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CPD Article

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Editor's Note

We are already teetering on halfway through this year. It seems to have just galloped past. I hope everyone is well and safe.

This edition has three homegrown authors - myself writing out of my field (apologies Gerhard) but on something we see often in general practice. Dr Kelly du Preez has recently obtained her MMeVet in clinical pathology and works together with the Clinical pathology section at the Faculty. She is writing on a new measurement available to us veterinarians from our laboratories to diagnose iron-deficient decrease in erythropoiesis before it becomes full-blown anaemias. The last claim to homegrown authors is Dr Liza S. Köster, who also obtained her basic and specialist qualification at the faculty at Onderstepoort. It is very rewarding to see people following and achieving their dreams. Liza is now a cardiologist at Tennessee Veterinary Hospital.

In the previous edition I made an offer that I would take emails involving issues or questions which I would then hand over to the specialist best able to answer them. I have received no questions !? Please feel free to send in questions - generally, if one person has a query or a grey area, then others also do and the question will help many others. This Question/Answer page will, I hope, become a staple of the magazine.

Liesel

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Advisory Board

VET360 aims to be a leader in the field of continuing veterinary development in Southern Africa by providing veterinary professionals from diverse disciplines with tools to help them meet the challenges of private practice. The magazine aims to make information accessible, both paper and electronic, and provide clinical, business and other veterinary information in a concise form to enable the practitioner to rapidly acquire nuggets of essential knowledge.

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How & Why Technology Can Become Toxic & What You Can Do About It

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"Today with the advent of digital, we're even more connected than previous generations... can we really afford to not unplug?" - Eric D. Garcia

We've all been there before. Maybe you promise yourself that this is the last email of the night, or that you won't be logging back into Facebook until it's really important.

Whether it's for work or play, we find more and more that it's all too easy to be consumed in the digital world around us. Sure, new advents like smartphones have boosted productivity and given us a convenience never before possible. The world is literally at our fingertips, and that level of seamless tech integration into our daily lives is nothing short of miraculous.

Still, if we start to spend more time in that world, than we do in our own physical, that's when we start to run into problems...

Work might start to lack the same meaning it once had, and even important relationships might start to feel ordinary and unengaging. What's hardest to pinpoint (and often eludes even veterinarians and industry specialists alike) is distinguishing when the use of technology is actually being used for more harm or good.

The reality is, the world of non-stop news and ongoing social media activity creates a constant noise; a buzz that serves as an undercurrent to world events and social interactions at every level. This type of noise, compounded over time, can start to exhaust us and wear our energy down to the point of total burnout, while creating a level of stress that can actually be detrimental to overall health.

Stress is linked to a variety of severe conditions, from heart disease to asthma, depression, accelerated aging and more. When stress inhibits your sleep (like it used to impede mine and from time to time still does) this can damage your body's ability to recharge, heal, build antibodies and perform optimally. This

can eventually lead to an increased likelihood of developing dementia and even Alzheimer's. Side note: Arianna Huffington's book "The Sleep Revolution" will scare you into getting more sleep: https://www.amazon.com/Sleep-Revolution-Transforming-Your-Night/dp/1101904003 .

So, it's important to be mindful, and even vigilant, of the power that technology wields. Even though new technology is an incredible thing, it can actually catalyze a great deal of harm when misused.

That's why it's more important now than ever, to consider the benefits of carving time away from technology and understanding the rewards that this sort of practice can yield. I recently spoke with dvm360 about the benefits of unplugging, which entails disconnecting from all technology in a methodical way, in order to enhance your physical and mental well-being.

In this article, and in past posts, I have shared tips for an extended unplug, which can last two weeks (or more) and promotes a total disconnect from your smartphone or other technology you typically engage with frequently.

Today, I'll share tips for #UNPLUGGEDMoments, which instead allows you to carve out the time you need to recharge, anywhere and anyhow you see fit! This can be particularly empowering if you don't have the time or resources to take an extended getaway or leave your home for a full-on vacation. Instead, these moments are there whenever you choose to seize them. It can be an afternoon spent in the park with your phone on silent, or taking a few hours in the morning for breakfast and exercise before turning your phone on.

These moments can help to hold you over during particularly stressful times, and you can get better at identifying when they'll be most beneficial over time. For example, if you've started to lose sleep over work, are especially agitated or otherwise anxious, it may be the perfect time for your next #UNPLUGGEDMoment.

So, go ahead, steal the moment!

These tips will help you to make the most of the unplugged moment that's right in front of you:

1 Decide what rules are right for your unplugged moment. On my unplugged moments, I prohibit phone calls, text, email and social media. If there's an exception to the rule (like an emergency phone contact, which I do recommend) make a note of it and follow accordingly.

2 Decide what you want most out of the experience. If you've been feeling distant from a friend or family member, you may decide to invite them to a dinner where you both leave your phones at home. You may just find that your interactions are more focused and meaningful without the extra distraction. If they're confused about why leaving their phone behind is important – you might want to share this article with them.

3 Stay a step ahead...of yourself. If you've decided to unplug for the weekend, try turning your home computer off. Maybe even take the chord out of the wall (a literal unplug gets extra points). Out of sheer habit, you may head over to the computer to turn it on, but by making sure it's actually disconnected from the power, it will stay off and serve as a buffer against your muscle memory.

4 The same goes for smartphones. I move all of my most impulsive apps to a less accessible screen, so that if I get the sudden urge to open Facebook out of impulse, the icon isn't where I usually find it. This serves as a reminder to myself, "I've hid my apps for a reason."

5 Get creative! It can be tempting to wait to unplug, searching for the perfect vacation or sometime in the future when everything aligns just right. Those extended moments can be hard to come by, so plan an hour, or a day, depending on what's best for you and realistic. You can decide to walk to a new part of town with your phone turned off, so you can enjoy a lunch or coffee in peace and quiet. You might spend a whole weekend camping, which is also an amazing way to connect with the world around you, without depending on technology.

When you unplug properly, whether for a moment or for a month, you find that something incredible happens. Not only do your senses come alive (food tastes richer, colors are more vivid), but your sense of curiosity and joy starts to grow too.

Life is no longer about getting back to work, but it's instead about loving what you're doing, whatever that may be in the moment...

About the Author:

Eric Garcia is an IT expert. Digital marketer. Industry thought leader. When it comes to helping veterinary practices streamline their technology and attract and retain clients, Eric Garcia has a proven track record of educating the industry and producing results. Eric is an IT and Digital Marketing consultant working exclusively with veterinary practices. In addition to a long list of satisfied clients, Garcia's work has been recognized throughout the industry. He speaks regularly at conferences all throughout the world. Facebook: facebook.com/EricGarciaFL Twitter: @EricGarciaFL Instagram: @EricGarciaFL

Reticulocytes and the Value of Reticulocyte Haemoglobin as a Marker of Iron Deficient Erythropoiesis

Reticulocyte haemoglobin is measured as part of a routine complete blood count (CBC) by a number of haematology analysers used in South Africa, making it widely available.

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Reticulocytes are immature erythrocytes that are produced in the bone marrow through the process of erythropoiesis. In health, low numbers of reticulocytes are continuously produced to replace aged erythrocytes. When blood loss or haemolysis results in anaemia, increased numbers of reticulocytes are produced and released in order to restore the red blood cell mass.[1]

Historically, we only evaluate reticulocyte percentages and absolute counts when anaemia is present to determine whether there is erythrocyte regeneration. In other words, to determine if the bone marrow is able to respond and restore the red blood cell mass by increasing reticulocyte output. Using the absolute reticulocyte count (ARC), anaemia may be classified as regenerative or non-regenerative.[1]

The ARC is usually reported with the reticulocyte percentage and is calculated using the following formula:[1]

ARC (cells x 10^9/L) = reticulocyte percentage (%) x red blood cell count (cells x10^{12}/L) x 10

Each reference laboratory or in-clinic analyser will use a different cut-off value to distinguish between regenerative and non-regenerative anaemia but as a rule of thumb the following values may be used:[2]

Dogs: ARC <95 x 10^9/L is non-regenerative
Cats: ARC <60 x 10^9/L is non-regenerative

Anaemia may be also be classified as "pre-regenerative". Because it takes 3 – 4 days for reticulocytes to be produced from immature precursor cells in the bone marrow, reticulocytosis may not be evident for a few days after anaemia develops.[1] The classification of pre-regenerative anaemia is reserved for anaemic patients in transition from a non-regenerative towards a regenerative state. Thus, by using the reticulocyte percentage and ARC, we measure the quantity of regenerative response.[3]

But how do we measure the quality of regeneration? Recently, reticulocyte haemoglobin content has been investigated as a means of measuring the quality of the reticulocytes that are produced. Iron is vitally important for the production of haemoglobin and is preferentially used for erythropoiesis.[4] When insufficient iron is available for erythropoiesis, less haemoglobin in produced resulting in reticulocytes with a lower haemoglobin content and smaller cell volumes.[5] This condition is known as iron deficient erythropoiesis or IDE (also iron restricted or limited erythropoiesis).

Iron deficient erythropoiesis (IDE) is a relatively common condition in dogs and cats, with a prevalence of 7 to 10.3%.[4, 6, 7] Disorders that may cause IDE are broadly categorised as true or functional iron deficiencies (Table 1).

- True iron deficiencies are characterised by depletion of total body iron resulting in inadequate iron stores in the bone marrow, spleen and liver.[8]
- Functional iron deficiencies are caused by sequestration of iron and/or inadequate iron mobilisation from adequate or increased iron stores.[8]

In addition to decreased reticulocyte haemoglobin content, IDE may also cause a decrease in the mean cell volume (MCV) and mean corpuscular haemoglobin concentration (MCHC) i.e. microcytosis and hypochromasia.[3, 4] These markers however, have limitations:

- MCV and MCHC are mean values and a significant proportion of erythrocytes must be affected before a decrease below the reference interval is seen.[5]
- Considering the lifespan of erythrocytes (100-120 days in dogs; 70 days in cats), a decrease in MCV or MCHC is likely to occur weeks after an iron deficiency is experienced and acute and sub-acute IDE will go undetected.[3, 5]
- Microcytosis and hypochromasia have also been described in a proportion of healthy dogs from certain breeds such as the Chow Chow, Chinese Shar-Pei, Akita and Shiba Inu.[4, 9]
- Decreased MCV and/or MCHC is uncommon in anaemia of inflammatory disease, unless it is prolonged (usually manifests as a normochromic, normocytic anaemia).[3, 10]

These limitations reflect the insensitivity of MCV and MCHC for the detection of IDE.[4]

Similar to MCV and MCHC, serum iron is an insensitive and non-specific marker of iron status as it is influenced by a number of factors including time of day, corticosteroid administration, and diet.[4,5] Additionally, serum iron is a negative acute phase reactant and will decrease with acute inflammation in a matter of hours.[4, 6]

In an attempt to overcome the limitations of MCV, MCHC and serum iron concentration, researchers focused on assessing the characteristics of the reticulocytes, rather than the entire red cell mass.

The advantages of using reticulocyte haemoglobin content as a marker for IDE are numerous:

- Aggregate reticulocytes only circulate for 1 – 2 days in dogs and cats, making their haemoglobin content a recent picture of the iron availability for erythropoiesis.[4, 6, 10]
- Conditions, apart from IDE, which decrease reticulocyte haemoglobin content in humans, such as thalassaemia, have not been reported in dogs and only rarely reported in cats.[4, 11]
- Reticulocyte haemoglobin content is measured as part of a routine complete blood count (CBC) by a number of haematology analysers used in South Africa, making it widely available. These analysers include the ADVIA 120/2120 (measured as CHr), Sysmex XT-2000iV (measured as RET-He), and ProCyte Dx haematology analysers (measured as RET-He or RETIC-HGB). [4, 6, 7]
- Reticulocyte haemoglobin is stable for up to 24 hours in canine and feline blood stored at room temperature.[6, 7]

Studies on reticulocyte haemoglobin were initially focused on confirming its value as a marker of IDE in dogs with proven iron deficiencies. These investigations showed that reticulocyte haemoglobin content decreased before the classical markers of IDE (microcytosis and hypochromasia) and before the dogs became anaemic.[4, 6] In fact, 25 – 74% of all the dogs with low reticulocyte haemoglobin had no microcytosis or hypochromasia (i.e. MCV and/or MCHC within the reference interval).[4] Furthermore, serum iron was decreased in almost all the adult dogs, regardless of whether they had IDE or not.[4]

Adult dogs with possible functional iron deficiencies were also investigated. Dogs with systemic inflammation were identified using serum C-reactive protein (CRP), a positive acute phase protein.[10] Increased serum CRP was associated with low reticulocyte haemoglobin content but also with increased serum ferritin, a protein used to store iron.[10] Increased ferritin implies that there are increased body iron stores and a decreased iron availability secondary to sequestration within storage pools.[6, 10]

Table 1 – True and functional iron deficiencies: causes and pathomechanisms[3, 5, 6, 8]

	Cause	Pathomechanism
True iron deficiency	Chronic blood loss	Increased iron loss
	Nutritional deficiency (rare)	Inadequate iron intake or absorption; may occur with vegetarian/homemade diets or malabsorption
Functional iron deficiency	Anaemia of inflammatory disease	Iron sequestration in macrophages, entero-cytes and hepatocytes due to pro-inflam-matory cytokines
	Hepatic disease (including portosystemic shunts)	Compromised production of iron metabo-lism proteins
	Marked erythropoietic stimulus (e.g. immune mediated haemolytic anaemia; erythropoie-tin administration)	Imbalance between excessive iron demand and slow iron mobilisation

Based on the previous investigations, the ability of reticulocyte haemoglobin content to differentiate between the causes of IDE was questioned.[3, 5] Can we differentiate between true and functional iron deficiency using reticulocyte haemoglobin content? In short, not reliably.

Two studies compared adult dogs with evidence of chronic blood loss to adult dogs with portosystemic shunts, inflammation or breed associated microcytosis.[3, 5] Regardless of the cause of IDE, reticulocyte haemoglobin content was decreased.[3, 5] There was also a large overlap in the reticulocyte haemoglobin results between the groups.[3, 5] Consequently, no defined cut-off can be used to differentiate between these disease processes.[5]

However, some useful patterns and cut-offs have been found:

- Low reticulocyte haemoglobin content is identified as a RET-He <20.9 pg or CHr <22.3 pg.[4,6]
- Dogs with anaemia of inflammatory disease did not usually have a haematocrit below 20%.[5]
- Dogs with microcytosis and low reticulocyte haemoglobin secondary to portosystemic shunts or breed-associated microcytosis were not usually anaemic.[5]
- Dogs with true iron deficiencies had the lowest reticulocyte haemoglobin values.[5]

Only two studies have investigated reticulocyte haemoglobin in cats. Healthy adult cats have a much a lower reticulocyte haemoglobin content than dogs and different cut-off values for the diagnosis of IDE must be used.[7,12] There is also a large degree of overlap between cats with IDE and those without, making this marker much less valuable in cats.[7] When applying a cut-off value of <12.5 pg, there were a very high number false negatives but a very low number of false positives.[7] Consequently, reticulocyte haemoglobin content of <12.5 pg can confirm the presence of IDE in cats but even if the reticulocyte haemoglobin content is >12.5 pg, IDE cannot be excluded.

Using reticulocyte haemoglobin content as a marker for IDE does, however, have limitations. An important limitation is, as mentioned earlier, the inability to reliably differentiate between true and functional iron deficiencies. Another limitation to bear in mind pertains specifically to reticulocyte haemoglobin content as measured by the Sysmex XT-2000IV and ProCyte Dx (measured as RET-He). Due to the method used by these analysers, macrocytosis may result in a false increase.[6] In cases where the RET-He is only mildly decreased, this false increase may push RET-He into the reference interval (i.e. a false negative) and the diagnosis of IDE may be missed.[6, 7] Therefore, in cases of macrocytosis, RET-He must be critically assessed and the result interpreted with caution. These cases are fortunately uncommon as IDE usually results in a blunted regenerative or non-regenerative response.[5]

So what to do if the reticulocyte haemoglobin content is below the cut-off value?

Diagnostic investigation must be focused on determining whether a true and/or functional iron deficiency is present. A thorough clinical examination, minimum database (including urine and faecal analyses), routine haematology and routine clinical chemistry will go a long way in providing valuable information.

True iron deficiencies are most commonly caused by chronic external blood loss.[13] The GIT, urogenital tract and skin or mucosal surfaces should thus be investigated for a source of bleeding such as verminosis, bleeding neoplasm or gastric ulceration.

If a true iron deficiency is excluded, causes of functional iron deficiencies should be investigated. This may include testing hepatic function (e.g. pre- and post-prandial bile acid concentrations) and blood smear evaluation and Coomb's test to rule in/out immune mediate haemolytic anaemia. Anaemia of inflammatory disease may be assumed if other causes of iron deficient erythropoiesis are excluded.

Take-home message

AID is the most common cause of anaemia in dogs and cats and may be caused by any chronic inflammatory (e.g. neoplasia, chronic infection).[13]

Reticulocyte haemoglobin is a useful marker for the detection of IDE, specifically in adult dogs, and is superior to the traditional markers of IDE, namely microcytosis (as measured by MCV), hypochromasia (as measured by MCHC) and haematocrit.

Using a RET-He cut-off value of <20.9 pg (Sysmex XT-2000iV and ProCyte Dx haematology analysers) or a CHr cut-off value of <22.3 pg (ADVIA 120/2120 haematology analysers), IDE may be accurately identified in most cases.

That said, reticulocyte haemoglobin should not be used as a sole means of diagnosis as it is not reliable in differentiating true iron deficiencies (most commonly chronic blood loss) from functional iron deficiencies (most commonly anaemia of inflammatory disease). Using the appropriate cut-off values, reticulocyte haemoglobin is reliable for the detection of IDE and a low value should prompt further investigation as to its cause.

References available online: www.vetlink.co.za

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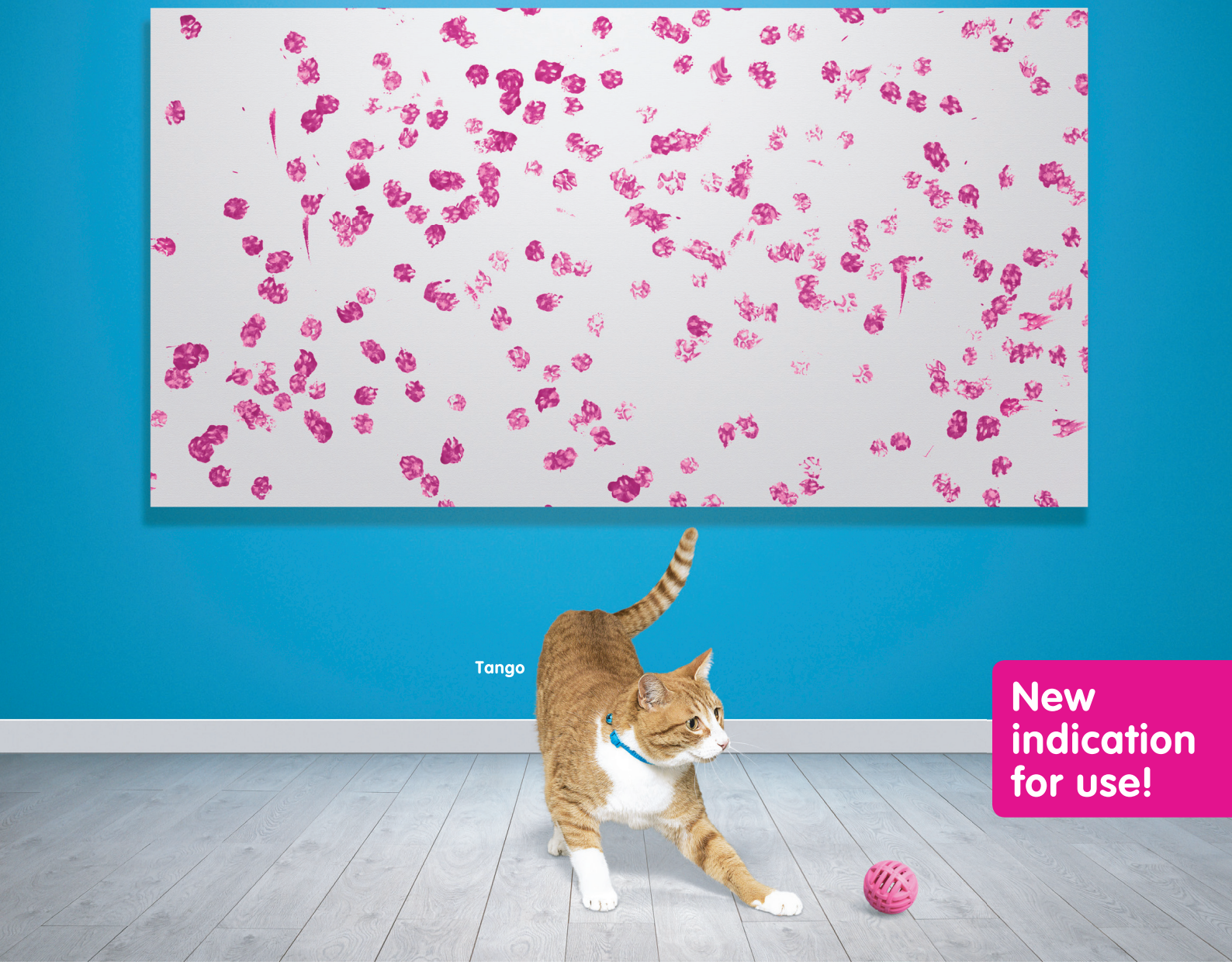

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Feline Stomatitis

From just a bit of halitosis to pain and anorexia, gingival inflammation in cats can have a huge impact on their quality of life.

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There are two forms of stomatitis in cats - the well described lymphocytic- plasmacytic gingivitis/ stomatitis (LPS) and a more recently described juvenile gingivitis. This article aims to summarise the two syndromes and discuss the treatment options available.

Juvenile gingivitis

Feline juvenile gingivitis is a severe generalised inflammation of the gingiva occurring just after the permanent teeth have erupted. It is generally non-painful but causes quite a severe halitosis. Examination of the oral cavity will show inflammation of the gingiva with varying degrees of hyperplasia and little or no tartar accumulation. The gingiva is friable and may bleed during eating and oral examination. In some cases gingival proliferation may develop into pockets. The exact cause is unknown but factors such as inflammation during permanent teeth eruption, viral infection, immune system hypersensitivity to plaque, bacterial infection and a genetic predisposition are postulated.

Cats generally present before one year of age. Certain breeds such as Maine Coone, Somali , Persian and Siamese cats are predisposed. The immune response will generally resolve once the cat reaches approximately 2 years of age, however by this time most of the patients will have required tooth extractions due to the development of periodontal disease prediposed to by the inflammation and gingival pockets..

Treatment has of this condition has main two goals: keeping the mouth clean and managing the immune response.

Daily tooth brushing and oral rinsing is the mainstay of home treatment. This is often combined with dental scaling and polishing every 3-6 months. During the dental procedure hyperplastic gingiva is resected to remove pseudopockets which promote plaque formation and thus increase inflammation. If teeth are loose elective extractions are advised to limit periodontal inflammation. Patients should also be evaluated for tooth resorption (TR) using dental radiographs as inflammation can trigger TR.

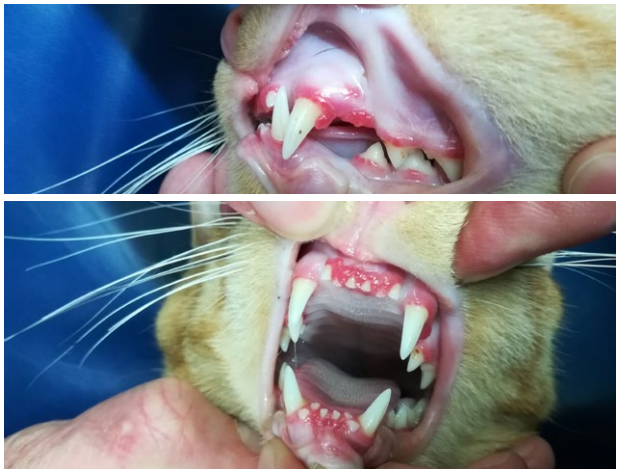

Fig. 1 - A 10-month-old DSH presented for castration with a complaint of halitosis. Juvenile gingivitis was diagnosed. (Images courtesy of final year student, Chante Kritzinger).

Inflammation should be controlled with NSAIDS. The use of corticosteroids, after an initial short period of improvement, will worsen the disease in the long term. The NSAIDS will also help reduce the discomfort from periodontal disease. discomfort and manage anorexia. Opiods or gabapentin may be required in some cases. Antibiotics are generally not required.

Due to the inability for most cat owners to manage the oral care required full mouth extraction is the curative treatment in these cases. However due to the young age of the cats, most vets and clients will try conservative treatment first with an initial dental and gingivoplasty and re-evaluation.

Lymphocytic Plasmacytic Stomatitis (LPS)

Lymphocytic plasmacytic stomatitis is a very painful condition affecting middle aged to older cats. These patients present with weight loss and are anorexic. They often drool, are unable to chew food, drop food from the mouth and drool when drinking water. They

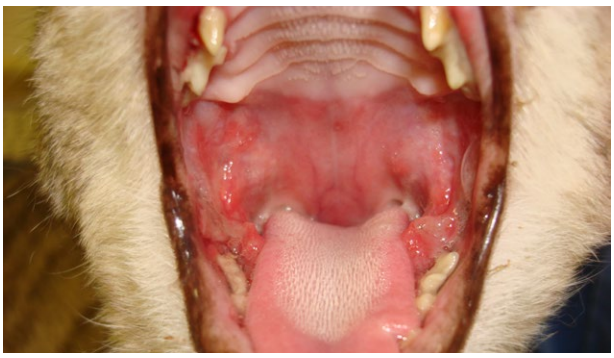

Fig. 2a - Moderate to mild lymphocytic plasmacytic stomatitis in a cat. (Image courtesy of Dr Gerhard Steenkamp).

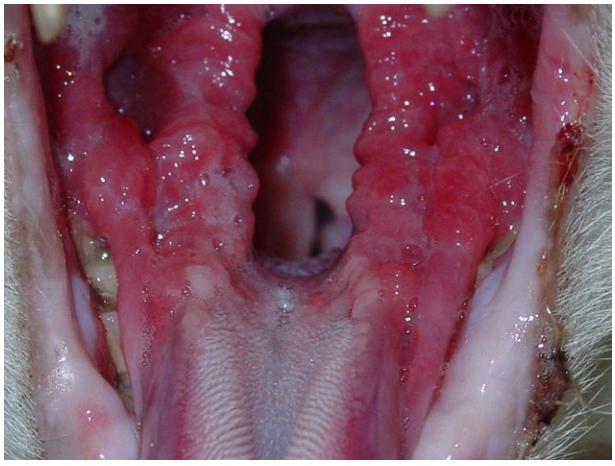

Fig. 2b - Severe lymphocytic plasmacytic stomatitis in a cat . Note how the caudal faeces and gums are preferentially affected. (Image courtesy of Dr Gerhard Steenkamp).

also run away from the food or water as they develop food aversion anorexic and drooling. They also often have a matted coat as it is too painful to groom.

Adequate examination of the oral cavity of these cats often requires heavy sedation or anaesthesia. A tentative diagnosis of LPS should be made when the severity of the gingivitis is excessive, rather than just severe peridontal disease, when compared to the amount of calculus and there is a significant amount of proliferative tissue and/or the inflammation extending to caudal areas of the mouth and fauces where no teeth are present. It is an inflammation of the oral mucousa and not just of the tissues surrounding the teeth. The lesions are usually bilateral. (Fig. 2)

Differentiation of LPS from other feline dental disease is important. LPS is a symmetrical condition. Any proliferative unilateral lesion on the fauces, base of the tongue, palate and in the tonsillar region requires a biopsy to rule out neoplasia and eosinophilic granuloma complex. Squamous cell carcinoma can occur in the fauces and base of the tongue. (Fig. 3)

Antigens postulated in the genesis of LPS include plaque bacteria, other bacteria, feline calici virus, food antigens and autoantigens (dental / periodontal tissues). The majority of cats with LPS have positive

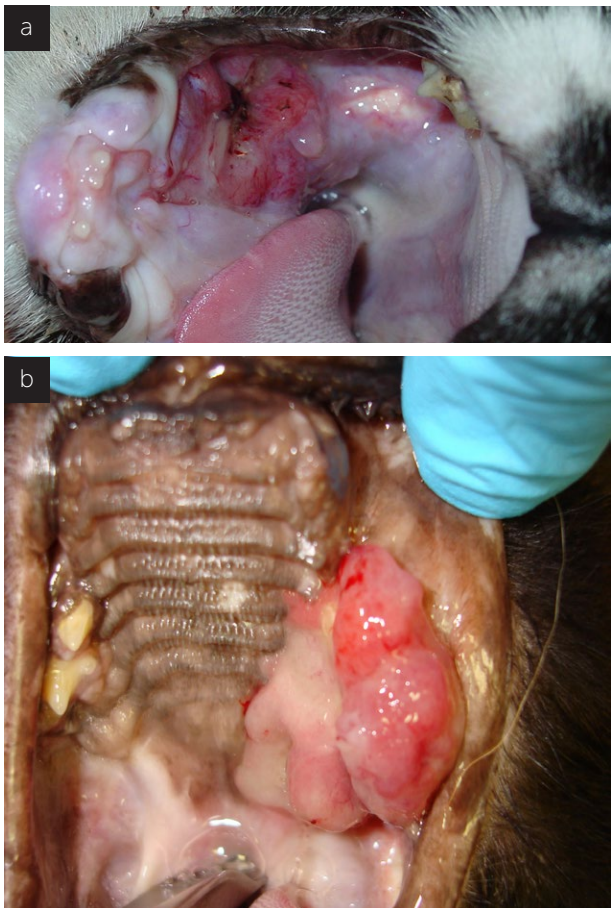

Fig. 3 - Oral squamous cell carcinoma as presented in 2 cats. The proliferative inflammatory reaction is similar to a severe LPS, except that it is unilateral whereas LPS is always bilateral. (Images courtesy of Dr Gerhard Steenkamp).

calicivirus swabs, but then many other cats with calicivirus infection do not develop LPS. Cats with LPS also have a less diverse oral bacterial commensal population and increased prevalence of *Pasteurella multocida* sub species in cats with LPS has been demonstrated. Immunocompromised patients (FIV, FeLV, diabetes mellitus) are more predisposed to gingivostomatitis - this may be due to an impaired ability to control bacteria or viruses in the oral tissues or due to impaired immunoregulation. Lymphocytic plasmacytic stomatitis is associated with a high likelihood of FIV and FeLV positivity.

Due to the immune mediated reaction to plaque or the tooth itself, these cats will generally have a hyperglobulinaemia due to hypergammaglobulinaemia. In some cases, there is even a monoclonal gammaglobulinaemia due to the immunologic reaction and lymphoid proliferation. The diagnosis can be confirmed with a deep biopsy of the lesion - making sure you don't just get superficial secondarily infected parts of the affected tissue. Biopsy will not however determine the cause of the chronic immune mediated response.

Most LPS cases are only temporarily responsive to medical management.

Corticosteroid is the main form of therapy in these cases to initially manage the condition. Oral administration is preferred to depot injectable formulations, but in many cases the mouth is too painful for the owners to dose and treatment is initiated with a depot formulation (methylprednisolone 10-20 mg/cat). If oral treatment is possible then prednisolone (remember cats cannot effectively utilise prednisone) at 2-4 mg/kg/day, tapering as the condition improves. The owners can also start using oral prednisolone when the effect of the injectable prednisolone is expected to start waning. If clinical response is poor, chlorambucil at 1 mg po q24 or 2 mg po q48 can also be tried.

Antibiotics to control oral bacteria are recommended. Doxycycline is concentrated in saliva and in the inflammatory fluid produced by the gingiva. It has antibacterial as well as anti-inflammatory properties.
Pain control is essential in these patients. The corticosteroid treatment will naturally have some effect but in certain patients additional pain control can be given using Tramadol, fentanyl patches or transmucousal buprenorphine.

Changing to a hypoallergenic diet has been recommended, but there is no proven effect and supplementation with additional omega 3 and 6 supplementation did not reduce the inflammation in these cats. Bovine lactoferrin oral spray has shown benefit by reducing the immune reaction and inhibiting calicivirus proliferation/infection into healthy cells. None of the combination of medical interventions had any lasting effect on the improvement of the condition.

The most successful treatment is complete dental extraction of most or all teeth and the earlier this is performed the better the long term outcome in the cat. If, in some cases where just molars are affected, only the molars are extracted, the remaining teeth will require a daily oral hygiene regimen. As this is not an easy task full mouth extraction is usually the way to go. No tooth roots must remain, so pre and post-operative dental radiographs must be taken to identify all teeth, even those with feline odontoclastic resorptive lesion (FORL) and then to make sure no roots accidentally remain as this will cause ongoing inflammation.

Due to the development of food aversion some cats on either medical or surgical management may require tube feeding for a period of time. An oesophagostomy tube is indicated in these cases.

The majority of cats (90%) will require no further medication after a full mouth extraction. Slight inflammation may remain in the fauces in some cats but it doesn't cause discomfort. These cases with residual inflammation are also generally more responsive to medical management once the teeth are extracted.

Cats with longstanding inflammation or those which have received long term medical management prior to extraction seem to have a poorer prognosis even after full mouth extraction. So this is definitely a situation where you need to decide early on if the owner is willing for a FME and get on with it - for the best long term result.

Take home message

Juvenile stomatitis

- Begins at tooth eruption until approximately 2 years of age
- Usually non-painful. Severe halitosis
- Causes postulated include viral infection, immune system hypersensitivity to plaque, bacterial infection and a genetic predisposition
- Treatment is home brushing and dental scale and polish. NSAIDs may help. Corticosteroids and antibiotics don't help.
- Full mouth extraction is generally required

Lymphocytic Plasmacytic Stomatitis

- Middle aged to older cats
- Very painful, dysphagia present
- Symmetrical inflammation of the oral mucosa – most severe caudally.
- Postulated causes include bacteria, calici virus, FIV/FeLV and immune mediated,
- Full mouth dental extraction is usually required.
- Interim treatment is prednisolone, doxycycline (initally), pain control

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Classifying Tooth Resorption in Cats and Dogs

Amanda Carrozza

Understanding how tooth resorption is diagnosed and classified enables you to provide better patient care and to increase patient comfort.

Tooth resorption is a centuries-old disease that continues to puzzle the veterinary profession. Although its development may seem unpredictable at times, resorption is a common condition that veterinarians and technicians must understand to provide optimal care. At a recent Fetch dvm360® conference, Benita Altier, LVT, VTS (Dentistry), detailed the diagnosis and classification of tooth resorption in cats and dogs.

The word resorb, by definition, means to break down and assimilate (absorb) something. The tooth and alveolar bone are 2 distinct structures held together by periodontal ligament fibres. Tooth structures resorb via several mechanisms, progressing from internal or external locations on the tooth. Resorption can be inflammatory or non-inflammatory in nature.

Altier noted that the pathologic process involved in tooth resorption is not fully under-stood. Even without a concise understanding of genetic or environmental factors which make an animal susceptible to tooth resorption, researchers do have a clear image of what occurs once the disease is initiated. Classification systems have been established for cats and dogs to map the locations of lesions, the severity of disease, and treatment options.

Altier was adamant that the best—and often only—way to diagnose tooth resorption is through a comprehensive dental evaluation that includes intra-oral radiographs. She added that full-mouth dental radiographs should be obtained every time a pet is anaesthetised for dentistry as things can change rapidly from year to year.

"We cannot clinically evaluate a pet until it is under anaesthesia," she said, adding that the rates of pets anaesthetised annually for dentistry are extremely low. "Unfortunately, we know that not all pets presented to the veterinary hospital receive diagnosis and treatment, so many, many cats and dogs are suffering with tooth resorption left untreated and painful."

In addition to radiographs, Altier recommends that each tooth be examined with a dental explorer for signs of mobility, red gingival mounds, and broken crowns. From a strictly visual standpoint, tooth resorption may present as gingival redness focal to a single tooth. This is a red flag, she warned, and in these cases the client should be informed that tooth resorption is likely. However, you don't need direct evidence of resorption to anaesthetise a patient. "Just like we don't need evidence of disease to decide to recommend routine blood testing, in dentistry, we are screening for everything," she said.

Classifying tooth resorption

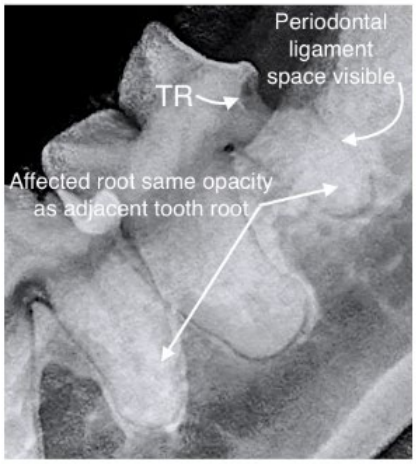
Cats

The overall incidence of feline tooth resorption varies widely among published studies, from 20% to 75%, Altier explained. Despite the wide range, she feels strongly that if you own 2 cats, it is very likely that 1 of them will develop tooth resorption.

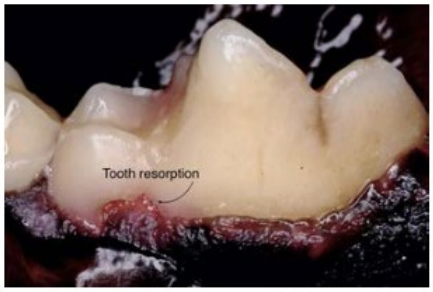
"It might seem like semantics to classify tooth resorption individually, but it is important," Altier noted. During a dental examination, veterinary professionals should record a diagnosis for each tooth based on clinical and radiographic evidence. When assessing a feline patient, both type and classification of the disease should be documented.[1] Treatment is dictated by the type of resorption present. Three types of tooth resorption are recognized in cats:

- **Type 1 (T1):** The tooth retains normal density and the periodontal ligament space is unchanged. Resorption tends to originate at the cementoenamel junction and will destroy the tooth in a coronal direction, an apical direction, or both.
- **Type 2 (T2):** Radiography will show some narrowing of the periodontal ligament space and decreased radiopacity. It is also common for the root structure to transform into a bone substance that has the same density as the adjacent bone.
- **Type 3 (T3):** T3 resorption occurs in multi-rooted teeth and presents as a combination of T1 and T2.

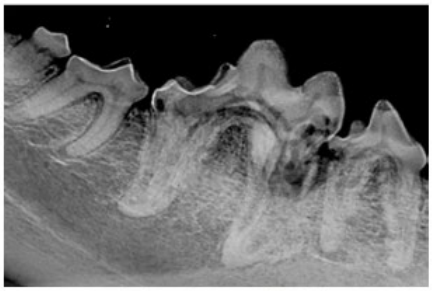
Stages indicate the progressive nature of the disease and also the location and extent of the lesions. Five

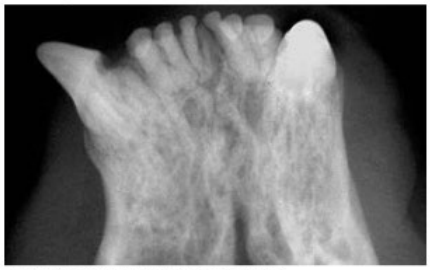


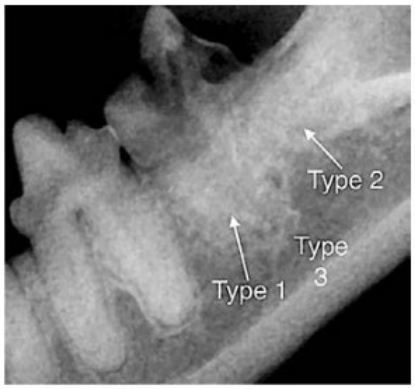
Type 1 tooth resorption in a cat.



Resorption of the right mandibular first molar in a dog.

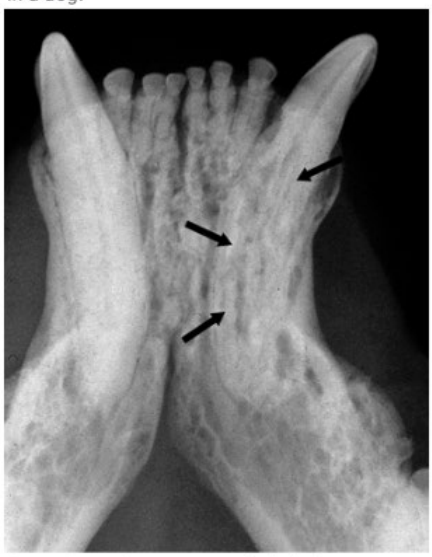

Resorption of the right mandibular first molar in a dog.


Type 2 tooth resorption in a cat.



Type 3 tooth resorption in a cat.


Stage 2 tooth resorption of the right mandibular canine in a cat.


Stage 4 tooth resorption of the left mandibular canine in a cat.

stages of tooth resorption are recognised in cats:

- **Stage 1 (TR1):** Stage 1 resorption presents with only mild clinical evidence of hard tissue loss and is rarely detected, especially if the lesion is hidden behind soft tissue.
- **Stage 2 (TR2):** Some radiographic evidence is detectable, such as a change in the dentine. Granulation tissue may be seen creeping into even a pinpoint-sized hole. The pulp is not affected, but the tooth is painful.
- **Stage 3 (TR3):** Lesions become recognisable. The pulp has been invaded and the tooth is extremely painful. The integrity of the tooth is intact, and the crown has not broken off.
- **Stage 4 (TR4):** Stage 4 is divided into 3 subcategories. In stage TR4a, the crown and root are equally affected, there is extensive damage to the cementoenamel pulp, and much of the tooth's integrity is lost. In TR4b, most of the damage is present in the crown, and in TR4c the root is affected more severely than the crown.
- **Stage 5 (TR5):** Total resorption results in loss of the crown, with roots muddled into the bone.

Altier is not familiar with any studies that provide a definitive answer on how long it takes to advance from stage 1 to stage 5. "The progression is most likely specific to that patient and that tooth," she said, "but in my experience, it takes months to years for a tooth to go from beginning to end-stage resorption."

Dogs

Because much of the veterinary research on tooth resorption has focused on cats, less is known about the pathogenesis of the condition in dogs (in which it is sometimes called idiopathic root resorption). However, a 2010 study from the University of California, Davis (UC Davis) provided an unprecedented look into how the disease progresses in dogs.[2] The 2 most common types of tooth resorption were detected in 120 of the 224 (53.6%) study dogs and in 943 of 8478 (11.1%) teeth studied. External replacement resorption was found in 34.4% of dogs and external inflammatory resorption in 25.9%.

Based on the findings from this study, as well as the similarities in how tooth resorption develops in humans and dogs, the classification of canine tooth resorption mimics the system used in human dentistry.[2,3] Categories are listed in the order of most common occurrence in the UC Davis study dogs where resorption affected 53% of dogs[2]:

- **External replacement resorption:** This is the most commonly occurring tooth resorption in dogs (34.4%). Radiographs reveal an indiscernible

periodontal ligament space and remodeling of root structure into the alveolar bone.

- **External inflammatory resorption:** Nearly 26% of the study dogs were found to have external inflammatory resorption, in which inflammation is thought to be the initiating factor. Radiographs may show areas of peri-apical lucency around resorbing tooth roots.
- **External cervical root surface resorption:** Just 0.3% of study dogs fell under this classification. Radiographs show the progression of these lesions beginning at the cervical area of the tooth where cementum meets enamel.
- **External surface resorption:** Only 0.2% of study teeth were affected by external surface resorption, a condition often unaccompanied by clinical signs and rarely apparent on radiographs. When it is seen on radiographs, it appears as shallow discontinuances of the tooth structure on the lateral edges of the root, and it only involves the cementum and dentin.
- **Internal inflammatory resorption:** This was a very rare finding in study dogs (0.1%). These oval-shaped enlargements, thought to result from endodontic disease, are often located in the cervical area of the root canal.
- **Internal surface resorption:** This type of resorption was found in only 1 study dog that did not have concurrent periodontal or endodontic disease. Mild trauma may be the initiating factor causing these oval-shaped enlargements found in the apical third of the root canal.
- **Internal replacement resorption:** Not found in any study dogs, internal replacement resorption is a progressive condition that typically presents as an uneven expansion of tunnel-like areas near the coronal segment of a root fracture.

The bottom line

Educating pet owners well and often about the importance of yearly dental evaluations is the best way to advocate for your patients and help those that may be suffering in silence. "Like any dental disease, tooth resorption is progressive," Altier said. "And without treatment, it will never get any better than it is today. You have to intervene."

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Preparing Owners for the Death of a Pet

Deciding to euthanase a pet is a difficult decision. Veterinarians can guide owners through the process and make the road to death more comfortable for both the patient and client by using a combination of compassion and medical expertise.

Kate Boatright, VMD

How do you know when it is time? Veterinarians are commonly asked this question. According to Lynn Hendrix, DVM, CHPV, cofounder and outgoing president of the World Veterinary Palliative Medicine Organisation, sometimes it is asked because the owner is looking to an expert for permission to go ahead with a decision they've already made, and sometimes it is because they truly do not know. During a recent Fetch dvm360® virtual conference, Hendrix discussed how veterinarians can help their clients prepare for the death of their pet.

The psychology of end-of-life decisions

Hendrix said that the reactions to decisions surrounding the death of a pet are often driven by fear. This fear may be about the impending loss, the cost of treatment, of waiting too long to help their pet, or of feeling shame about their decision.

This fear can manifest in many ways, usually through the fight, flight, or freeze response seen when the body is under stress. Those whose fear manifests as anger are usually in fight mode. Those in flight mode may leave the clinic without making a decision or even taking medications. The last group are those who seem to be in denial but who are in fact experiencing the freeze response. In Hendrix's experience, "most people who seem to be in denial are not. They are fearful."

She encourages veterinarians to give owners time to process and take the time to discuss with them what they may be fearful of. It is essential for the patient's well-being that the veterinary team and client be on the same side. Trust must be established, and judgment withheld. This can be difficult when you and the client are seeing different things when looking at the pet.

Hendrix reminds us that perceptions vary among individuals and are influenced by past experiences. She differentiated the perception of patient suffering from that of patient distress. Those without medical training may not perceive that their pet is suffering as we might. Distress, on the other hand, can be observed. We can advise owners on clinical signs to watch for, such as changes in respiratory patterns or posture that may be indicative of respiratory distress or pain. Once an owner can recognise signs of distress in their pet, they are more likely to be open to a conversation about palliative options.

Patient assessment

The first step in guiding owners through end-of-life decision-making for a pet is an examination. Pain assessment is crucial, as Hendrix finds that "many diseases have pain at the end stage." It is important that the veterinarian identify signs of pain and help the owner to recognise them as well. Hendrix recommends utilising validated chronic pain assessments. For dogs, the Helinski Brief Pain Index and Canine Brief Pain Inventory provide good options. For cats, the Feline Musculoskeletal Pain Index is useful. Hendrix noted that the Feline Grimace Scale was recently validated for acute pain but may be useful in end-of-life situations as well.

When it comes to quality-of-life assessments, many exist but most are not validated. Hendrix does not use the current scales as she feels many of them miss the more subtle changes in the patient, emotional impact on the family, the patient's will to live, or considerations of the family's quality of life in caring for the patient. Quality of life is multifactorial, nuanced, and influenced by the client's perceptions, so assessing it should be done through conversations between the family and veterinary team.

Advanced planning

When Hendrix works with families, she encourages them to have a plan in place for how they want their pet's death to happen, regardless of whether it be euthanasia or palliated death. She encourages veterinary teams to discuss several factors with clients:

- Where will the pet's death happen, and what items would the family like to have around the pet in this place?
- Who will be present? This decision should include both family and veterinary team members. Hendrix recommends that families have a backup plan in case the primary veterinarian they have chosen is unavailable when the time comes.
- How will the death happen? Will it be euthanasia or a palliated death?
- What discussions need to happen with children and other family members before and after the death?
- What plan does the family have for grief or spiritual support, if desired?
- What are the aftercare plans?

Another component of planning is to develop an advanced directive for the pet. Just as humans have advanced directives for their own medical care in the event of an emergency, this plan can be determined ahead of time to avoid the family having to make difficult decisions during an emergency. Factors that should be considered in the advanced directive should include what the family wants their pet to be able to do, what they don't want their animal to experience, the amount of acceptable medical intervention, and considerations around the family's quality of life in caring for the pet (eg, waking throughout the night, nursing care).

Be prepared: Crisis kits

For families whose pet in a hospice situation, Hendrix provides a crisis kit that contains medications and instructions for when they might need to be used. These instructions are specific and explicit and include when to give the medication, how to administer it, how long it will take to have its intended effect, and when the next dose should be given if needed.

Crisis kits are also known as comfort kits and should include medications that anticipate potential emergency situations the pet and family might face during the end stages of the disease. Pain management for acute, severe, or unrelenting pain is a must. This is often in the form of an opioid that can be given by injection or via the transmucosal route.

Medications to manage anticipated signs of the disease should be included as well. The make-up of each kit will vary by patient and disease but may include these components:

- **For management of respiratory distress:** a short-term oxygen supply, sedation to relax the pet, or nebulised medications
- **For acute seizures:** benzodiazepines, gabapentin, and honey or karo syrup in case there are concerns for hypoglycaemic seizures
- **For haemorrhage:** aminocaproic acid and Yunnan Baiyao
- **For sleep disturbances, anxiety, and agitation:** Acepromazine, melatonin, or a benzodiazepine(or alprazolam ed)
- **For nausea and vomiting:** maropitant (Cerenia; Zoetis), ondansetron, or metoclopramide
- **For diarrhoea:** metronidazole and clay products (eg Pectrolyte - ed)

Finally, for owners who have planned a palliated death, items to prepare for caring for a recumbent pet should be included, including lubricating eye drops. For those who have planned for euthanasia, higher doses of sedatives can be included and administered when the owners feel it is time to euthanase. The goal of these medications is to sedate the pet enough for a peaceful and safe transport to the veterinary office or until a house call veterinarian can arrive.

Palliated death

While most veterinary professionals would prefer a pet in the end stages of disease to be euthanased, not all owners are comfortable with or ready to make that decision. A palliated death allows the pet to die from

Editor's Note / Tip

I place an IV catheter and an extension set during euthanasia when the owner want to be present - this means that the owners stand close to their pet and the vet is standing further away out of the picture.

Additionally with cats especially I will use the IP route in older patients rather than keep trying for vein. Injection of pentobarbitol IP is not at all painful and will cause less distress than the restraint required to place a catheter in many patients. The patient gradually loses consciousness - the speed depending on the perfusion of the area injected. Aiming the needle for the liver also helps as the organ is highly perfused and that makes the process faster. Injecting into the kidney is painful as it is encapsulated. Intra-cardiac (IC) injection can then be given after the patient is unconscious. IC injection should never be performed in an awake animal as it is painful.

the disease, but with interventions to control pain during the end stages.

In many cases, palliative sedation is used, which maintains life while providing pain relief and keeping the patient subconscious to unconscious for the remainder of the disease course. It is important to discuss with owners that for many palliated deaths, 24-hour recumbent care and extensive nursing care are needed. In Hendrix's experience, once owners are educated on this process, many elect euthanasia for their pet.

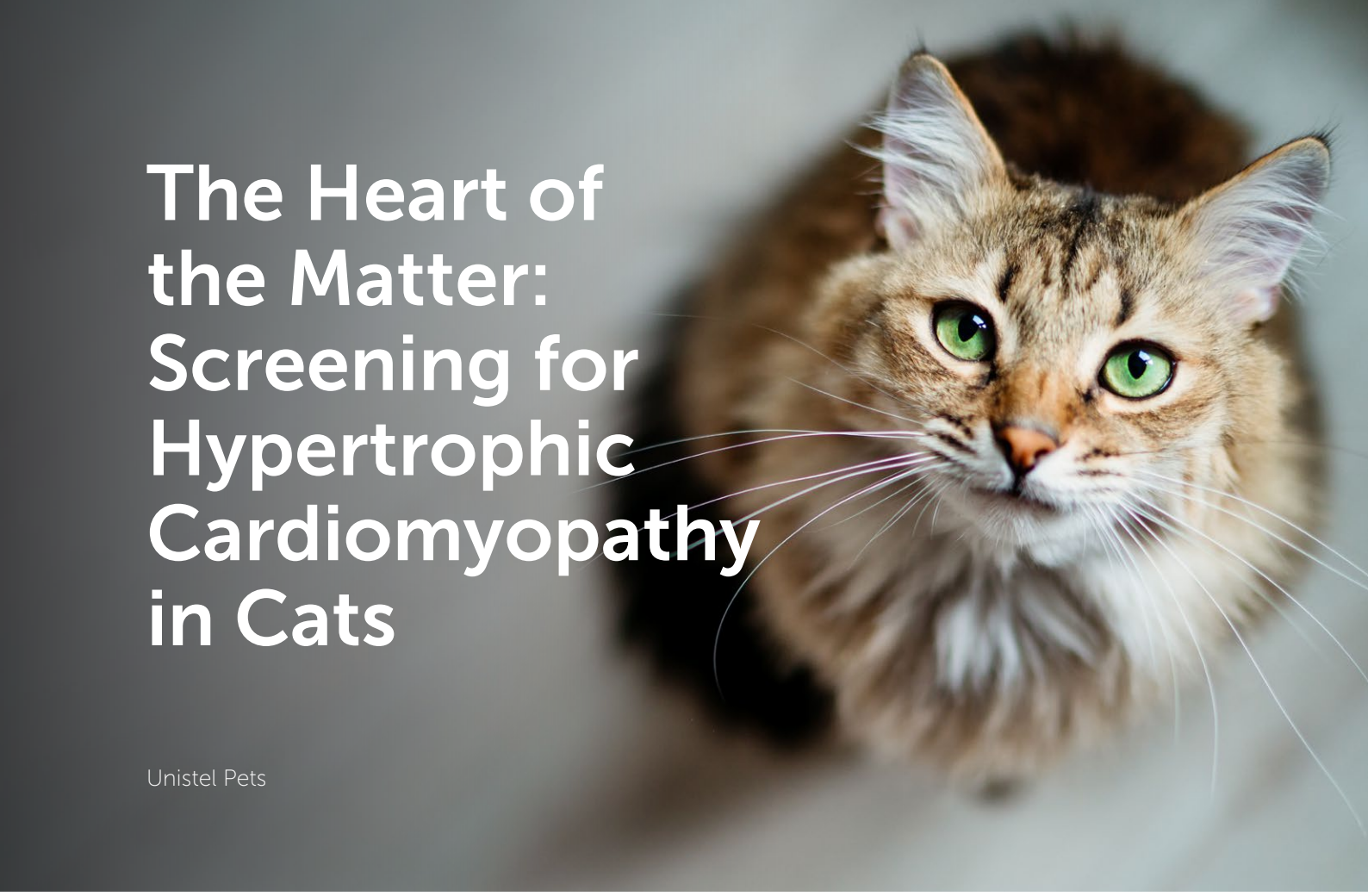
The gentle euthanasia

For owners who choose to euthanase their pet, minimising distress and providing a gentle euthanasia experience should be the veterinary team's priority. Hendrix encourages veterinarians to consider how each drug and step in the process makes the client and the patient feel. She recommends administering sedation and/or pain medications via a subcutaneous or intramuscular route prior to placing an intravenous catheter. The use of topical numbing gels should be used during the placement of the catheter to minimise discomfort for the pet. She noted that some clients prefer their pets to become sedated gradually, allowing them to spend time with their beloved pet, while others prefer a fast experience. These preferences can influence choices of sedation and route of administration.

Take-home points

For each family, the decisions they make are related to their relationship with their pet during life. Having a veterinarian who can help them through the process and the many decisions involved in creating the best possible death for their individual family and pet is invaluable.

Kate Boatright, VMD, a 2013 graduate of the University of Pennsylvania, is a practicing veterinarian and freelance speaker and author in western Pennsylvania. She is passionate about mentorship, education, and addressing common sources of stress for veterinary teams and recent graduates. Outside of clinical practice, Boatright is actively involved in organised veterinary medicine at the local, state, and national levels.



The Heart of the Matter: Screening for Hypertrophic Cardiomyopathy in Cats

Unistel Pets

While most people are acutely aware of the dangers of heart disease in humans, very few consider the risk of cardiovascular diseases in their pets. Even fewer are aware that defects within their pet's heart muscles, referred to as Hypertrophic Cardiomyopathy (HCM), are the leading cause of heart failure in cats. In short, the heart consists of four chambers, with the top (atrium) and bottom (ventricle) right and left cavities separated by a barrier called the septum. Although some variation in wall thickness of the chambers is natural, most healthy cats fall within an average documented range (diastolic failure).

Pathophysiology of HCM

Cats who suffer from HCM, however, have walls that continue to thicken progressively, reducing the heart's effectiveness at filling and then ejecting its chambers.

This thickening has the most detrimental effect on the left ventricle, which is responsible for supplying the body (via the aorta) with blood, as well as the lower parts of the septum. As the chamber walls thicken, the muscles become more rigid, reducing their ability to expand and enlarge the chambers to allow the influx of sufficient blood. In addition, as the thickening ventricle tries to pump blood out, a portion of the valve may get sucked into the aortic outflow, obstructing blood flow and making it even more difficult for the heart to pump blood through the aorta. This phenomenon, referred to as systolic anterior motion (SAM), occurs in approximately two-thirds of HCM diagnosed cats, and can eventually lead to fluid retention in the lungs and congestive heart failure.

Signalment and clinical signs

Although HCM can occur in both male and female cats, the condition affects, on average, more males than it does females. The onset of the disease can greatly vary, with cats as young as 3 months and as old as 17 years being diagnosed with HCM; however, affected cats most commonly present with symptoms in their middle (4-8) years. Clinical signs also vary greatly from case to case, with as many as 55% of cats showing no outward signs of heart disease. Sadly, many of these cats are only diagnosed with HCM after being rushed to the vet with congestive heart failure already in progress, or sudden paralysis due to blood clots breaking off from the atriums. Should they occur, symptoms to look out for include a lack of appetite and weight loss, lethargy, reluctance to socialize, panting after exercise, laboured breathing and heart murmurs. Once the cat starts to retain fluid in the lungs, aggressive medical intervention will be necessary. Even with medical assistance, the cat will still be at risk of sudden heart failure due to the risk of irregular heart rhythms.

Diagnostic Approach

Although a physical examination may reveal the presence of a heart murmur, irregular heart rhythms (gallop rhythm), or abnormal lung sounds, the most effective screening method for picking up the presence of HCM seems to be performing an echocardiogram. Radiographs will not be of much use in diagnosing HCM in the early stages of the disease and performing an electrocardiogram (ECG) will only be effective in picking up HCM in 30% of cats with HCM as most cats do not show any disturbances in their electrical conduction systems. While the probable causes of HCM in cats have not yet been fully elucidated, a growing body of research indicates that certain breeds may have a genetic predisposition to hypertrophic heart failure. For example, mutations within the *MYBPC3* and *MYH7* genes, which result in alterations to the protein structure of the heart, have been causatively linked to the development of HCM in the Maine Coon, Ragdoll, and Domestic shorthair breeds. As the mutations are dominantly inherited, only a single defective copy of the gene is required for a cat to possess a predisposition for HCM. Fortunately, since the location of these mutations is now available, genetic screening tests for HCM are offered by several laboratories, enabling breeders to eliminate the mutation from their breeding animals through selective breeding.

Treatment

Although no medication will be able to reverse the thickening of the heart muscle, the condition can be managed as it progresses. The initial strategy is aimed at slowing down the heart rate, which in theory will allow more time for the heart to fill and empty its chambers. More aggressive therapies will be necessary once the cat progresses to congestive heart failure. This can include the use of diuretics to try and reduce the amount of fluid accumulated in the lungs, additional oxygen, using anticoagulants to prevent thrombosis, converting enzyme (ACE) inhibitors to reduce blood pressure, and cage rest. Cats that are asymptomatic for HCM may not immediately require medical intervention, but routine check-ups are advised to monitor the progression of the disease.

Prognosis and survival

The rate of progression and the prognosis will also differ from case to case. Some cats will progress to heart failure very rapidly, while others may remain relatively healthy for years. But ultimately, all diagnosed cases will lead to congestive heart failure. Cats that are asymptomatic and have HCM detected by chance during a routine check-up, have better survival rates than those who are diagnosed due to blood-clot-induced paralysis or congestive heart failure collapse.

Cats that have already developed blood clots have the most unfavourable prognosis: those who survive the first 24 hours have an average survival rate of two to six months. Cats that are diagnosed with congestive heart failure, will on average survive between three to eighteen months, and those who show no clinical signs can live between three to five years after initial diagnosis. Ultimately, the only way to prevent HCM from developing in breeds with known mutations is to breed with lines that are clean of the mutation.

In the case of an affected individual, knowing their HCM status before the onset of symptoms can assist greatly in managing the condition. While no medication can cure the condition, some steps can be taken to slow down the progression of the disease and delay the need for immediate medical attention for quite some time.

Cardiac Tumours in Dogs and Cats

Considerations for the use of echocardiography to evaluate suspected tumours located in the heart.

Liza S. Köster, BVSc(Hons), MMedVet(Med), DECVIM-CA (Cardiology and Internal Medicine), EBVS , P. Brent Lawson, DVM , Josep Aisa, DVM, DECVS

Although rare in veterinary medicine, cardiac tumours can cause life-threatening complications, including pericardial effusion and tamponade, congestive heart failure, blood flow obstruction, and arrhythmias.[1-5] However, because presenting clinical signs in patients with cardiac tumours are usually nonspecific, a low level of suspicion often hampers diagnosis.

Reports from owners may include hyporexia, lethargy, weakness, neurologic signs, vomiting, and abdominal distention. Except for abdominal distention and perhaps weakness, however, none of these signs are likely to prompt a clinician to image the heart.[5-7] Triage of emergent patients by point-of-care focused cardiac ultrasound has revolutionized the ability to diagnose pericardial effusion in clinical practice, overcoming the deficiencies of low sensitivity and specificity reported with thoracic radiography.[2,8,9]

The leading cause of pericardial effusion in dogs is cardiac neoplasia. Over 70% of cases have either right atrial or heart-based masses, often concurrent with pleural effusion, ascites or, more commonly, tricavitary effusions.[7] Pericardial effusion is reported in at least 16% of all dogs diagnosed with cardiac tumours on necropsy, whereas 42% of dogs with antemortem echocardiographic diagnosis, later confirmed by necropsy or cytology, have a concurrent pericardial effusion.[5,7] Arrhythmias are reported in dogs associated with haemangiosarcoma and chemodectoma, with ventricular ectopy or tachycardia most frequently described. Other findings include electrical alternans in dogs with concurrent pericardial effusions and, rarely, supraventricular tachycardia, atrial fibrillation, and atrio-ventricular and bundle branch blocks.[2-4]

The role of echocardiography

Echocardiography is the screening test of choice in dogs with pericardial effusion. Similarly, dogs presenting with dysrhythmias require echocardiography as structural heart diseases, including infiltrative tumours, are considered possible aetiologies. Echocardiography has its limitations, but because ante-mortem confirmation of a cardiac tumour is rarely obtained by cytology or biopsy, necropsy is often the only means of confirming the diagnosis. Echocardiography is considered moderately accurate, with 86% agreement on location and only 65% agreement on tissue-type diagnosis when compared with necropsy.[5] Diagnostic accuracy vastly improves in the presence of pericardial effusion, with a reported sensitivity of 82% and specificity of 100% for detection of a cardiac mass.[2]

Echocardiography can provide information on the mobility, infiltrative nature, attachment to, and hemodynamic consequences of a cardiac mass.[10] Despite the high specificity quoted, the differential diagnosis should include infectious vegetations and sterile thrombi. Due to the inherent risk for false-negative echocardiographic diagnoses, particularly in the absence of pericardial effusion, it is always prudent to keep cardiac neoplasia as a potential diagnosis despite apparent negative findings on echocardiography. Factors such as patient signalment and the high rate of pericardial effusion recurrence may increase the suspicion of a tumour. In mesothelioma, a solid mass may not be visible, and this neoplasia should remain a potential working diagnosis.[11] It is worth noting that these results are operator dependent and reports are based on data collected from presumptive diagnoses made by board-certified cardiologists.

Tumour classification and echocardiographic phenotype

Cardiac tumours are either primary or secondary (metastatic) and benign or malignant.[6,12] Clinical diagnosis in dogs is predominantly of primary and malignant tumours, with the most common aetiologies being haemangiosarcoma followed by chemodectoma (also known as a neuroendocrine tumour, chromaffin cell tumour, aortic body tumour, and carotid body tumour). Cardiac metastatic disease may be more prevalent and silent than clinically

seen, with up to 86% of cardiac tumours in dogs diagnosed as metastatic at the time of necropsy and 36% of malignant neoplasias metastasizing to the heart.[6,7,13] In cats, cardiac tumours are almost exclusively primary and the majority are extra-nodal lymphomas.[6,14,15] Excluding lymphomas, which tend to develop at an earlier age, cardiac tumours usually affect dogs ranging in age from 7 to 15 years.[7] There is no reported gender predisposition, and the risk may be lower in intact animals.[7] Breed consideration is important; breeds with reported high cardiac tumour incidence include the Saluki, French bulldog, Irish water spaniel, flat-coated retriever, golden retriever, boxer, Afghan hound, English setter, Scottish terrier, Boston terrier, bulldog, and German shepherd.[7]

Knowledge of the echocardiographic phenotypic appearance of cardiac tumours is helpful to the clinician, as it remains the undisputed ante-mortem diagnostic test of choice for this condition. This understanding will allow the clinician to provide appropriate treatment options and give an estimate of prognosis. As noted earlier, ante-mortem cytologic and histologic diagnosis is often not possible.

Treatment and prognosis

Tumours are often described according to anatomic location (Fig. 1), including right atrium, heart base, inflow or outflow tract of the left or right heart chambers, atrio-ventricular valves, ventricular

Tumour histologic type	Epidemiology and Imaging findings	Treatment options and median survival
Canine cardiac haemangiosarcoma	• Most common primary cardiac tumour in dogs • German shepherds and Labrador retrievers overrepresented • Primarily located in the right atrial appendage or atrium, rare reports in the right ventricle, interventricular septum, and pericardium • Described as circular or irregular within the right atrial wall or protruding from the right atrial wall, but can be located surrounding the ascending aorta; often have small, multifocal, echolucent areas within the tumour • Solitary or can be accompanied by splenic masses in up to 29% of cases • Often accompanied by pericardial effusion, which provides ultrasonographic contrast to improve visualisation	• Shortest MST compared with other tumours • Intermittent pericardiocentesis with gross disease (MST up to 12 days) • Palliative pericardiectomy monotherapy may risk fatal bleeding • Single-agent (MST 116 days) or combination protocol (MST 172 days) chemotherapy • Surgical resection (including thoracoscopic) is preferred for resectable masses (e.g. pedunculated right atrial appendicular) and patients that are appropriately staged (MST up to 86 days without adjuvant chemotherapy or up to 189 days with adjuvant chemotherapy) • Yunnan Baiyao, although safe, has not been proven to delay recurrent clinical signs or increase survival in dogs with suspected hemangiosarcoma • Role of radiotherapy needs further evaluation
Aortic body tumour (chemodectoma)	• Second most common cardiac tumour in dogs • Originates from the carotid or aortic body in the region of the aortic arch • Thought to be less invasive and have a lower matastatic rate than the carotid body tumours, often behaving in a benign manner • Old brachycephalic dogs overrepresented • Often diagnosed incidentally or late in the course of the disease secondary to pleural or pericardial effusion, arrhythmias, cranial caval syndrome, or coughing due to tracheal or bronchial compression • On echocardiography associated with ascending aorta without right atrial involvement; tumours are homogenous, seen to move with the aorta, avoid in shape, well-defined and localised.	• Surgical resection is, in general, not feasible or indicated due to slow tumour growth and high morbidity released to the procedure • Non-resectable masses (due to local invasion, questionable invasion, or owner limitations) without pericardial effusion can be monitored with echocardio therapy • Pericardiectomy, even in the absence of pericardial effusion, significantly increases survival (MST up to 730 days with pericardiectomy vs 42 days without) • Toceranib phosphate (MST 823 days) • Role of radiotherapy needs further evaluation
Lymphoma	• Although cardiac and pericardiac lymphoma in cats is rare, about 20% to 30% of pericardial effusions in cats are still caused by cardiac neoplasia • In cats, a wide range with an MST of 5 years; in dogs, reported in young animals and one puppy • Echocardiography may not detect a discrete mass but rather hypokinesis or segmental hypertrophy mimicking hypertrophic cardiomyopathy • Pericardial lymphoma appears as a nonhomogeneous echogenicity with a thickened pericardium • Description also includes a hypoechoic, space-occupying mass of the right atrium. • Cytology of the pericardial effusion may be helpful in antemortem diagnosis	• Pericardiocentesis is the presence of pericardial effusion followed by combination chemotherapy is the standard of care (MST 157 days in dogs; 750 days in 1 cat compared up to 11 days with prednisone monotherapy)

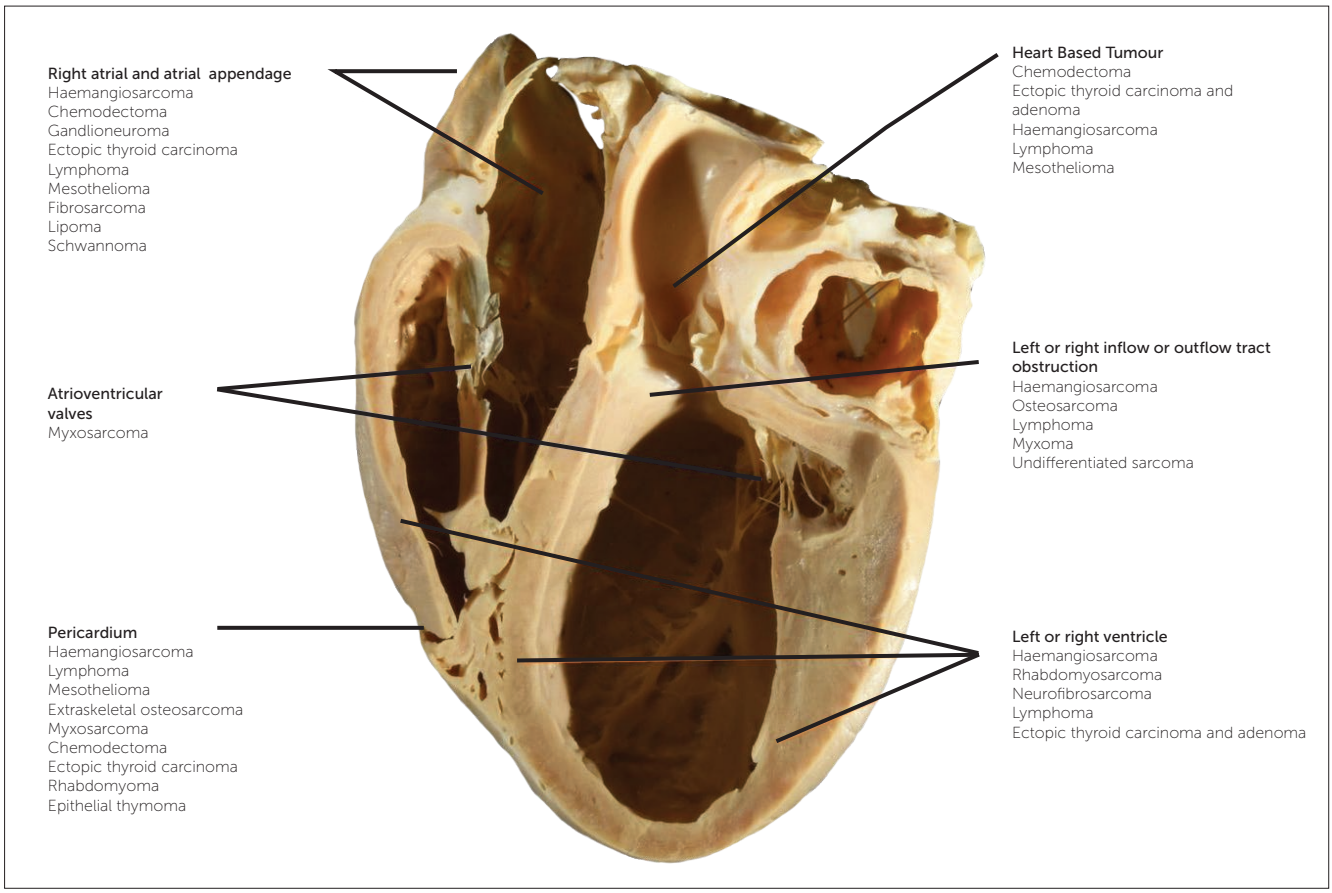


Fig. 1 - Locations of presumptive and definitive primary cardiac tumors according to echocardiographic anatomic location in dogs and cats. All images courtesy of Liza S Köster, BVSc(Hons).

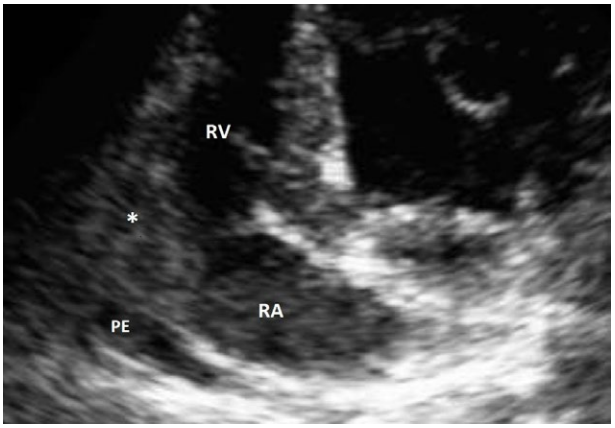


Fig. 2 - Left apical 4-chamber view with the right heart chambers depicted on the left side of the image, the right ventricle in the near field, and the right atrium in the deeper field. A roughly circular, well-defined hypoechoic mass (asterisk) can be seen in the junction between the right atrium and ventricle; the mass is heterogeneous due to multifocal hypoechoic areas. A moderate-volume pericardial effusion is visible as the anechoic area surrounding the heart. The echocardiographic diagnosis in this geriatric German shepherd dog was a right atrial mass; due to tumour location and patient signalment, the tentative diagnosis was haemangiosarcoma.

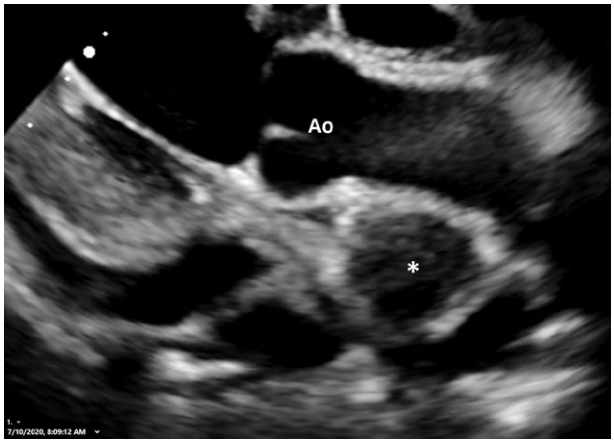


Fig. 3 - Left intercostal long-axis view depicting the left ventricular outflow tract. A well-demarcated, homogeneous, soft tissue mass (asterisk) can be seen along the ascending aorta. The echocardiographic diagnosis in this middle-aged boxer was a heart base tumour. Based on the mass location and patient signalment, the tentative diagnosis was an aortic body tumor (chemodectoma). The differential diagnosis would include an ectopic thyroid carcinoma or adenoma.

walls (including the inter-ventricular septum), and pericardium. Tumour histologic prediction is invariably made by a combination of signalment, echocardiographic phenotype, and anatomic location within the heart. Treatment options and prognosis for the most common cardiac tumours seen in dogs and cats are described in the Table.[12] (Fig. 2 to 4) depict the typical echocardiographic appearance of the most common cardiac neoplasia encountered in small animal practice: haemangiosarcoma, aortic body tumour, and lymphoma.

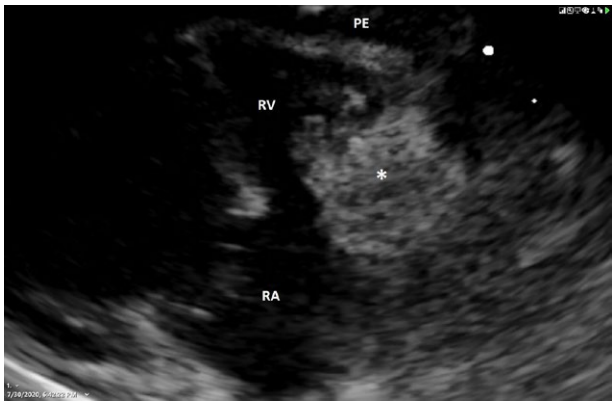


Fig. 4 - Left intercostal view of the right ventricle (RV) and atrium (RA) depicting the RV in the near field and the RA and atrial appendage in the far field. Trace pericardial effusion is visible as the anechoic stripe between the pericardium and RV. The asterisk indicates hyperechoic segmental thickening of the RV and atrial wall. The tentative diagnosis in this geriatric domestic shorthair cat was an extranodal lymphoma. Pericardial fluid analysis may have provided cytologic evidence of this tumour, but it was not attempted due to the scant volume.

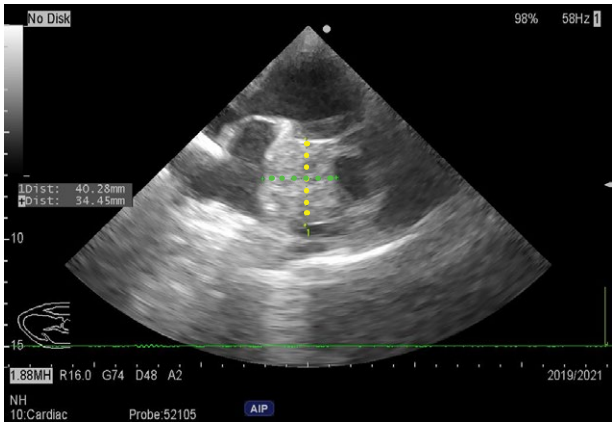


Fig. 5 - Right parasternal short axis view of the heart of a 13 year-old Jack Russel Terrier showing showing a well defined hyperechoic mass at the heart base. The echocardiographic diagnosis is a chemodectoma. (Image courtesy of Dr Nicolene Hoepner, Companion Animal Clinical Studies Diagnostic Imaging Section. OVAH).

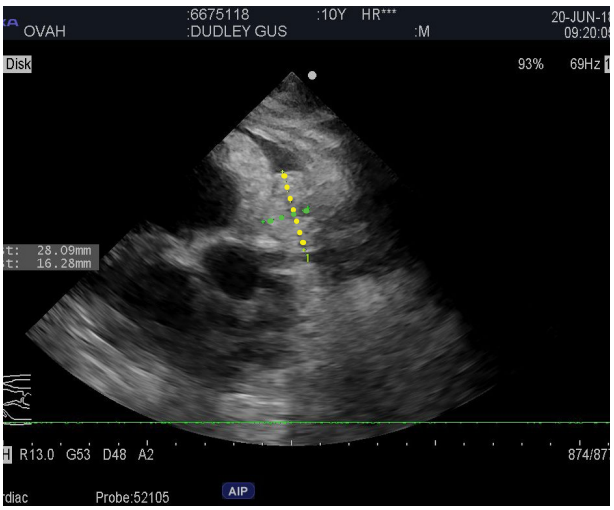


Fig. 6 - Right parasternal view adapted to visualise a cardiac mass in a 10 year-old Bull Terrier dog. The mass can be seen protruding into the right atrium and a small amount of pericardial effusion is visible in the near field.

Tumours are often described according to anatomic location (Fig. 1), including right atrium, heart base (Fig. 5), inflow or outflow tract of the left or right heart chambers (Fig. 6), atrioventricular valves, ventricular walls (including the interventricular septum), and pericardium. Tumour histologic prediction is invariably made by a combination of signalment, echocardiographic phenotype, and anatomic location within the heart. Treatment options and prognosis for the most common cardiac tumours seen in dogs and cats are described in the Table.[12] (Fig. 2 to 4) depict the typical echocardiographic appearance of the most common cardiac neoplasia encountered in small animal practice: haemangiosarcoma, aortic body tumour, and lymphoma.

Conclusion

Although uncommon, cardiac tumours can have catastrophic consequences for dogs and cats. Knowledge of the sonographic appearance and location of cardiac tumours can help the clinician make a tentative diagnosis. Although invaluable when formulating appropriate management and treatment options, consider the limitations of echocardiography.

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Cardiac Tumours in Dogs and Cats

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1. Which one of the complications listed below does NOT commonly occur with cardiac tumours?

a. Pericardial effusion and possibly tamponade.
b. Congestive heart failure.
c. Obstruction to the flow of blood.
d. Embolic disease due to hypercoagulability.
e. Cardiac arrhythmias.

2. Which one of the statements below regarding pericardial effusions and cardiac neoplasia in dogs is INCORRECT?

a. The leading cause of pericardial effusion in dogs is cardiac neoplasia.
b. Cytology of the pericardial fluid assists in definitive diagnosis of the neoplasia.
c. Over 70% of cases with pericardial effusion have either right atrial or heart-based masses.
d. Pericardial effusion is often concurrent with pleural effusion and ascites.
e. Pericardial effusion is reported in at least 76% of all dogs diagnosed with cardiac tumours on post mortem.

3. Which one of the following statements regarding echocardiography and cardiac tumour diagnosis is INCORRECT?

a. Echocardiography is the screening test of choice in dogs with pericardial effusion.
b. Diagnostic accuracy for tumour diagnosis vastly improves in the presence of pericardial effusion.
c. Echocardiography has a reported 100% sensitivity for detection of a cardiac mass in a patient with a pericardial effusion.
d. Echocardiography is considered moderately accurate with 86% agreement, with post mortem, on location of tumour mass.
e. Echocardiography only has 65% agreement on tissue-type diagnosis compared to post mortem.

4. Which one of the statements below, regarding the echocardiography of cardiac tumours, is INCORRECT?

a. Structural heart diseases, including infiltrative tumours, are considered possible aetiologies for arrhythmias.
b. Infectious vegetations and sterile thrombi may cause false positive diagnoses.
c. Echocardiographic evaluation of the heart for cardiac neoplasia is very sensitive, even in the absence of a pericardial effusion.
d. Echocardiography can provide information on the mobility and infiltrative nature of a cardiac tumour.
e. Patient signalment and a high rate of pericardial effusion recurrence may increase the suspicion of a cardiac tumour.

5. Which one of the following statements regarding cardiac tumours in dogs is INCORRECT?

a. Dogs are predominantly diagnosed with primary and malignant cardiac tumours.
b. Cardiac tumours in cats are almost exclusively primary and the majority are extra-nodal lymphomas.
c. In mesothelioma, a solid mass may not be visible on echocardiography.
d. Cardiac metastatic disease is uncommon.
e. Breed consideration is important; breeds with reported high cardiac tumour incidence include the French bulldog, flat-coated retriever, golden retriever, boxer, Scottish terrier, Boston terrier, bulldog, and German shepherd.

6. Which one of the following statements regarding haemangisarcoma (HSA) cardiac tumours is INCORRECT?

a. HSA is the most common cardiac tumour in dogs.
b. German shepherd dogs and Labrador retrievers are over represented with HSA.
c. HSA are accompanied by splenic masses in the majority of patients.
d. The primary location for cardiac HSA is the R atrium.
e. Echocardiographic presentation is circular or irregular within the R atrial wall or protruding from the R atrial wall.

7. Which one of the statements below regarding chemodectoma is INCORRECT?

a. Chemodectoma originates from the carotid or aortic body.
b. Chemodectoma behaves in a relatively benign manner.
c. Older brachycephalic dogs are overrepresented with chemodectoma.
d. The diagnosis of chemodectoma is most commonly incidental.
e. Common owner history in these cases is of dogs being restlessness and having difficuty in sleeping.

8. Which one of the following conditions listed is NOT a complication of a chemodectoma?

a. Syncope
b. Pericardial effusion.
c. Pleural effusion.
d. Cranial caval syndrome.
e. Coughing due to tracheal compression dogs being restlessness and having difficuty in sleeping.

9. Cardiac tumours are described by anatomic location and this plays a big part in the description and identification. Which one of the following statements is INCORRECT?

a. Haemangiosarcoma often accompanied by a pericardial effusion.
b. HAS often have small multifocal hypoechoic areas within the mass.
c. Chemodectoma associated with ascending aorta.
d. Chemodectomsa are ovoid well defined homogenous tumours.
e. Chemodectomas may present as more than one mass at the heart base.

10. Which one of the following statements regarding cardiac lymphoma in cats is INCORRECT?

a. Cardiac and pericardial lymphoma is rare in cats.
b. Cardiac and pericardial lymphoma oaccounts for 30% of tumours causing pericardial effusion.
c. Cardiac lymphoma is generally part of a stage 4 lymphoma in the cat.
d. Echocardiography may show segmental hypertrophy and may appearas segmental HCM.
e. Cytology of the pericardial fluid would assist in confirming a diagnosis.

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Pericardial Effusion:
Causes and Clinical Outcomes in Dogs

Kristin MacDonald, DVM, PhD, DACVIM (Cardiology)

Pericardial effusion is a fairly common acquired heart disease in dogs, and prevalence has been reported to be 0.43% (or 1 dog per 233 cases) of dogs presenting to a referral veterinary hospital, and accounts for approximately 7% of dogs with clinical signs of cardiac disease.

It is a multi-aetiologic disorder (including infectious, inflammatory, and neoplastic causes) with a wide spectrum of prognoses ranging from good to grave. Careful echocardiographic evaluation for cardiac masses is necessary, and localisation of the mass to specific anatomic regions such as the heart base or right atrium is very critical to provide prognostic value.

The most common presenting complaint of dogs with pericardial effusion is collapse, weakness, syncope, or lethargy. Dogs may present with abdominal distension and ascites secondary to cardiac tamponade. Heart sounds are muffled, and lung sounds may also be muffled if there is pleural effusion. Femoral pulses are weak, and sometimes pulsus paradoxis may be palpated when the pulse is stronger during exhalation and weaker during inhalation. If there is cardiac tamponade, the animal may have signs of cardiogenic shock including pale mucous membranes, cold extremities, hypotension, tachycardia, and collapse. These cases require immediate triage for emergency diagnostics and treatment.

Initial triage of a dog with cardiovascular collapse includes a brief triage echocardiogram to assess if there is significant pericardial effusion as a cause of the collapse, as well as an abdominal scan for free abdominal fluid in cases of haemoabdomen. An intravenous catheter should be placed and shock doses of fluids given. Immediate pericardiocentesis is necessary in dogs with low output heart failure and collapse.

The animal is placed in left lateral recumbency, and echocardiography is used to define the most optimal site for pericardiocentesis at the right 5-6th intercostal spaces, where there is greatest amount of pericardial fluid the furthest away from the heart and great vessels. In animals that are stable and do not have signs of cardiac tamponade, it is advisable to postpone pericardiocentesis until a detailed echocardiogram is done, as long as it is within a relatively short period of time.

Echocardiography is necessary to diagnose presence and severity of pericardial effusion and a detailed echocardiogram is needed for diagnosis of cardiac masses. Masses are typically classified as right atrial (infiltrating the right atrium or auricle) or heart base (adherent to the ascending aorta), and other less common masses may be located on the pericardium or ventricles.

Cardiac tamponade is diagnosed when there is diastolic collapse of the right atrium and/or right ventricle, and indicates that the pericardial effusion is haemodynamically compromising and requires timely pericardiocentesis. Very mild pericardial effusion may be seen with severe right heart disease such as dilated cardiomyopathy or severe tricuspid regurgitation, but is haemodynamically insignificant. Absence of a mass places idiopathic pericarditis, mesothelioma, or infectious causes as highest differentials.

Fluid analysis and cytology is necessary to diagnose infectious causes of pericardial effusion, but is not

Editor's Note

Ultrasonography is the most sensitive tool to detect pericardial effusion. Any probe will be sufficient for a diagnosis, although careful examination using the cardiac probe will be necessary to determine the cause of the effusion and eliminate neoplasia. If thoracic radiographs are made the cardiac silhouette is likely to be enlarged and will have the appearance of an apple with the pericardium bulging like shoulders around the heart base. (Fig. 1).

The presence of signs of cardiac failure is not correlated to the degree of distension but rather to the distensibility (stretch) of the pericardium. Some patients may have only moderate amounts of fluid in the pericardial sac but show signs of cardiac tamponade due to fibrosis of the pericardial sac. as the pericardium is not able to stretch to accommodate the fluid.

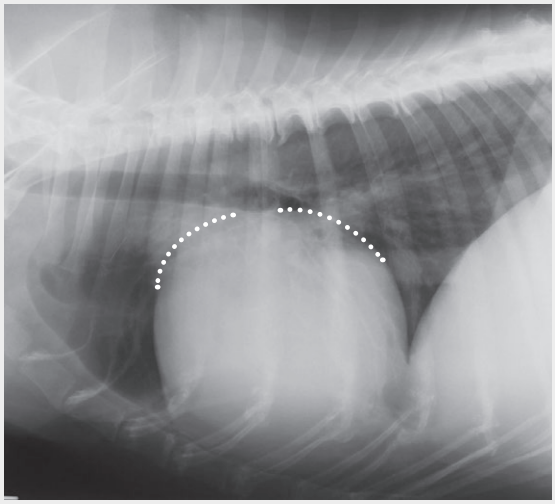

Fig.1 - R lateral thoracic radiograph showing the typical curved appearance of the cardiac silhouette with pericardial effusion. The very rounded shape at the heart base is not typical of a patient with DCM

usually helpful to diagnose neoplastic causes or to differentiate idiopathic pericarditis from neoplastic causes. Most pericardial effusion is classified as haemorrhagic, and often there is mesothelial reactivity. Lymphoma is one neoplastic aetiology that may be reliably diagnosed by fluid analysis. Over-interpretation of mesothelial hyperreactivity as mesothelioma based on fluid cytology has been common in the past.

Approximately 50% of idiopathic pericarditis cases have recurrent pericardial effusion. In cases of recurrent pericardial effusion without an identifiable mass, subtotal pericardectomy is necessary, and histopathologic evaluation of the pericardium is necessary (often with special immunohistochemical stains) for differentiation of idiopathic pericarditis from mesothelioma. Subtotal pericardectomy is curative for idiopathic pericarditis. Partial pericardectomy is indicated for dogs with heart base masses, as it relieves cardiac tamponade and is associated with a significant prolongation of survival time (median survival time of 730 days pericardectomy versus 42 days without pericardectomy). Pericardectomy is not recommended for dogs with haemangiosarcoma, unless it is combined with mass resection. In dogs with right auricular haemangiosarcoma, surgical resection is a feasible option, followed by chemotherapy. In a small study of 23 dogs with surgically resected right atrial or right auricular haemangiosarcoma, administration of chemotherapy increased survival time (MST with chemotherapy and surgery 175 days versus 42 days with surgery alone) No studies have evaluated whether chemotherapy alters survival time in dogs that do not have surgical mass resection. In the author's experience, recurrent acute haemorrhage and cardiac tamponade is common in dogs with cardiac haemangiosarcoma, and usually is lethal before the animal succumbs to metastatic disease.

Retrospective study of 107 dogs with pericardial effusion

The speaker has recently authored a recent retrospective study evaluating 107 dogs diagnosed with pericardial effusion that underwent pericardectomy or necropsy. Surgery and/or necropsy identified 66 dogs with cardiac masses and 41 dogs without a mass. The location of masses included: 38 right atrial masses, 23 heart base masses, 2 concurrent right atrial and heart base masses, 2 pericardial masses, and 1 right ventricular mass.

Echocardiography by a board certified cardiologist or supervised cardiology resident identified 53 of 66 dogs with cardiac masses, including 32 of the 38 dogs with right atrial masses and 17 of the 23 dogs with heart base masses. Echocardiography was sensitive (80%) and specific (100%) for detection of a cardiac mass, and was equally sensitive (84%) and specific (100%) for distinguishing right atrial masses from all other aetiologies, or distinguishing heart base masses from all other aetiologies (sensitivity 74% and specificity 98%).

Of the 12 dogs which echocardiography missed masses on initial examination, only 4 dogs had repeated echocardiograms, which identified cardiac masses. This increased the sensitivity of echocardiography to detect a cardiac mass to 88%. This illustrates the importance of repeating echocardiograms in dogs with recurrent pericardial effusion to identify masses that were not evident on the initial examination. Eight of the 12 dogs with masses that were missed on the first echocardiographic examination had a small volume of pericardial effusion at the time of the echocardiogram.

Aetiology of pericardial effusion

Neoplasia was the most common cause of pericardial effusion (71% of dogs). Haemangiosarcoma was the most common aetiology of pericardial effusion (34%), followed by idiopathic pericarditis (20%), mesothelioma (14%), chemodectoma (8%), thyroid adenocarcinoma (6%), infective pericarditis (5%), lymphoma (3%), sarcoma (2%), and 1 case of each of the following: carcinomatosis, ruptured left atrium secondary to severe mitral regurgitation, sterile foreign body, and granuloma. 33% of cases of mesothelioma had discrete cardiac masses, most often of the heart base (4/5) and rarely of the right atrium (1/5).

A majority of right atrial masses were haemangiosarcoma (35/40 dogs, 88%), followed by 1 case (2.5%) of each: neuroendocrine tumour, thyroid adenocarcinoma, mesothelioma, lymphoma, and sarcoma. Heart base masses were most often neuroendocrine tumours (40%), followed by thyroid adenocarcinoma (25%), mesothelioma (20%), and haemangiosarcoma (15%). 1 dog had concurrent right atrial haemangiosarcoma and a neuroendocrine heart base mass. The pericardial masses were caused by one case each of lymphoma and a granuloma. The right ventricular mass was an undifferentiated sarcoma. 3 of 5 cases of infective pericarditis were caused by fox tail foreign bodies with secondary bacterial infections.

Half of dogs with pericardial effusion had evidence of right heart failure, with equal occurrence of ascites or pleural effusion (50% and 47% respectively), with fewer dogs having concurrent ascites and pleural effusion (33%). There was no difference in occurrence of bicavitary effusion, pleural effusion, or ascites between neoplastic and non-neoplastic causes (P= 1, P= 0.84, and P= 0.93 respectively).

Cardiac tamponade was subjectively suspected on echocardiography in 39% of dogs. A globoid shaped heart was identified in 50% of dogs on thoracic radiographs, giving a poor sensitivity for detection of pericardial effusion. Pulmonary metastases were only identified on thoracic radiographs in 33% of dogs with confirmed pulmonary metastases on necropsy or thoracotomy. There was a suspicion of a right atrial or heart base mass on thoracic radiographs in 10 of 63 dogs with masses, with a specificity of 100%. The most common electrocardiographic abnormalities included: electrical alternans (28%), sinus tachycardia (28%), dampened QRS complexes < 1 mv (19%), and ventricular arrhythmia (13%). Other less common abnormalities included: supraventricular tachycardia (3%), atrial premature complexes (2%), atrial fibrillation (2%), ST segment elevation (2%), high grade second degree atrioventricular block (1%), and right bundle branch block (1%).

Fifty nine dogs had complete necropsies performed. The metastatic rate was not different between dogs with haemangiosarcoma (56.6%), mesothelioma (62.5%), thyroid adenocarcinoma (50%), or chemodectoma (66.6%) (P = 1 for all comparisons).

- 21.6% of dogs with right atrial haemangiosarcoma also had splenic haemangiosarcoma.
- All dogs with lymphoma had metastatic disease. The most common organ of metastasis for all neoplastic cases was the lung (18 dogs; 31%).
- In dogs with cardiac haemangiosarcoma, the most common organs of metastasis included: lungs (40%), spleen (27%), liver (27%), and kidney (13%).
- The most common organs of metastasis in dogs with mesothelioma included: intrathoracic lymph nodes (63%), lungs (25%), and pleura (13%).
- In dogs with neuroendocrine tumours, 50% of metastasis involved the lungs, followed by the spleen (17%) and liver (17%).
- The most common organ of metastasis for dogs with thyroid adenocarcinoma was the pericardium (33%), followed by equal metastatic rate to the following organs: lung, transcoelomic, and myocardium (each 25%).

Survival analysis

Based on echocardiographic classification, dogs with no cardiac mass lived longer (MST 10.1 months) than dogs with echocardiographic evidence of a cardiac mass (MST 0.5 months, P= 0.0001) Dogs with a heart base mass diagnosed by echocardiography lived longer (MST 5.2 months) compared to dogs with a right atrial mass diagnosed by echocardiography (MST 0.03 months, P= 0.0002) All deaths recorded were caused by the specific aetiology of the pericardial effusion and not due to other systemic disease. Regarding specific aetiologies of pericardial effusion, dogs with non-neoplastic aetiologies lived longer (MST 24.83 months) than dogs with neoplastic aetiologies (MST 0.63 months, P < 0.0001). Dogs with haemangiosarcoma had shorter survival (MST 0.07 months) than all other neoplastic aetiologies combined (MST 5.17 months, P= 0.0001). Survival time of dogs with mesothelioma was not different from dogs with heart base masses including chemodectoma, ectopic thyroid, or non-specific aetiology (MST 6.5 months vs. MST 5.17 months respectively, P= 0.51).

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