1, but the significant potential side-effects should make this the last option. Additional therapies that may benefit some cats include antihistamines, chlorambucil (more effective on lymphocytes) and gold salts. One practitioner has had success using Prozac in one patient with oral EGC that was refractory to all other treatments.

Surgical removal of these lesions has been performed with some success, including laser and cryosurgery. Finally, radiation treatment has been used effectively in some cases.

PERIODONTAL DISEASE

Periodontal disease is a very common problem in veterinary patients. It has numerous severe local and systemic ramifications, however outward clinical signs occur only **very** late in the disease course. This means that it is significantly underdiagnosed, and even when recognized and treatment recommended, clients are reluctant to comply as they do not perceive the problem. Regardless, proper and

prompt therapy of periodontal disease is beneficial for the health of the patient as well as financially for the practice.

Juvenile gingivitis occurs in young cats around the time of permanent teeth eruption and is associated with marginal and free gingiva inflammation circumferentially. This is inflammation of the gingiva during and just after tooth eruption and may be accompanied by persistent deciduous dentition. FOT The gingival bleeding index is II or III so bleeding may occur on probing or spontaneously. Thick plaque deposition is present but gingival probing depth non necessarily exceeds 1mm. This is self limiting in most cases, however home care (brushing or chlorhexidine rinses are recommended to decrease the inflammation. If the condition does not resolve in a short period of time, additional diagnostics and therapy is recommended as this could tend to switch into juvenile periodontitis.

FELINE OROFACIAL PAIN SYNDROME (FOPS)

Feline orofacial pain syndrome (FOPS) is a pain disorder of cats with behavioural signs of oral discomfort and tongue mutilation. FOPS is suspected to be a neuropathic pain disorder and the predominance within the Burmese cat breed suggests an inherited disorder, possibly involving central and/or ganglion processing of sensory trigeminal information. The disease is characterised by an episodic, typically unilateral, discomfort with pain-free intervals. The discomfort is triggered, in many cases, by mouth movements. The disease is often recurrent and with time may become unremitting - 12% of cases in this series were euthanased as a consequence of the condition. Sensitisation of trigeminal nerve endings as a consequence of oral disease or tooth eruption appears to be an important factor in the aetiology - 63% of cases had a history of oral lesions and at least 16% experienced their first sign of discomfort during eruption of permanent teeth. External factors can also influence the disease as FOPS events could be directly linked to a situation causing anxiety in 20% of cats. FOPS can be resistant to traditional analgesics and in some cases successful management required anti-convulsants with an analgesic effect.

References available upon request jgawor@pp.com.pl

RESTORATIVE DENTISTRY

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Restorative dentistry is an exceedingly common procedure in human dentistry. The main reason for restorative treatment in humans is bacterial caries. Treatment is often sought due to dental pain (sensitivity) caused by dentin exposure. Our animal patients feel the same discomfort as we do, but they typically do not show outward clinical signs.

The common indications for restorations in veterinary patients are: enamel hypocalcification, caries, uncomplicated crown fractures, attrition/abrasion, and access sites for endodontic therapy. In addition, there are occasional other enamel defects which may benefit from restoration. Restoring of feline tooth resorption is no longer recommended due to the exceedingly high failure rate.

The most common types of restorations are bonded sealants and composite restorations as these can cover the majority of conditions. However, there are several indications which may benefit from glass ionomer or amalgam restoration, or even full crown coverage.

Tooth Anatomy:

Teeth are roughly broken up into three layers: enamel, dentin, and pulp.

The innermost layer is the endodontic system (root canal or pulp). It contains the nerves, blood vessels, and connective tissue which supply and nourish the tooth during life. The blood and nervous supply enters the tooth through the very bottom or apex of the root.

The outer layer of the tooth crown is enamel, which is an inorganic substance. It is virtually all (97%) calcium and phosphorus and is the hardest substance in the body. Enamel has no nervous or circulatory system. It is applied in a very thin layer (less than 1 mm thick in veterinary patients) over the tooth surface during development by a cell layer called amelioblasts. Once eruption has occurred, enamel cannot be replaced or repaired.

The central layer, which is the vast majority of the tooth structure in mature patients, is dentin. Dentin has roughly the same mineral content as bone. Dentin is a somewhat living structure which has a nervous supply and can occur can respond to stresses. Running at right angles to the root canal all the way around the tooth from the root canal out to the enamel are dentinal tubules. Each one of these dentinal tubules contains an odontoblastic process, which is basically a nervous supply; however they are only sensory and can only report changes as pain. There are approximately 50,000 dentinal tubules per mm^2 coronal dentin. Therefore, a 1 cm area of enamel loss will expose 3-4 ***million*** odontoblasts!

Response to Damage:

Exposure of the dentinal tubules will lead to much quicker dentinal fluid flow out through these dentinal tubules via the capillary effect. This increase in fluid flow deforms the A-*delta* C-*delta* fibers and thus will be perceived by the patient as pain. Anything that will change the flow rate will cause the nerves to fire and result in pain (sensitivity). This includes heat, cold, and desiccation. The sensitivity is actually a sign of low grade pulp inflammation known as *pulpitis*.

In addition to the sensitivity produced by the exposure of the dentinal tubules, there is a possibility of ingress of bacteria into the root canal system. In some cases this can result in endodontic infection and subsequent abscessation. This occasionally can be seen clinically as a swelling or draining tract, but is generally subclinical and therefore undiagnosed. The only way to diagnose this infection is via dental radiographs.

Once exposure has occurred, material will accumulate on the surface which can mineralize and block the tubules (Smear Layer). In addition, the tooth will sense the disruption of its normal protection and will attempt to shield itself from the harmful invaders of the oral environment as well as decrease the patient's pain. This will take the form of creating either tertiary or sclerotic dentin. Eventually (as long as the causative problem is not allowed to progress), this may result in the end of sensitivity. However, this process is lengthy (likely months) and the patient is painful and susceptible to infection during this time.

Indications for restorations:

Uncomplicated Crown Fractures: These are very common in large breed dogs. They occur when a piece of the crown is broken off, which exposes the dentin but not the pulp. Occasionally, these teeth can become infected through the dentinal tubules. Again, this infection will go undiagnosed without dental radiology. However, teeth with no to small pulpal exposures tend to be the ones with clinical abscessation.

Complicated Crown Fractures:
These teeth have direct pulp (nerve) exposure. Prior to tooth necrosis, the viable nerve is excruciatingly painful.Following tooth death, the root canal system will act as a bacterial super-highway creating not only local infection, but also a bacteraemia which has been linked to more serious systemic diseases. The owners of these patients may be reluctant to pursue therapy as "It does not seem to bother the dog". Fractured and/or infected teeth do affect the pet and they will often act better following therapy. Veterinary patients are known for being stoic, and therefore lack of outward signs of oral pain should not be misinterpreted as a benign state.
All teeth with direct pulp exposure must be treated with endodontic or exodontic therapy. A restoration alone is NOT sufficient therapy as the tooth will almost

invariably become non-vital and subsequently infected. Restorations for direct pulp exposures are indicated ***only*** following standard root canal or vital pulp therapy.

Caries:

True bacterial caries are rare in dogs and almost unheard of in cats. They are most common on the occlussal surface of the upper first molars, but can be seen on any tooth. In addition, the most common breed is a German Shepherd dog. Early lesions can mimic wear, and are best diagnosed by tactile feel of the defect with a sharp explorer. If it is sticky, like wax, it is likely a caries lesion. These lesions can progress into the endodontic system resulting in pain and infection. Caries are most common on the occlusal surface of the maxillary first molars. Other possible locations are occlusal surfaces of the other molars and maxillary fourth premolar as well as the deep developmental grooves between the cups of the mandibular first molar and maxillary fourth premolar teeth. Smooth surface caries can occur on the sides of the teeth, especially on contact surfaces (i.e. the walls of the teeth which face each other). Finally, root caries are possible.

Caries lesions can only really be initiated in areas where food can become trapped, allowing the digestion by bacteria to begin.31 Therefore homecare can help avoid these lesions. Initially the pit and fissure lesions develop on the lateral walls of the defects. The initial lesion is a surface decalcification of the enamel caused by a drop in the pH. The demineralization follows the direction of the enamel rods. Eventually the caries will break through the enamel and invade the dentin. Once this occurs, the lesion will expand laterally fairly quickly as dentin is less mineralized than enamel. Once the dentin is destroyed, the unsupported enamel will collapse, quickly expanding the lesion. Finally, these lesions can progress into the endodontic system resulting in pain and infection *Enamel Hypocalcification (EH):* Hypocalcification results from disruption of the normal enamel development. Ameloblasts are very sensitive and minor injuries can result in enamel malformation. Areas of enamel hypocalcification will generally appear stained a tan to dark brown (rarely black) color, and may appear pitted and rough. The tooth surface is hard however, as opposed to the soft/sticky surface of a caries lesion. The areas of weakened enamel are easily exfoliated which will expose the underlying dentin, resulting in staining. Dentin exposure will result in significant discomfort for the patient (see uncomplicated crown fractures above). The roughness of the teeth will also result in increased plaque and calculus retention, which in turn leads to early onset of periodontal disease.

Abrasion/Attrition

These lesions are somewhat similar to uncomplicated crown fracture, however are due more to a long term wearing of the tooth as opposed to a one time traumatic event. *Attrition* is caused by tooth on tooth contact, whereas *abrasion* is caused by a tooth wearing against something foreign.

Access site for endodontic treatment.

Proper restoration of fracture and access sites of endodontic treatment is very important. This is due to the fact that restorative leakage is thought to be a major cause of endodontic failure, at least in humans. Therefore, proper site preparation is critical. This includes removing all endodontic filling materials and placement of a glass ionomer intermediate layer. Finally removal of unsupported enamel rods and a composite restoration is recommended

Materials

Glass Ionomers are supplied as two separate materials, usually an acid soluble silicate glass powder as the dry component and an aqueous solution of polyacrylic acid or a copolymer of polyacrylic acid with other acids as the liquid component. The polyacrylic acid chains are cross-linked by calcium ions to form a solid mass. The initial set occurs in about five minutes, after which the material can be finished. However, a more complete set continues for 24 hours, during which time it is still hardening. Moisture in the material is an important part of the reaction, and it must be protected from desiccation. It must also be protected from external moisture contamination during the set, which can result in sorption and a weaker material. To provide this protection a layer of unfilled resin or varnish should be placed immediately after placing the material and after any finishing.
The mixture ratio of powder to liquid is important and must be measured precisely. The biggest advantage of using a glass ionomer is its ability to bond inherently to tooth structure. Therefore it does not require the use of a bonding agent. For use in humans, some operators also like the fact that fluoride ions are leached from the surface and may exert a local cariostatic effect, possibly protecting against recurrent caries.
The main disadvantages of glass ionomers are their higher wear rate, greater susceptibility to abrasion, poorer polishability, and less resistance to fracture compared to composites.
Their major indications are: the first layer of restoration in caries, vital pulp therapy, endodontic restorations and sub gingival restorations.

Composites - they are a composite of two distinctly different materials that have been combined to take advantage of the strong qualities of each. The mixture is generally a dimethacrylate monomer and an inorganic filler.
Composite materials need an initiator to start the polymerization reaction. There are two main categories: auto-initiated and light-initiated. The auto-initiated composites are supplied in two separate containers that are mixed together. The mixing creates free radicals that initiate the polymerization reaction. Light-initiated composites

require a light-activation unit to begin the reaction. This material always require bonding systems.
The biggest disadvantage of composite resins is their tendency to shrink when they polymerize. When the restorative shrinks, stresses are placed on the bonded interface. This can result in gaps between the restoration and the dentin, and can open margins that cause microleakage. Additionally composites require very clean and dry conditions to polymerize which is sometimes difficult to obtain.
Composites are the best restorative material for esthetic results and have several forms available: flowable, dense flowable and solid.

Amalagams

Dental amalgam is a special alloy that uses liquid mercury to combine other solid metals at room temperature. It remains the hardest material available today for direct placement restorations, although some new posterior composite materials are now rivaling its strength.

In veterinary dentistry, amalgam can still be a good selection for treatment of occlusal table (Class I) caries on molar teeth. Although this is not a common lesion, it occurs occasionally. This surface is subjected to heavy occlusal loads and therefore benefits from a very hard material. It is also an area in which esthetics is not an issue. The undercuts required to provide mechanical retention are still needed even when bonding agents are used, since their bond strength to amalgam is not strong enough for retention of the filling. These undercuts should be extremely small - almost microscopic - to be functional without removing excessive sound tooth. Generally, a very small undercut with a number ó to 1 round bur is plenty; a round bur is better than an angled bur such as an inverted cone to prevent sharp angles that would create a stress riser. When shaped, a Class I cavity should not have the enamel margins beveled, nor should any cavity of any class have beveled enamel margins when using amalgam as the restorative. After shaping the defect, a layer of a dentin bonding agent should be placed to seal the dentin. Then the amalgam is triturated in an amalgamator and is placed and condensed incrementally into the defect. After final compaction, the restoration is carved to the desired shape, burnished, and later polished.
The most important disadvantages of amalgams are: presence of mercury, need for specific cavity shaping, marginal leakage and poor esthetic results of restorations

Therapy:

Diagnosis: First, perform a thorough visual exam to determine the presence of pulp exposure or other extensive damage. Finally, expose a dental radiograph to rule out endodontic disease. If there is radiographic evidence of endodontic disease root canal therapy or extraction is indicated.

Tooth Preparation: Scale and polish the surface of the tooth to be treated. Make sure to use fluoride free pumice for polishing to avoid interfering with future acid etching.

If treating a small uncomplicated crown fracture, no actual restoration will be placed. Therefore smooth the rough edges with a white stone or fine diamond bur. This can be followed with sanding discs if necessary.

In cases where a restoration will be placed, it is recommended to use a coarse diamond or carbide bur for the preparation. This will leave a rough surface and increase bond strength. Furthermore, all non-occlusal edges should be beveled. This will make a more gradual transition of color as well as increase the amount of enamel for bonding.

For EH cases, remove all weakened diseased enamel with a coarse diamond bur and bevel the edges

For caries cases remove all carious dentin as well as extend the prep into area where there is a high probability of extension. Then make sure that all of the unsupported enamel edges are removed. The bottom of the prep should be flat and the sides of the dentin parallel or very slightly undercut.

Bonding:

Acid etching: This step is performed with a 37% phosphoric acid. The purpose is to remove all impurities from the tooth surface and slightly demineralize the tooth surface of the tooth. This will lead to increased surface area for bonding. Place the supplied acid on the tooth surface and let stand for 10-30 seconds. After the prescribed time, rinse thoroughly (20 seconds) as insufficient rinsing will result in residual acid remaining in the dentinal tubules and result in sensitivity. Finally, dry the area lightly (do not desiccate) as over drying will weaken bond strength.

Place bonding agent

There are many options for bonding agents that fall into two main types: "One Step" which combines the primer and bonding agent in 1 bottle and "Two Step" which have separate primer and bonding agents. In addition, the self-etching systems have shown promise and are less technique sensitive.

The bonding agent should be applied in a very thin layer. After it is applied, it is light cured with an intense blue light in the visible range for 10 seconds. There are many options for bonding agents that currently fall into two main types:

Fourth generation: have separate primer and bonding agents
Fifth generation: combines the primer and bonding agent in 1 bottle.

There currently exist products that combine all three steps in one step (Self-etch bonding adhesives (SEBA)). While there is some research that these have good

bonding strength, they do not have sufficient clinical results to be recommended at the time of this writing.
Below are generalized recommendations and times, always follow manufacturers recommendations for the particular product you are using.

4th generation
a. The primer should be applied in a very thin layer
b. After 10 seconds, it is air thinned for 15 seconds
c. The bonding agent is applied (C) and thinned,
d. Finally, the bonding agent is cured with an intense blue light for 10 seconds

5th generation
These are placed as above, except that the priming step is not performed as the primer and bond are mixed. It is applied, thinned, and light cured.

Restoration

For uncomplicated crown fractures, place a layer of unfilled resin over the bonding agent and light cure. This completes the therapy.

For defects to be filled, the composite is placed and then manipulated to fill the defect. This can be done with a plastic filling instrument or a beaver tail coated with unfilled resin. Once the defect is filled (to slightly overfilled) and the restoration roughly contoured, the restoration is light cured. After light curing, the restoration can be smoothed and shaped with white stones, fine diamonds, or sanding discs. Once finished. A layer of unfilled resin should be pace to fill in areas of polymerization shrinkage and smooth the final restoration.

Smoothing and finishing the restoration: The restoration is them smoothed and shaped to fit the restorative margins. This can be done with a medium to fine diamond or finishing bur. Further smoothness can be achieved with sanding discs. The restoration is then carefully checked, ensure that the restoration is not too high.

The margins should be carefully checked with a fine explorer. If all margins are smooth a final layer of unfilled resin can be placed over the defect to add further smoothness and fill in any microcracks created by polymerization shrinkage.

Step by step:
1. Etch the dentin and enamel with 37% phosphoric acid for 15 seconds,
2. Rinse well
3. Dry with a cotton pellet to remove any pooled water or gently air dry dentin
4. Apply resin (or primer if multiple component system),
5. Completely but gently dry
6. Light-initiate for time depending on light-cured lamp and type of bonding system
7. Place a first layer (GI or flowable composite)

8. Light cure
9. Place restorative composite
10. Light initiate for 30 to 40 seconds,
11. Contour and finish with finishing discs,
12. Acid etch for 10 seconds, rinse, and dry,
13. Place a final film of unfilled resin to seal any margins that may have opened due to polymerization shrinkage

Follow up: The patient can eat and drink normally following the restoration. Recheck dental radiographs are strongly recommended in 6-9 months.

The goal of restorative therapy:

1. Reduce sensitivity in vital teeth
2. Block off the pathway for bacterial infection
3. Smooth the surface of the teeth to reduce periodontal disease
4. Strenghten weakened hard dental structures to improve their functionality and resistance.

Common restorative options include:

1. Bonded sealants
2. Composite restorations
3. Glass ionomer restorations
4. Crowns
5. Implants

Restoration following endodontic treatment

Restoration of an endodontically treated tooth is performed following obturation of the cleaned and shaped root canal. The quality of the restorative dentistry performed after root-canal treatment directly impacts the prognosis of the endodontically treated tooth.[1] Two types of final restorations, direct intracoronal restoration and prosthodontic crown restoration, are commonly used in veterinary patients following endodontic treatment.

Intracoronal Restoration

In veterinary patients, endodontic access sites will typically result in Class I defects (occlusal surface) for the premolars and molar teeth, and Class V defects (mesiobuccal surfaces) for the canine teeth. Common fracture configurations involving the canines or carnassial teeth may result in Class VI defects of the incisal edge or cusp. Pulp cavity openings at any fracture site, as well as each endodontic access sites, must be restored to complete the closure of the root canal system. As a

general rule, endodontic access sites should be limited as much as possible. Adequate access for root canal instrumentation is a must, but there is no replacement for healthy tooth structure. The techniques outlined below represent an example used by this author of a modified preparation and a placement of bonded composite restoration using the "total etch" technique with 5th generation bonding agent. The specific methods and times stated may vary depending on the individual materials used. Manufacturer's directions should be strictly followed to ensure proper bonding and setting of the restorative materials.
Any unsupported enamel should be removed using a high-speed handpiece and appropriate sized round carbide or diamond bur. Any unnecessary enlargement to the site during preparation should be avoided. To allow for a better resin adaptation, all internal line angles should be rounded and cavity walls should be smooth. The cavosurface margins on occlusal surfaces should not be beveled. Any beveling should be restricted to the gingival and proximal margins where enamel is present.

Use of a Liner or Base An initial restorative layer of low viscosity material such as glass ionomer, resin-modified glass ionomer or flowable composite may be placed in the deep part of the preparation. The flowable nature of these materials will aid in adaption of the restoration to the deep confines of the preparation. If a eugenol-based sealer was used for obturation, a glass ionomer can be placed over gutta percha to prevent any remaining eugenol from affecting the composite setting. Flowable composites may also be used as a base/liner if the dentin is properly cleaned of any excess sealer during site preparation[11] or if a non-eugenol root canal sealer was used. Because the final restoration of bonded composite is retentive and strong, the use of a base liner may not always be necessary.[8] If a glass ionomer is placed, the manufacturer's directions should be followed to ensure proper placement and setting of the material. The following steps describe the use of flowable composite for an initial base layer.

The remaining part of the restoration of endodontic access is just as for restoration of any cavity.

Further Reading

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HOW TO MANAGE AVIAN EMERGENCIES

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Most birds presented to a veterinary clinic are prey animals and so will hide signs of their problem until disease is advanced. Deterioration can be rapid and any sick bird should be considered as a potential emergency. Initial history-taking should usually be kept brief and concentrate on key predisposing factors such as toxin exposure, presence of any new birds, a history of trauma and any environmental or dietary changes. Initial clinical examination may also need to be staged in particularly stressed or sick individuals. Pre-oxygenation is advised and examining in a quiet dim space may help to reduce stress.

Most avian emergencies will require stabilisation in hospital and can this can be broken down into five key steps in addition to treatment of any specific problems such as wounds.

1. Oxygen – ideally piped into the bird's own enclosure if this can be placed within an incubator or similar to reduce stress
2. Warmth – ideal temperature will depend on species but most small birds will benefit from extra warmth (e.g. in an incubator) so that they do not need to expend energy keeping warm when already compromised
3. Fluids – these can be provided via the oral, subcutaneous, intravenous (IV) or intraosseous (IO) route. Hydration status can be difficult to ascertain on clinical exam but dehydration should be assumed following any period of anorexia. In particularly critical patients, placement of an IV or IO cannula is recommended. For the IV route, the right jugular vein, basilic vein or medial metatarsal vein can be used. For the IO route, the proximal tiobotarsus or distal ulna are recommended. Sedation or anaesthesia are usually required unless the patient is collapsed in which case, local anaesthesia should be used.
4. Nutrition – ideally the normal diet should be offered but most sick birds will be anorexic and require supplementary feeding with a recovery formula (herbivore / omnivore / carnivore depending on species). Crop tubing usually needs to be performed multiple times per day.
5. Analgesia – assessment of pain can be very difficult in the avian patient so analgesia should be administered for any potential painful condition. NSAIDs appear to work well in birds but should be used with caution in

dehydrated individuals. Opioids can also be effective, with many species of birds having more kappa receptors than μ receptors so butorphanol is recommended. Other analgesics can also be considered for potential longer term use.

After initial stabilisation has been started, diagnostics should be considered. Most diagnostics need to be performed under sedation or general anaesthesia which can carry high risks in critical avian patients, so it is important to prioritise which diagnostics are going to change treatment at this stage. Bloodwork (for haematology and biochemistry) and radiography usually provide the most information for emergency decision making. Even if full blood results cannot be analysed in-house, PCV, assessment of a blood smear, checking blood glucose and ionised calcium can significantly change treatment plans. Coelomic ultrasound can also be helpful for cases with potential ascites. Following initial diagnostics, treatment can then be tailored to the specific case for the best possible outcome. Ultimately a fast response and intensive approach to stabilisation will be associated with the best outcome for the avian patient.

TOP 5 TORTOISE AND TURTLE PRESENTATIONS

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Tortoises and turtles may present to the veterinary clinic for many different reasons, but the most common problems recorded are often those which can be easily visualised by the owner. Overgrown beaks, nails and shell abnormalities are particularly common presentations in tortoises. Shell abnormalities are also common in aquatic species with ocular and respiratory problems also frequently reported. A selection of these disorders will be discussed, specifically;

1. Beak and nail problems – predominantly seen in tortoises, overgrown beaks and nails are thought to occur for a variety of reasons. In the case of overgrown beaks, this can limit the animals' ability to prehend food. Nutritional factors, congenital disorders, trauma and hepatic disease have all been suggested as possible causes, alongside lack of exercise and inappropriate substrate for overgrown nails. Overgrowths can be managed by regular trimming with a burr or nail file but identification of the underlying cause is critical to improve welfare.

2. Shell trauma – seen for a variety of reasons including predator attack, fall from a height, vehicle trauma or burns. On presentation, it is important to establish the depth of wounds and extent of underlying structures involved. Initial stabilisation involves warmth, fluid therapy and analgesia. Wounds then require careful flushing and bandaging to clean and protect from further damage. As most shell injuries are already contaminated, antibiotic treatment is usually necessary. Imaging may be helpful especially if considering surgical debridement for more extensive or displaced injuries.

3. Ocular disorders – specifically blepharoconjunctivitis secondary to hypovitaminosis A is the most common ocular disorder seen in turtles. Nutritional deficiencies are common in captivity especially if selective feeding is allowed and long-term lack of sufficient vitamin A will result in squamous metaplasia of orbital glands and their ducts. Opportunistic bacterial infections can then flourish especially if water quality is poor. The subsequent periocular swelling generally results in pain and anorexia so worsening the original problem. Diagnosis is often presumptive based on husbandry and diet history. Treatment is mostly supportive with anti-inflammatories, gentle flushing, analgesia, nutritional support, antibiotics

if indicated and vitamin A supplementation. Tortoises in contrast, usually present with infectious blepharoconjunctivitis secondary to infectious processes such as mycoplasmosis. Sampling and diagnosis at an early stage is vital to guide effective treatment and warn owners about prognosis.

4. Respiratory disorders – signs of respiratory disease can be very subtle in both tortoises and turtles until disease is advanced. Sometimes buoyancy issues may be reported in a turtle but this is generally at the point of lung consolidation. Pneumonia usually occurs secondary to husbandry deficits and subsequent overgrowth of commensals. Stomatitis and upper respiratory tract disease may also be associated. In tortoises, pathogens such as herpesvirus, mycoplasma and picornavirus should be key differentials for upper respiratory signs. Imaging is vital to establish the extent of any lung changes with advanced imaging providing much clearer detail if available. Once location of the problem has been established, sampling can then be performed +/- endoscopy if required. Treatment is usually prolonged and involves antibiotics (based on culture if possible), NSAIDs, nebulisation and treatment of any concurrent disease in addition to correction of any predisposing factors.
5. Anorexia – a common presentation but a very non-specific sign in both tortoises and turtles. Significance may vary depending on time of year especially if associated with brumation or aestivation. However if prolonged and not part of a typical seasonal cycle, supportive treatment and investigations are recommended. Initial treatment is likely to involve warming (if required), fluid therapy and then nutritional support once rehydrated. Diagnostic approach will depend on the presence of any more specific clinical signs but usually involves bloodwork and imaging. Return to a normal appetite can be slow in many cases and owner's expectations should be managed accordingly.