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Cardiac Murmurs in Dogs and Cats

General Anaesthesia in Patients With a Heart Murmur

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Editor's Note

I wish everyone the best for 2016.

This edition concentrates on cardiology. Old small breed dogs have dental issues and often also concurrent heart disease. It is vital to realise that a heart murmur is not a contraindication to performing procedures under GA. Cognisance needs to be taken of the condition and the protocol adjusted in some patients - but the risk in stable animals should be minimal.

The article by Dr Zeiler elaborates on this topic. The article by Dr Leadsom clarifies heart murmurs and hopefully makes them a little simpler to prioritise. And then Dr Carter has written a neat summary on canine DCM.

I also want to draw your attention to the congress offered by the National Veterinary Clinicians Group in March. The speakers are extremely well qualified and the case study format promises to be very interesting and educational.

Regards

Liesel

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- Employee Relations within a Vet Practice - Part 1

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Madaleen Schultheiss

Part 2

Employee Relations Within A Vet Practice

Andrew Christie
BComm (Industrial Psychology)

This article is the second in a series of three which deal with maintaining positive and mutually fulfilling employee relations within a Vet Practice.

The first article dealt with the start of the employment relationship (recruitment, selection and appointment); this article explores management of the employment relationship (performance management, the grievance procedure and the creation of an HR policy) and the third will examine the termination of the employment relationship (resignations and dismissals, as well as the disciplinary procedure).

1 Introduction

Maintaining good relations with employees is crucial, not only for a positive and productive work environment, but also to limit the possibility of future disciplinary action and unfair labour practices which could result in CCMA hearings.

The first step in achieving this is recruiting and selecting the appropriate staff correctly – as outlined in the first article in this series. Maintaining an optimal employee relationship consists of 3 main components, which will be examined in this article:

1. Managing performance on an ongoing basis
2. The creation and upkeep of a Human Resource Policy and related policies
3. The establishment and application of a formal grievance procedure

2 Performance Management

Most people understand performance management to be an annual event, one that causes fear and apprehension amongst staff and creates frustration among the practice management at the additional paperwork caused.

However, performance management should be an ongoing process and can very often be less formal.

This means fewer issues need to be addressed annually, and the staff members can address performance issues on an ongoing basis.

In the first article, the job description was outlined and briefly discussed. From the job description, the sections relating to "general responsibilities" and "perfor-

mance indicators" should be used as the foundation for the performance management process.

Guidelines for performance management:

1. Performance management should be conducted on measurables; in other words, "increase sales of vet food by 100 units per month" is far preferable to "try to increase sales".
2. Where there is no way to measure, a system should be implemented so that they can be measured. For example, "improve customer interaction" could require a rating form to be completed by each customer. While this could be seen as onerous, the benefit can be seen in a story that a vet related; a particular employee was observed by him to be providing poor service on a few occasions. When this was addressed during the performance review, the employee became very upset, which in turn led to an unhappy working relationship and ultimately resulted in the employee resigning. After she left, the vet told me, it seemed that every customer demanded to know where she was as they missed her helpful and gentle service! This goes to show that if something is not measured fairly and consistently, the wrong conclusion can be reached.
3. Timeframes should be established. For example, establishing that the front area should be kept tidy could be reviewed in a week, while increasing sales could only be reviewed after a longer time period.
4. Performance management is a two-way street. It is definitely not only an opportunity for the practice owner to manage performance. Rather, the employee should be given the opportunity to explain any perceived lack of performance, as well as make requests that could improve their performance. This could be as simple as asking to be shown how an procedure works, or more lengthy and time-consuming. The employee should also be given the opportunity to air any general grievances in the workplace that they may have.

3 HR Policy and Procedures

Perhaps because vet practices are relatively small or maybe because it is perceived as unimportant, an HR policy and its related procedures are often relegated to the "worry-about-it-when-it-happens" pile.

However, having detailed HR procedures making up a comprehensive HR policy is vital for the following reasons:

1. The practice employees will know exactly what is going on
2. You will treat everyone fairly and equally
3. Protection against the CCMA.

It is important to remember that your HR policy may not make your staff worse off than anything outlined in the labour legislation. For example, you couldn't specify that your employees are only entitled to one day of annual leave per 30 days worked because the Basic Conditions of Employment Act makes provision one day per 17 days worked. However, you would be allowed to specify one day per 16 days worked, or anything less than 17.

The following are the main things that should be included in your HR policy. Remember, when it comes to this, the more detail you have, the better it is for everyone.

Recruitment and Selection

Appointments and promotions
Service/employment contracts
Employee rights and entitlements
Employment of relatives and friends
Recruitment
Termination of services
Resignation or notice
Dismissal
Staff retrenchment

4 The Grievance Procedure

In the workplace, a grievance is any dissatisfaction felt by an employee with regards to their work, place of work or conditions of employment that requires the formal attention of management.

However, it excludes wage issues that are being dealt with by trade unions and appeals against disciplinary action as these are addressed by other mechanisms.

Note that this dissatisfaction may be between employees, or between the employee and management.

Examples of grievances are:

- Rumours
- Incompatibility
- Differing attitudes and values
- Prejudice
- Poor working conditions
- Sexual harassment
- Unfair company procedures

A situation may arise where an employee may feel that they have been unfairly treated at work. Regardless of the reason, the practice has an obligation to ensure that the employee is given the opportunity to address the matter formally.

This obligation should be met by using the Grievance Procedure, a formal process that allows employees to have their conflict resolved. The critical importance of having a fair Grievance Procedure is that it should aim to resolve conflict before it grows into a much larger problem – both for the employee and for the organisation.

Note that unlike many other aspects of employee relations, a Grievance Procedure will vary from practice to practice as each entity sets up their own, based on practice structure and number of employees.

Although the Grievance Procedure will vary between practices, the following are guidelines to ensure that it is fair and accessible:

- Employees are able to lodge their grievance without fear of victimisation
- All grievances are handled with discretion, and in a way that the dignity and privacy of the employee is maintained
- Grievances are settled quickly and as effectively as possible
- The owner of the practice or his / her delegate is available to act as advisor for both parties
- The employee has the right to be assisted by a co-worker at any stage
- And, most importantly, the Grievance Procedure should not replace normal communication between people at work.

The Grievance Procedure should ONLY be used if it is clear that normal communication has not resolved the issue. For example, an employee should discuss the matter with the other person first, unless they are fearful for valid reasons.

Finally, be aware of the fact that it is easy to fall into the trap of over-using the Grievance Procedure; while it is a vital part of managing employee relations, it can be abused when employees use it to seek retribution for personal issues, or "to get back at" someone. This can trigger a lengthy chain reaction which wastes time and resources.

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FEBRUARY WEBINAR: Labour Relations - Get your questions answered by a specialist.

Andrew will deal with general questions on the topic of Labour Relations. You will have the oppotunity to send him your questions. It can be send directly to Andrew before 15 February 2016 (andrewc@acahr.com)

SPEAKER:
Andrew Christie
BComm (Industrial Psychology)

After completing his studies Andrew spend 2 years at a leading bank where he was involved in the implementation of the new labour legislation that was rolled out in 1997, as well as managerial training. He followed this with a stint at a computer-based training company where he assisted in the development of learning centers for major South African corporations. In 2000 he indulged entered into a partnership in a bookshop. Over the next five years he drove the development of that shop into a successful chain of six. In 2005 he re-entered the corporate world by forming his own consultancy, specialising in providing financial, tax and human resource services for SMME's, as well as growth management for medium corporates. The natural extension of this work into developing his clients led to Andrew opening Acacia Training, a learning and development consultancy, specialising in Financial and Human Resource training. As well as training in Europe and all over Africa, Andrew has lectured in Strategic Human Resource Development at a post-graduate level at Wits Business School.

UPCOMING WEBINARS: 23 March 19 April 24 May 28 June 26 July 23 August 27 September 25 October 22 November 13 December

ERRATUM

Two of the images in the article Ultrasonographic Assessment of the Adrenal glands in Dogs and Cats by Dr Nicki Cassel can not be clearly reproduced in print format (December 2015 edition). It can however be viewed on the webpage or VET360 APP. Please follow this link to the webpage www.vet360.vetlink.co.za/digitalmagazines. Our apologies for the inconvenience. Editor (Figure 1B: Normal ultrasound appearance of the left (A) and right (B) canine adrenal gland of a medium sized dog. They are slender elongated homogenously hypoechoic structures. Figure 3A: Markedly enlarged left adrenal gland (A) measuring 4.4x 2.8cm in dimension and exhibiting heterogeneity and multifocal areas of mineralisation.)

Top 5 Liver Conditions in Cats

Craig B. Webb, PhD, DVM, DACVIM (Small Animal)
Colorado State University

The feline liver is distinctly different from the canine liver in dysfunction, diseases, and normal metabolic function. Feline liver enzymology, while suitable for a carnivorous species, increases susceptibility to insult from drugs, toxins, metabolites, and oxidative stress. The serum biochemistry profile of a cat's liver reflects differences in enzyme sensitivity and half-life, induction or lack thereof, and a seemingly unique response to anorexia.

1. Acute neutrophilic cholangitis (suppurative cholangiohepatitis)

The WSAVA Liver Standardization Group recognised both the acute neutrophilic form (ANF) and chronic neutrophilic form (CNF) of neutrophilic cholangitis, although these may be different stages of the same condition.[1] The acute suppurative form may be mediated by enteric bacteria (eg, *Escherichia coli, Enterococcus spp, Bacteroides spp*). ANF is found primarily in young to middle-aged cats presenting with acute

The serum biochemistry profile of a cat's liver reflects differences in enzyme sensitivity and half-life, induction or lack thereof, and a seemingly unique response to anorexia.

TOP 5 Liver Presentations in Cats

1. Acute neutrophilic cholangitis (suppurative cholangiohepatitis)
2. Chronic neutrophilic cholangitis (nonsuppurative or lymphoplasmacytic)
3. Lymphocytic cholangitis (nonsuppurative cholangitis or cholangiohepatitis)
4. Triaditis
5. Hepatic lipidosis

onset anorexia, vomiting, lethargy, and diarrhoea; they may be dehydrated, febrile, and jaundiced with abdominal discomfort. Liver enzymes and total bilirubin are elevated while other biochemical abnormalities (eg, *azotaemia*, electrolyte imbalances) are nonspecific.

Ultrasonography may reveal bile duct distention (secondary to pancreatitis) or gallbladder sludge and may help guide fine-needle liver aspiration and diagnosis of cholecystitis. Laparoscopy can be used to examine liver and biliary systems, procure samples for hepatic histopathology, and guide spinal needle (22-gauge) placement for gallbladder aspiration. Because of a possible bacterial component, bile cytology and culture and sensitivity (both aerobic and anaerobic) of the hepatic parenchyma and gallbladder contents are important. Vitamin K1 at 0.5 to 1.5 mg/kg SC q12h for 3 doses is warranted before sample acquisition.

Antibiotic therapy is the cornerstone for ANF, based either on culture and sensitivity test results or an empiric choice to cover enteric organisms (usually gram-negative, anaerobes) with good hepatobiliary penetration. Cephalosporins, amoxicillin-clavulanic acid, fluoroquinolones, or combination therapy (enrofloxacin 5 mg/kg q24h, metronidazole 7.5 mg/kg q12h) are logical candidates (≥4–8 weeks). Supportive care with fluids, electrolytes, and complete and balanced nutrition should also be considered. Ursodiol at 10 to 15 mg/kg q24h and pain medications, such as buprenorphine at 0.01 to 0.03 mg/kg OTM q8h, may be indicated.

2. Chronic neutrophilic cholangitis (nonsuppurative or lymphoplasmacytic)

Histopathology reflects a shift in the predominant inflammatory cell type from neutrophils to a mixed population, including lymphocytes and plasma cells. Biliary hyperplasia and fibrosis may be present, reflecting this condition's chronic nature. Cats with CNF cholangitis are usually older than those with ANF, and signs may be intermittent with weight loss. Cholestatic liver enzyme activity (ALP and gammaglutamyltransferase [GGT]) is elevated more consistently than ALT or AST, and these cats are often jaundiced with elevated total bilirubin. Ultrasonography may demonstrate bile duct and gallbladder changes, and the liver parenchyma may be hyperechoic because of fibrosis. Histopathology is required for definitive diagnosis, and the procedures and samples are similar to ANF. Both bacterial and immune-mediated processes may be involved in the CNF.

These cats may also be treated with an extended course of antibiotics (4–8 weeks); prednisolone at 1 to 2 mg/kg q24h may be started concurrently or after several weeks of antibiotic therapy. Ursodiol is frequently used, and liver protectants and/or antioxidants (eg, SAMe, vitamin E, silymarin) should be considered.

3. Lymphocytic cholangitis (nonsuppurative cholangitis or cholangiohepatitis)

Cats with lymphocytic cholangitis (LC) present with signs and biochemical abnormalities similar to other cholangitis classes, but LC is more slowly progressive; its clinical presentation, including hepatomegaly, ascites, hyperglobulinaemia, and lymphocytosis, may help distinguish this condition. Persian cats appear overrepresented. Differentials that can mimic LC include FIP, lymphoma, and extrahepatic bile duct obstruction (rare), making histopathologic diagnosis important. Even with histopathology, the distinction between LC and lymphoma may require special assays for designating clonality and gene rearrangement.

Opinions differ as to whether prednisolone at 2 mg/kg PO q12h for 14 to 21 days then tapered or ursodiol at 15 mg/kg q24h for ≥8 weeks should be the foundation of treatment. Both treatments are often used concurrently. Because cobalamin levels are measured more frequently and sometimes low (perhaps because of concurrent GI disease), cobalamin can be added at 250 µg/cat SC q7d.

4. Triaditis

Feline cholangitis is often complicated by concurrent pancreatitis and/or inflammatory bowel disease (IBD), forming triaditis. Although there may be subtle differences, the 3 conditions cannot be distinguished by presentation alone. For example, cats with IBD are more likely to present with diarrhoea and less likely to be anorectic, and necrotizing pancreatitis may result in hyperglycaemia. Therefore, a high index of suspicion is crucial for a complete diagnostic examination that may include fPLI testing (noting its high interassay variation, spiking, and recovery), folate, cobalamin, full abdominal ultrasonography, and histopathology of multiple abdominal organs.

Triaditis treatment is more complicated than that for one disease, but many supportive efforts are similar: adequate nutrition for an anorectic cat (mirtazapine, cobalamin, esophagostomy feeding tube [E-tube]), aggressive treatment for pain and vomiting (maropitant, buprenorphine), prolonged corticosteroid treatment for CNF cholangitis and IBD, or possibly more aggressive immunomodulation (chlorambucil, 4 mg/m2 PO q2d)

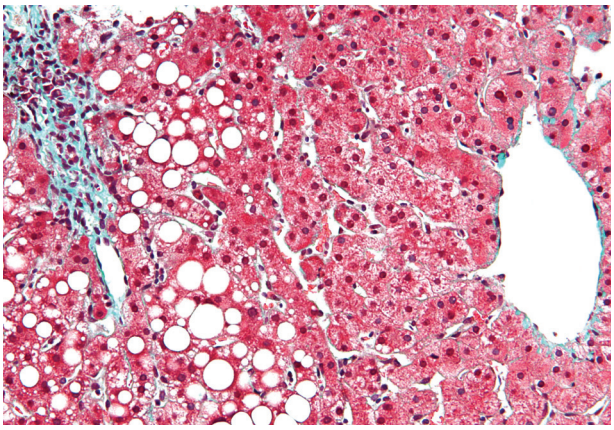

Figure 1: Micrograph of periportal hepatic lipid accumulation in the hepatocytes. Trichrome stain.

5. Feline hepatic lipidosis

Presentation of "fatty liver" or true hepatic lipidosis in cats is unique, characterized by excessive accumulation of triglycerides within hepatocytes and marked cholestasis resulting from biochemical changes induced by such factors as anorexia, insulin resistance, and arginine deficiency (Figure 1). A period of anorexia precedes presentation that may include lethargy, weight loss, jaundice, weakness, vomiting, and diarrhoea. Signs of hepatic encephalopathy (eg, excessive salivation) can be present, although rarely so. The history, marked elevation in ALP (>ALT elevation), and minimal change in GGT suggest diagnosis. Other abnormalities may be seen in CBC results (eg, poikilocytosis, Heinz bodies) and serum biochemistry profiles (eg, hyperbilirubinaemia, hypokalaemia, hypophosphataemia, low BUN). Clotting times may be prolonged, and proteins induced by vitamin K absence or antagonism (PIVKA) are often elevated. The liver is enlarged on radiographs and hyperechoic on ultrasound. Determining underlying disease is essential, as hepatic lipidosis is frequently secondary to another condition, such as pancreatitis, intestinal disease, cholangitis, neoplasia, or infectious disease.

Hepatic lipidosis is frequently secondary to another condition, such as pancreatitis, intestinal disease, cholangitis, neoplasia, or infectious disease

Hepatic lipidosis can be treated primarily through nutrition, frequently requiring an E-tube. Appetite stimulants (eg, mirtazapine) may be contraindicated in cats with hepatic lipidosis because of unknown metabolism variability by the liver. A nasogastric tube may be used while achieving IV fluid support and electrolyte correction during critical care (with attention to potassium, phosphorus, and magnesium), but E-tube use is more common. The cat should be treated with vitamin K1 at 0.5 to 1.5 mg/kg SC q12h for 24 to 48 hours before E-tube placement, fine-needle aspiration of the liver, or obtaining hepatic biopsy specimens.

A high-protein, low-carbohydrate diet can be blended and small, frequent feedings initiated, gradually increasing to the cat's total daily requirement (usually over 4–5 days). Nonspecific supportive care may include maropitant at 1 mg/kg q24h or ondansetron at 0.22 to 0.5 mg/kg q12h (should vomiting occur), continued vitamin K1 at 2.5 mg/cat q24h, cobalamin at 250 µg/cat SC q7d, L-carnitine at 250 mg/cat q24h, and SAMe at 20 mg/kg q24h (increasing the dose by 50% if crushed and given through a feeding tube).

Nonspecific clinical signs and biochemical changes highlight the importance of a diligent diagnostic examination, remembering that although the liver may be the center of the disease process, it might also be only one component of what is making the cat ill.

ANF = acute neutrophilic form, CNF = chronic neutrophilic form, E-tube = esophagostomy feeding tube, fPLI = feline pancreatic lipase immunoreactivity, GGT = gamma-glutamyltransferase, IBD = inflammatory bowel disease, LC = lymphocytic cholangitis, OTM = oral transmucosal

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Cardiac Murmurs in Dogs and Cats

Causes, Diagnosis and Significance

Dr Russell Leadsom
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A murmur is an audible sound produced by vibrations of cardiovascular structures as a result of disturbed or turbulent blood flow replacing normal laminar flow.

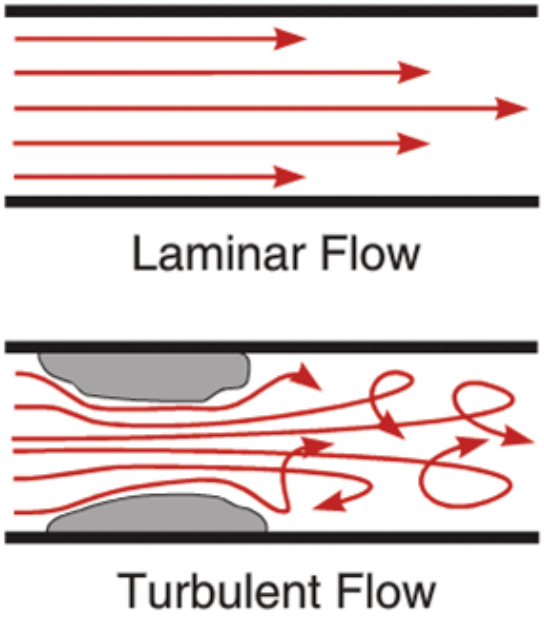

Fig 1. Laminar and turbulent blood flow

There is a critical velocity at which laminar flow becomes turbulent, it is expressed by Reynold`s number and is dependent on the velocity of the blood, viscocity and the vessel diameter (fig. 1).

Diagnosis needs good auscultatory skills that require patience, time and practice. A quality stethoscope and a quiet environment are vital.

Murmurs may be masked by obesity, pleural and pericardial effusions, and respiratory sounds. Tachypnoea breathing, panting may be mistaken for murmurs.

Murmurs are described by their point of maximum intensity (PMI) (figures 2 & 3) noting the location, intercostal space, and valve over which the murmur is loudest; intensity on a 1/6 to 6/6 scale; radiation over the thorax and neck; timing within the cardiac cycle - systolic, diastolic or continuous; and quality such as "soft", "musical" or "harsh".

Further descriptive terms include the timing within the systole or diastole e.g. early or late systole. The fast heart rates of dogs and cats usually make this difficult without phonocardiography.

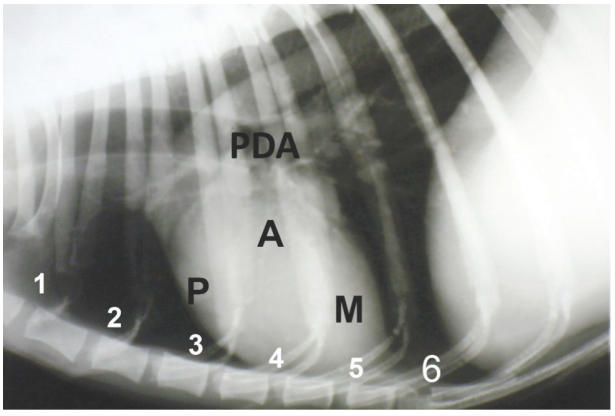

Fig 2. Left side PMI's. (P = pulmonic valve, A = aortic valve, M = mitral valve, PDA = patent ductus arteriosus).

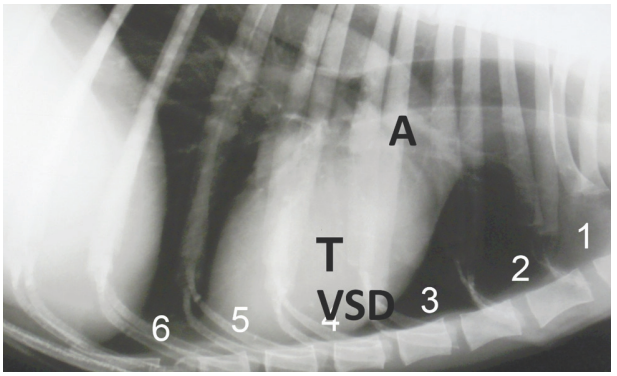

Fig 3. Right side PMI's. Right side (T = tricuspid valve, A =aortic valve, VSD = ventricular septal defect)

Intensity of murmur	
Grade 1	Very soft and localized, and is initially inaudible.
Grade 2	Soft, but heard immediately and is localized.
Grade 3	Soft and radiates to more than one location.
Grade 4	Moderate-intensity to loud that radiates, but has no precordial thrill (a palpable `buzzing` sensation on the hand)
Grade 5	Loud and accompanied by a precordial thrill.
Grade 6	Loud with a precordial thrill and is still audible when the stethoscope is lifted off the chest wall.

As a rule low grade murmurs (< 2/6) are mild and probably not haemodynamically important. There are exceptions to this, notably a ventral septal defect (VSD) – see below.

Again, as a general rule, an increasing murmur grade correlates with increasing cardiac pathology.

CAUSES

1. High velocity blood flow

These murmurs have normal valves, with no cardiac disease and are known as physiological, functional or flow murmurs. Examples include exercise, fever, hypotension and hyperthyroidism. Anaemia also produces increased flow velocity with audible murmurs (haemic) when the haematocrit is less than 25% in dogs and 20% in cats; conversely, polcythaemia may mask a murmur. Young animals often present with low grade (< 3/6) soft systolic "innocent" flow murmurs that usually disappear by 6 months of age. Lower haematocrit and faster heart rates are causes. They are often intermittent, even during the course of auscultation.

Bradycardias result in increased stroke volume, as will an `athletic` heart. These murmurs are usually soft, early systolic and <3/6. Certain breeds, notably boxers, are often ascribed as having a normal but relatively narrow aortic outflow tract which results in increased velocity. These murmurs are often more readily detectable in the thoracic inlet area.

2. Congenital cardiac defects.

Ventricular outflow obstruction (ejection) murmurs are systolic, harsh, and described as having a crescendo-decrescendo configuration with a PMI in the heart base area. Causes include pulmonic stenosis, sub-aortic stenosis and bacterial endocarditis.

- Regurgitant murmurs from abnormal (dysplastic) mitral and tricuspid valves are systolic with PMIs over the relative valve area, and described as having a plateau configuration.
- Abnormal congenital conduits between cardiac chambers or major vessels are frequently encountered, and include ventricular septal defects (VSD), and patent ductus arteriosus (PDA).
- Other defects include atrial septal defects and aorticopulmonary septal defect.

SPECIFIC MURMURS DUE TO CONGENITAL DEFECTS

A knowledge of breed predispositions for both congenital and acquired conditions, the age of the animal, sex, and murmur description will often lead to a presumptive diagnosis. Radiography will help confirm many cases, but definitive diagnosis requires echocardiography and Doppler studies.

1. Sub-aortic stenosis (SAS)

This outflow murmur may be detected with an equal or even greater intensity on the right heart base area, (figs. 2, 3) but unlike the murmur of a pulmonic stenosis. It will frequently radiate up the carotid arteries, and is occasionally audible on the cranium. A precordial palpable thrill is usually detected. It is more common in larger breeds, and is a defect that continues to develop over the first few months of life, even up to 12 months, consequently it may be low grade when first detected, and then develop over several months. (fig.4) It is occasionally encountered in cats

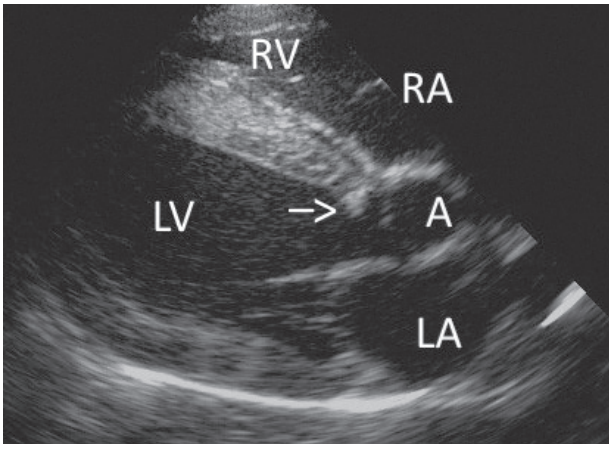

Fig 4. Echocardiographic image of a canine heart. Right parasternal long-axis view. (RV = right ventricle, RA = right atrium, A =aorta, LA = left atrium, LV = left ventricle. Arrow points to the ridge of sub-aortic obstructing tissue.

2. Pulmonic stenosis (PS)

This is more common in smaller breed dogs, and English Bulldogs. It is best heard high in the left heart base area (fig. 2) and if severe, may be accompanied by a palpable thrill. Unlike the murmur of SAS it does not usually radiate widely, and may be accompanied by jugular vein pulses.

3. Ventricular septal defect (VSD)

This is one of the most frequent cardiac defect encountered in cats, it is less common in dogs. The

murmur is loudest along the right sternal border (fig. 2). A very large shunt volume will result in a functional flow murmur across the pulmonic valve and a left heart base ejection murmur. Unlike other murmurs, a small defect results in higher velocities into the right ventricle producing more intense (louder) murmurs. Conversely very large defects may produce a barely audible sound.

4. Mitral and tricuspid valve dysplasia

They are most common in the larger dog breeds, and are not uncommon in cats. Auscultatory findings are similar to endocardiosis (see below) with PMIs over the affected valve area (figs. 2,3).

5. Patent ductus arteriosus (PDA)

Female dogs are about 3 times more frequently affected. Smaller breeds (eg poodles, collies, pomeranians, and Maltese) are more predisposed. It is occasionally encountered in cats.

The murmur is loudest high in the left heart base (fig. 2), and is easily overlooked if the stethoscope is not correctly placed. A palpable thrill is often detected. The murmur is described as `continuous/machinery`, but will wax and wane with its peak at the end of systole and early diastole. It frequently radiates widely over the left and right heart base areas. In cats the PMI is typically more caudoventral, and the diastolic component is often more difficult to detect.

ACQUIRED CARDIAC LESIONS

1. Mitral and tricuspid regurgitation

Myxomatous degeneration of the mitral valve (endocardiosis) accounts for most murmurs encountered in middle-age and older small breed dogs. Approximately 30% will have accompanying tricuspid valve involvement. It is unusual for the tricuspid valve to be solely affected. Occasionally a loud (4/6) murmur with a high frequency is heard and is described as having a whooping, musical or seagull character; this murmur can be alarming but usually represents a small orifice with a high velocity regurgitant jet and a minimal volume overload response; high frequency vibrations of the chordae tendinae are involved in the generation of this murmur. Dogs with dilated cardiomyopathy will often present with a soft low-grade murmur over the mitral valve area due to stretching and distortion of the valve annulus.

2. Endocarditis

Infectious endocarditis is *recognised* in both dogs and cats, mostly involving the aortic valve, mitral valve or both, with large breed male dogs more affected. A high index of suspicion is required for diagnoses, the murmur will be recent and often changes in character and even position over a short period of time. PMIs will be over the affected valve(s). Predisposing factors include pre-existing endocardiosis and congenital defects including SAS and MD.

CATS

Cats frequently present with murmurs. It is sometimes stated that 30% of all cats presented to clinics will have a detectable murmur. In a recent investigation of 57 apparently healthy cats with murmurs, cardiac pathology was found in over 80%. (Dirven 2010) Atrioventricular valve dysplasia, aortic stenosis, and VSD are common congenital defects encountered.

Unlike dogs, the PMI of many murmurs is along the right and left sternal area due to the sternum acting as an acoustic enhancer

Cats frequently present with an ejection murmur that is `dynamic` in nature i.e. it increases with the heart rate and is due to certain forms of hypertrophic cardiomyopathy (HCM) called hypertrophic obstructive cardiomyopathy (HOCM) and also systolic anterior motion (SAM) of the mitral septal leaflet that leads to both aortic outflow stenosis and mitral valve regurgitation (fig. 5).

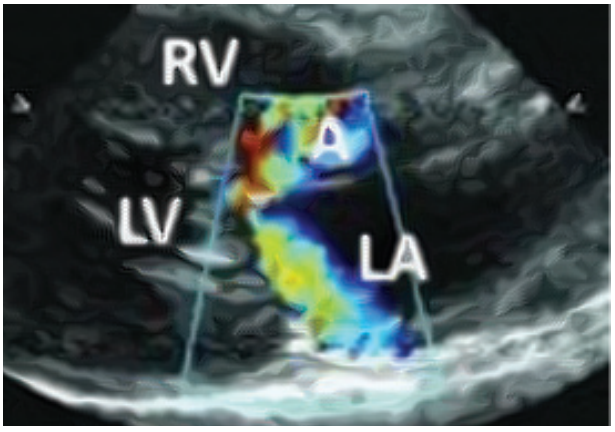

Fig 5. Echocardiographic image of a feline heart, from right parasternal long-axis. Colour Doppler shows turbulent flows into aorta and left atrium due to SAM.

These dynamic murmurs do require an index of suspicion necessitating a protracted period of auscultation waiting for the cat to relax upon which the murmur may disappear completely, it is then confirmed by gentle stimulation/stress such as shaking the cat or opening a door and the faster heart rate will cause the murmur to reappear.

- Older cats often have aortic outflow physiological murmurs due to age related anatomical changes to the aorta ("redundant aorta") and systemic hypertension.
- Iatrogenic murmurs are readily produced in cats by applying excessive pressure to the stethoscope.
- Diastolic murmurs are unusual, more difficult to identify, always pathological, and beyond the scope of this brief description of murmurs.

References available on www.vet360.vetlink.co.za

Can Milk Thistle be Used to Treat Liver Disease?

Hackett ES, Twedt DC, Gustafson DL. Milk thistle and its derivative compounds: a review of opportunities for treatment of liver disease. J Vet Intern Med2013;27(1):10-16.

Summarised by Jennifer L. Garcia, DVM, DACVIM

Why they did it

Natural remedies abound for treating common diseases in people and pets, but what exact role they should play remains undetermined. This study looked specifically at the use of milk thistle for helping pets with liver disease.

What they did

In this article, the authors review the pharmacokinetics and potential treatment applications of *Silybum marianum*, or milk thistle. Silibinin is the predominant compound in silymarin and the compound most often reported in milk thistle formulations.

What they found

Milk thistle has poor oral bioavailability because of extensive conjugation and biliary excretion. It is often complexed with phosphatidylcholine to improve oral bioavailability.

Silibinin is thought to provide benefit via free radical scavenging and inhibition of lipid peroxidation as well as through anti-inflammatory effects associated with inflammatory cytokine suppression and leukotriene formation inhibition. Silibinin has also demonstrated antifibrotic properties by interrupting myofibroblast formation and by limiting fibrous tissue production. In rats, silibinin has been shown to enhance protein synthesis that is necessary for hepatic regeneration and repair after injury. Studies in people and rats or mice have demonstrated a wide margin of safety for this drug.

The authors discuss the current therapeutic applications of this drug based on human and animal data:

Intoxication

- It has been shown to be hepatoprotective in dogs after Amanita phalloidesintoxication by limiting oxidation and inhibiting uptake of phalloidin toxin into hepatocytes.
- It has been shown to prevent an increase in hepatic enzyme activity in rats exposed to carbon tetrachloride, acetaminophen, and arsenic as well as in studies involving radiation exposure and doxorubicin use.

Hepatitis

- It has been used in people with alcoholic liver disease to decrease ethanol-induced production of free radicals.
- For nonalcoholic fatty liver disease, it is thought to limit glutathione depletion and peroxide production and to prevent hepatic mitochondrial dysfunction.

Cirrhosis

- It is thought to improve antioxidant status, decrease collagen production, and limit glutathione depletion.

Take-home message

There are no randomized clinical trials evaluating milk thistle use in clinically affected animals with liver disease, but studies in animal models thus far are promising. It is important to remember that this drug should not be used as the sole therapy for patients with liver disease and that there is currently limited pharmacokinetic and pharmacodynamic information about milk thistle in dogs and cats.

Physiological Heart Murmurs are More Common in Our Veterinary Patients than We May Think

Drut A, Ribas T, Floch F, et al. Prevalence of physiological heart murmurs in a population of 95 healthy young dogs. J Small Anim Pract. 2015;56(2):112-118.

Summarised by Jennifer L. Garcia, DVM, DACVIM

Murmurs may not only be limited to specific breeds or growing dogs.

Why they did it

Researchers sought to determine the prevalence of physiological heart murmurs (i.e. a murmur in the absence of a structural abnormality of the heart and great vessels) in young, apparently healthy dogs between the ages of 1 and 5 years and of various breeds.

What they did

Between November 2012 and July 2013, researchers enrolled 95 dogs (48 males and 47 females) with heart murmurs that were identified at the time of routine vaccination. All dogs were in apparent good health, were between 1 and 5 years of age (median age 32 months) and represented 30 breeds.

Cardiac auscultation for each dog was performed by at least three examiners—a board-certified internist, a cardiology resident and an internal medicine resident—all of whom were blinded to the others' findings. When noted, heart murmurs were graded as follows:

- Grade I/VI: Faint murmur; only heard with particular effort
- Grade II/VI: Faint murmur; clearly heard by an experienced examiner
- Grade III/VI: Moderately loud; easily auscultated
- Grade IV/VI: Loud murmur with no palpable thrill
- Grade V/VI: Very loud murmur with thrill; inaudible after removing stethoscope from chest wall
- Grade VI/VI:Very loud murmur; thrill still audible after removing stethoscope from chest wall

Dogs with arrhythmias or any clinical or biologic signs of disease were excluded. Indirect blood pressure evaluation was performed in all dogs, and the average of five consistent measurements was recorded. A patient with a systolic pressure > 180 mm Hg was considered hypertensive. A urinalysis, complete blood count, and serum chemistry profile were also performed in all dogs.

All dogs underwent a standard, unsedated, echocardiographic examination, and the ultrasonographer was blinded to the auscultation findings.

What they found

The researchers identified murmurs in 22 of 95 dogs (23%), and all were systolic, detected primarily over the left heart base and ranged between Grade I to III/VI. Ten of these murmurs were identified only by the board-certified internist and were Grade I to II/VI. All three investigators reported the same findings in 69% of dogs, and interobserver agreement was found to be fair to moderate; "[h]owever, the influence of the investigator on the detection of heart murmurs did not reach statistical significance (P = 0.44)."

No significant difference in sex, age, body weight or breed was present between dogs with murmurs and those without.

Eleven of the 22 dogs with a murmur had echocardiographic abnormalities including mild mitral regurgitation (n = 9), subaortic stenosis (n= 2) and pulmonic stenosis (n= 1). Given the lack of consensus on what is considered abnormal with respect to peak aortic flow velocity and tricuspid regurgitant flow velocity, the authors used both "strict" and "flexible" criteria to determine a diagnosis of subaortic stenosis or pulmonary arterial hypertension. In the absence of other echocardiographic findings and using the strict criteria, mild subaortic stenosis was diagnosed in four of the 22 dogs, and pulmonary arterial hypertension was diagnosed in one dog. All of these dogs, however, were considered to have physiological heart murmurs if the flexible criteria were applied. This resulted in a prevalence of physiological heart murmurs in this population between 6% and 12% depending on the echocardiographic criteria used, with 27% to 50% of the 22 murmurs considered physiological.

Take-home message

Physiological heart murmurs may not be limited to specific breeds of dogs or growing dogs but may also be found in young, healthy dogs. But a lack of consistent criteria for the diagnosis of subaortic stenosis and pulmonary arterial hypertension makes accurate assessment of the prevalence of physiological heart murmurs difficult.

SDMA Pinpointed as Biomarker for Feline Renal disease

References Summarised by
Jennifer L. Garcia, DVM, DACVIM

Does this form of testing hold the key to diagnosing deterioration of the kidney in cats earlier?

Why they did it

Chronic kidney disease in cats is often not *recognised* in the clinical setting until there is evidence of azotaemia and loss of urine concentrating ability. Identification of sensitive serum biomarkers, which would allow early identification of cats with renal disease, would facilitate early intervention and improve patient care. Symmetric dimethylarginine (SDMA) has been found to be an accurate surrogate for estimated glomerular filtration rate (GFR) in people and a more sensitive biomarker of renal function. These studies sought to evaluate the utility of this biomarker in the setting of feline renal disease.

What they did

Three studies have evaluated the possible role of SDMA as a biomarker for feline renal disease.

In the first article ("Comparison of serum concentrations of symmetric dimethylarginine and creatinine as kidney function biomarkers in cats with chronic kidney disease"), the authors retrospectively reviewed SDMA and serum creatinine concentrations in 15 cats with azotaemia for ≥ three months, four nonazotaemic cats with a greater than 30% decrease in GFR from normal, and two nonazotaemic cats with calcium oxalate kidney stones. Data from 21 healthy geriatric cats were also evaluated. Over a six-month period prior to enrollment, these cats had three normal GFR test results, three normal serum creatinine concentrations and three urine specific gravity assessments > 1.040.

In the second article ("Comparison of serum concentrations of symmetric dimethylarginine and creatinine as kidney function biomarkers in healthy geriatric cats fed reduced protein foods enriched with fish oil, L-carnitine, and medium-chain triglycerides"), the authors fed control food or one of two experimental diets supplemented with fatty acids to 32 healthy cats. Cats were fed these diets for six months, and serum chemistry profiles, including SDMA and serum creatinine concentrations, GFR, and metabolic profiles, were performed at baseline and at 1.5, three and six months.

In the third article ("Relationship between serum symmetric dimethylarginine concentration and glomerular filtration rate in cats"), the authors sought to determine whether SDMA concentrations would rise as expected in cats with reduced GFR. GFR was measured by using iohexal clearance in 10 client-owned cats. All cats were > 11 years of age, and both azotaemic and nonazotaemic cats were included.

What they found

In the first article, the authors found that both SDMA and serum creatinine concentrations correlated well to changes in GFR. However, SDMA was elevated before the serum creatinine concentration in 81% of cats (17 of the 21 subjects) by a mean of 17 months (range, 1.5 to 48 months). SDMA was elevated in all nonazotaemic cats with a subnormal GFR. The SDMA and serum creatinine concentrations remained within the reference range for all the healthy geriatric cats.

In the second article, the authors found that diets supplemented with fatty acids did not result in any changes to renal biomarkers over the six-month period. However, they did find that SDMA appeared to be a more sensitive marker of renal dysfunction over that time frame, particularly in older cats with decreased lean body mass. The authors noted that serum creatinine concentrations were positively correlated with total lean mass and that as total lean mass declined with age, serum creatinine concentrations declined as well. Conversely, SDMA concentrations did not correlate in the same manner with lean body mass; these concentrations increased in cats with declining GFR and body mass.

In the final article, the researchers found a reciprocal linear relationship between the decline in GFR and elevations in SDMA concentration. And as expected, a direct linear relationship was also noted between SDMA and serum creatinine concentrations.

Take-home message

These results suggest that SDMA is a sensitive biomarker for early detection of feline kidney disease. Further studies will be needed to determine if early intervention in these cases will result in improved outcomes.

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Effects of Dietary Protein on the Kidneys of Normal Cats and Those With CKD

Liesel van der Merwe BVSC MMedVet (Med)Small Animals

Cats are obligate carnivores and require high dietary protein levels and provision of the amino acids, arginine taurine, methionine and cysteine. Protein has also been demonstrated to be an integral component of glucose and lipid metabolism as well as a vital component in the management of diabetes mellitus, obesity and hepatic lipidosis in this species. [1]

Due to increased awareness of the benefits of feeding felines higher protein diets, these diets are being fed with greater frequency. There is thus a need to evaluate the biochemical effects of these increased protein levels. A study by Backlund (2011) evaluated 23 healthy spayed female cats fed, sequentially, high protein or low protein diet (random single cross over design). Each cat thus acted as its own control. Statistically significant changes in serum values were seen with BUN (urea), creatinine, phosphorous, albumin , ALT and USG within 1 month of starting the diet.[1]

In high protein diets, meat is typically the protein source, causing increased creatine and creatinine intake which can result in mildly increased serum creatinine concentrations. Plasma creatinine is the end product of protein degradation and is dependent on the total muscle mass of the individual as the primary source is the skeletal muscle. Creatinine is freely filtered through the glomerulus and is neither reabsorbed nor secreted, making it a rough estimate for GFR. Extra-renal factors which can influence creatinine are age – young animals have higher levels, diet, and exercise (greyhounds – increased levels.[1]

Creatinine concentrations in these cats were significantly ($p<.05$) lower when they were fed the HPdiet - which was unexpected. It is theorised that this may be due to a higher GFR which is caused by the increased dietary protein content.[1] The average pre-trial and washout creatinine level was 154 nmol/L. In cats fed the high protein diet the creatinine was 131 nmol/L and 139 nmol/L in cats fed the lower protein diet.
The majority of urea is synthesised by the liver which metabolises ammonia , a waste product of protein degradation on the GIT. Urea is not a good estimation of GFR as a large portion of that which is filtered is reabsorbed in the tubules and there are also pre-renal causes of increases: haemorrhage into the small intestine providing protein substrate, haemolysis, dehydration and high protein diets. It was thus expected that the urea was significantly higher in cats fed the high protein diet (approximately 50% above the baseline values).[1] The pre feeding urea in the cats was approximately 5.6 mmol/L and post high protein diet urea levels averaged 8.16 mmol/L and those in the low protein diet group were 6.8 mmol/L .

Serum phosphorous levels are dependent on the balance between dietary intake and renal excretion. The phosphorous levels were also lower ($p<0.05$) in the high protein group, in contrast to expectations. The increased GFR would also explain this as phosphorous is primarily controlled by renal excretion.[1]

Dietary protein is not a contributor to either initiation nor progression of chronic renal disease in dogs and cats. High protein feeding can however, exacerbate clinical signs by worsening azotaemia in patients with existing renal failure.This occurs because the loss of renal function leads to an accumulation of nitrogenous and non-nitrogenous end products of protein metabolism in the blood, not because of damage to the renal tissue.

Protein restriction in stage 3 naturally occurring kidney failure has a positive long term effect on outcome. Cats with CKD receiving renal diets instead of normal food survived significantly longer is several studies published: 20.8 months versus 8.7 months (Elliot *et al* 2000) and 16 months versus 7 months (Platinga *et al* 2005).[2]

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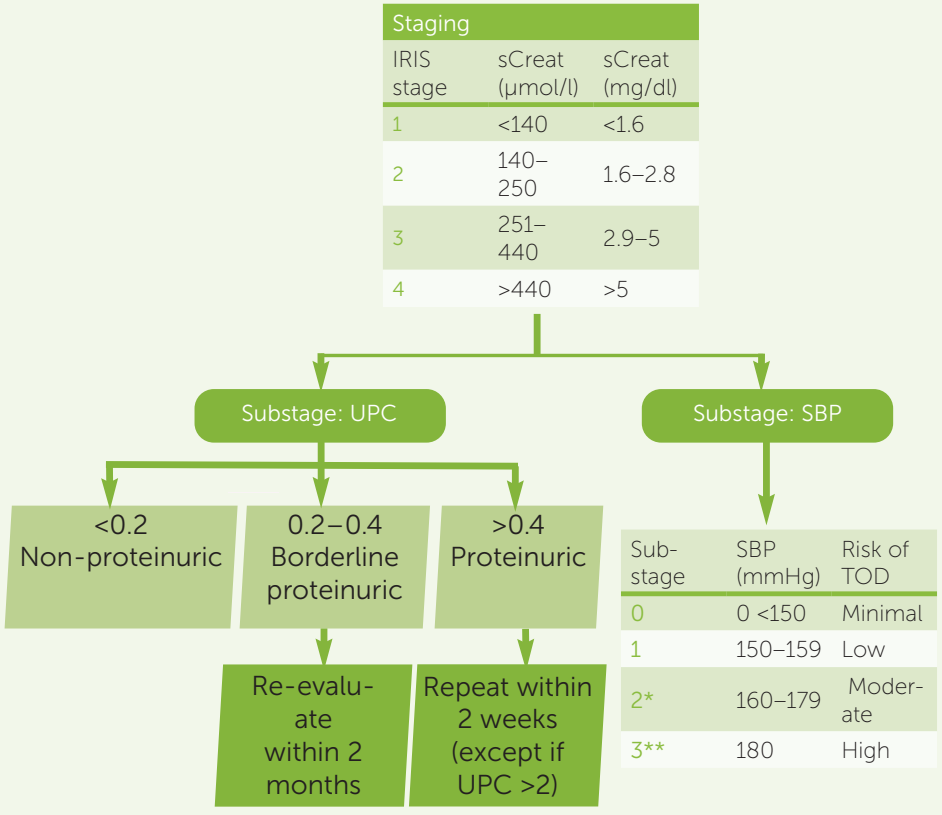
Renal diets are restricted in protein, phosphorus and sodium and supplemented with potassium, omega-3 fatty acids, B vitamins and fat content, and are alkalinising. It is unknown which alterations are responsible for survival benefits, although studies in experimental models support phosphate restriction and essential fatty acid (EFA) supplementation as potential mechanisms.[2]

Of all the CKD treatments used to date, dietary modification has the most positive long-term effect on outcome.[2] Additionally, a randomised, controlled, clinical trial (RCCT) compared feeding maintenance diets with renal diets in spontaneous CKD stages 2 and 3. Cats fed the renal diet developed fewer uraemic episodes (0% versus 23%) and none died from renal disease. [2] See Figure 1 for IRIS staging overview.

Criteria for the timing of initiation of dietary modification have been empirical. It is generally accepted that uraemic patients will benefit from protein and phosphate restriction.[3] Results of a study by Ross (2006), where 45 cats were divided into 2 grouped and fed either maintenance diet, or a renal diet, show that there is a benefit to dietary modification in the management of stage 2-3 CKD.[3] The study also found that affected cats did not show a slow progressive increase in creatinine over the 24 months of the study, but rather developed acute uraemic crises (6/45 cats). All 6 affected cats were in the maintenance diet group rather than the renal diet group. Only 3 cats survived the uraemic episode and the increased creatinine levels were sustained after the crisis.[3]

There is no evidence supporting dietary modification in stage 1 CKD, although, in the authors' experience, introducing a dietary change in a clinically well cat improves diet acceptance.[2] Over 90% of cats with CKD accepted renal diets when a very gradual transition was used. Attempting changes in sick, hospitalised, anxious patients can result in food aversion. Dietary modification should not be attempted until patients are well and discharged from hospital. There will always be some cats defiant of diet change.

Although home-prepared renal diets are attractive to some owners, dietary assessment identified numerous nutritional inadequacies.[2] Therefore, in cats refusing renal diets, use of senior diets with phosphate binding agents (PBAs) if hyperphosphataemia is present, while not ideal, may be better than provision of maintenance diets alone.[2]



The CKD stage is based on serum creatinine concentration, assessed on at least two occasions, and further substaging is based on proteinuria, assessed by UPC and blood pressure measurement. Only stable patients can be staged and blood should be drawn after a 12 hour fast, where access to drinking water is allowed

Figure 1: IRIS Staging Overview.

References available on www.vet360.vetlink.co.za

Canine Idiopathic Dilated Cardiomyopathy

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FOURWAYS VET HOSPITAL

Cardiomyopathies are diseases of the myocardium associated with cardiac dysfunction and dilated cardiomyopathy is characterised by dilation and impaired contraction of the left ventricle or both ventricles. Dilated cardiomyopathy (DCM) is the most common form of cardiomyopathy in dogs and the second most common form of acquired heart disease in dogs. Diagnosis, pathophysiology and current treatment recommendations will be discussed in this article.

Signalment

DCM tends to be a disease of large breed dogs with a higher prevalence in male dogs. A genetic predisposition to the disease has been identified in Dobermans, Irish Wolfhounds, Great Danes, New Foundlands, Spaniels and Portuguese Water Dogs. The disease in the American cocker Spaniels has been frequently characterised as a taurine deficient condition with supplementation markedly improving the outcome.

Pathophysiology

DCM is characterised by ventricular enlargement, diastolic and systolic dysfunction and resulting congestive heart failure. The symptomatic phase is preceded by an asymptomatic phase where a number of compensatory mechanisms work in concert to maintain cardiac output and prevent the development of congestive heart failure. These compensatory mechanisms include the rennin-angiotensin aldosterone-system (RAAS), catecholamine's and other vasoactive substances. These compensatory mechanisms are beneficial in the short to medium term but have deleterious effects in the long term. Increasing blood pressure with RAAS increases the cardiac work load as does increased plasma catecholamine's by increasing the heart rate.

The end result may increase the rate of myocardial cell death. These deleterious compensatory mechanisms are balanced by vasodilator, diuretic and natriuretic factors such as the natriuretic peptides.

In symptomatic DCM plasma catecholamines have been found to be significantly increased in both humans and dogs. Increased catecholamine activity results in down 1-receptors in the myocardium and decreased β-adrenergic responsiveness. This results is impairment of systolic and diastolic functions and exacerbation of arrhythmias.

It is widely accepted that congestive heart failure is associated with an increased RAAS activity. This results in vasoconstriction, sodium retention, water retention and an increase in thirst. A local RAAS in the myocardium is most likely the most significant area of angiotensin II production. Increased local RAAS may lead to myocyte hypertrophy, necrosis and fibrosis.

Clinical Findings

Dogs with DCM can be divided into those in the preclinical stage where heart disease is evident but there are no out ward signs of heart disease. In the clinical stage dogs will exhibit some or all of the following signs

- Breathlessness
- Dyspnoea
- Cough
- Exercise intolerance
- Anorexia
- Weight loss
- Syncope
- Abdominal distension
- Polydypsia

Clinical examination may reveal tachypnoea, crackles, tachycardia, pulse deficits, arrhythmias and in some dogs a systolic murmur, ascites, pale mucus membranes, weight loss and muscle wasting. The arrhythmia is described as irregularly irregular - and has no discernable pattern. A pulse deficit occurs when the pulse is lower than the heart rate. This occurs when

the ventricles contract prematurely and the chamber is not yet filled with blood - thus there is no bolus of blood being pushed into the arterial system and thus no pulse.

Large breed dogs are also predisposed to developing pericardial effusion. These dogs however have good body condition as it is not a chronic disease. In these patients the heart rate may be rapid, but is regular and there is no pulse deficit. Auscultation reveals softer heart sounds.

The right heart is compromised first as the myocardium is thinner and thus these animals develop ascites. The radiographs will also show cardiomegaly and careful evaluation is required to differentiate from DCM. Ultimately ultrasound is required to confirm a diagnosis.

Diagnosis of DCM

Radiographic findings in dogs with clinical DCM include cardiomegaly, venous congestion, prominent left atrium, pulmonary oedema, pleural effusion and sometimes ascites.

Echocardiography is required to appreciate increased chamber size and myocardial failure. It must also be borne in mind the DCM is not the only cause of increased chamber size and myocardial failure. Doppler echocardiography is required to exclude some acquired and congenital heart diseases that result in ventricular dilation and reduced myocardial function. For example patent ductus arteriosus results in volume over load of the left heart with ventricular dilation. Other differentials include tachycardia induced cardiomyopathy, doxorubicin induced cardiac damage, taurine/carnitine deficiency and systemic diseases such as hypothyroidism.

The echocardiogram should be carried out in lateral recumbency according to the standard views. 2D, M-mode and Doppler evaluation should be performed to evaluate the heart correctly. A full description of the echocardiographic evaluations is beyond the scope of this article.

The following criteria are used to make a diagnosis of DCM

- Left ventricular dilation (especially in systole). There are breed and weight tables for normal values.
- Depressed systolic function. Measurement of fractional shortening and ejection fractions. Fractional shortening of less than 20-25%. Fractional shortening does vary slightly between different breeds and there are tables available listing the expected fractional shortening for different breeds. Echocardiography of healthy Dobermans recently found that the average fractional shortening was 26% using a short axis view, and 22.5% using a long axis view. In other breeds a fractional shortening of 25% or less in the short axis view is considered abnormal. This either indicates that a large percentage of healthy Dobermans have occult DCM or that the Doberman heart at baseline is not comparable to that of most breeds.
- Left ventricular sphericity. A ratio of less than 1.65
- Left or bi-atrial enlargement.
- Increased E point to septal separation (EPSS) >6.5mm.
- Arrhythmias such as atrial fibrillation and ventricular arrhythmias. Atrial fibrillation more common in Irish Wolf Hounds and ventricular arrhythmias more common in Dobermans and Boxers.

Holter monitors can be used to look of sub-clinical myocardial disease which may present with episodic arrhythmias.

Treatment of DCM

When treating a patient with DCM we need to address the underlying myocardial dysfunction, the harmful counter regulatory mechanisms, pulmonary oedema, arrhythmias, ascites and possible pleural effusion. Supplementation with taurine, carnitine and free fatty acids can also be considered.

Before starting a patient on treatment it must be borne in mind that the median survival time from diagnosis is about 19 weeks. The best single variable for assessing prognosis is the left ventricular diameter at end systole. Other variables negatively associated with survival include the following

- Presence of pulmonary oedema
- Presence of ventricular premature complexes
- Higher plasma creatinine levels
- Lower plasma protein
- Great Dane breed.

Standard therapy includes an Angiotensin converting enzyme inhibitors (ACE inhibitor), Pimobendan and furosemide.

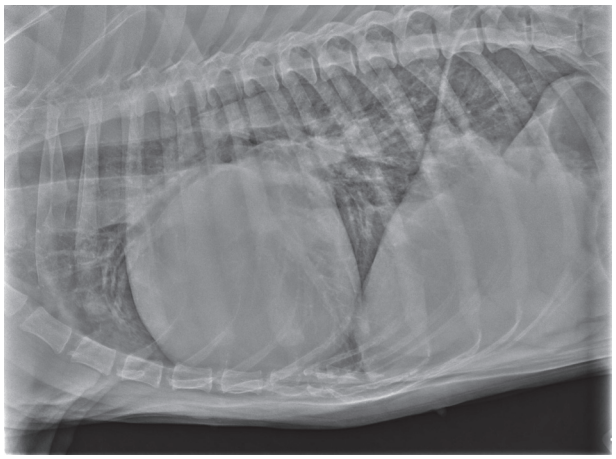

Figure 1: Lateral radiographs of dog with dilated cardiomyopathy

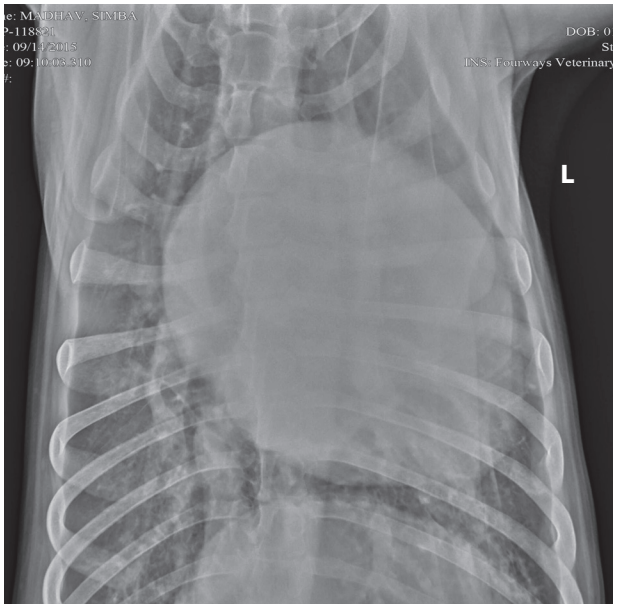

Figure 2: DV radiograph of dog with dilated cardiomyopathy

Pimobendan requires a special mention as it has demonstrated a marked increase in survival time compared to placebo treated groups. A study by Luis Fuentes and colleagues looking at Dobermans and Cocker spaniels demonstrated a median survival of 329 days in the Pimobendan groups compared to 50 days in the placebo group. A similar study by O'Grady and colleagues demonstrated a survival time of 130.5 days in the Pimobendan group compared to 14 days in the placebo group.

The Bench study looked at the use of Pimobendan to prevent the onset of congestive heart failure in Dobermans diagnosed with pre-clinical DCM. The Study found that Pimobendan significantly prolonged (by 9 months) the median time to onset of heart failure or sudden death in Dobermans with pre-clinical DCM. This drug has now been registered for this use in Dobermans.

Pimobendan acts by inhibiting phosphodiesterase III and by increasing the calcium sensitivity of the cardiac myofibrils to calcium. The effect is increased contractility without any increase in myocardial oxygen consumption. Pimobendan results in a reduction of pulmonary capillary wedge pressure, an increase in cardiac output and an increase in stroke volume

Beyond standard therapy other therapies addressing atrial fibrillation such as digoxin can be considered. Digoxin also has a weak positive inotropic effect to cause increased cardiac output however its main benefit is from its central effect of promoting vagal tone and thus slowing SA discharge and AV conduction rates. This will decrease the heart rate thus reducing myocardial oxygen consumption. It is vital to initiate therapy at the appropriate dosage and digitalise the patient. Digoxin should be dosed on lean body mass at 0.22mg/m2, po, bid, and the dose reduced in animals with ascites.

Due to the variable half- life in dogs accurate dosing is difficult and serum levels should be checked 5-7 days after initiating treatment. Trough concentrations (8-12 hrs post-pilling) of 0.8 – 1.2ng/ml (1 – 1.5 nmol/L being optimal. Beta-blockers can also be considered as they may have a positive long term effect by reducing the sympathetic drive and thus will protect the myocardium in the long run. Beta blockers should be used with care and introduced very slowly and never used in a patient with decompensated patient with signs of pulmonary oedema or hypotension.

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References available on www.vet360.vetlink.co.za

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EARLY DETECTION OF HEART DISEASE IN LARGE BREED DOGS.

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Identifying dogs with dilated cardiomyopathy (DCM) that are not yet showing obvious clinical signs can be challenging. A diagnosis can be made with echocardiography, but screening every at-risk dog with an echocardiogram (ECHO) is impractical.

Follow this simple screening process to determine which large-breed dogs have early evidence suggestive of DCM and are suitable for further screening.

DOGS AT RISK

Dog >3 years of age
+
Dog >20 kg
AND/OR
At risk breed*

→ If there is a high index of suspicion, proceed directly with ECHO† if available

PERFORM PHYSICAL EXAMINATION

- Abnormal pulse or pulse deficits
  OR
- Any arrhythmia
  OR
- Gallop sounds
  OR
- Soft systolic murmur

AND/OR

NOTE SUBTLE CLINICAL SIGNS

- History of mild exercise intolerance or syncope
- Increased resting respiratory rate (RRR) or effort (>30 at home or >40 in clinic)
- Inappropriate/ unintended weight loss

YES ← → **NO**

YES → Proceed with ECHO† if available. If not, consider additional tests

NO → HIGH RISK BREEDS*†

CONSIDER ADDITIONAL TESTS

Electrocardiogram	OR	Chest radiograph	OR	NT-proBNP[2] biomarker
(non-sinus arrhythmia or VPCs[1] = abnormal)†		(vertebral heart score >10.7 = abnormal)		(>900 pmol/L = abnormal; >500pmol/L for Dobermans)

1. Ventricular premature contraction 2. N-terminal pro-brain natriuretic peptide

- **NORMAL** (DCM possible but less likely) → RE-EXAMINE IN 1 YEAR (1 YEAR)
- **ABNORMAL** (High probability of DCM) → ECHO†
  - **NORMAL** → RE-EXAMINE IN 1 YEAR (1 YEAR)
  - **ABNORMAL** → DIAGNOSIS: PRECLINICAL DCM

Contact Boehringer Ingelheim at salesAH@boehringer-ingelheim.com to obtain your Screening Algorithm Poster

GIVING DOGS MORE LIFE TO LIVE.

* Doberman Pinscher, Boxer, Great Dane, Irish Wolfhound, Dalmatian, Newfoundland, Afghan Hound, Bullmastiff, Neopolitan Mastiff, German Shepherd Dog, Great Pyrenees, Golden Retriever, Scottish Deerhound, Rottweiler, Saint Bernard, Cocker Spaniel, Leonberger, Flat-coated Retriever, Airedale Terrier, Bernese Mountain Dog, Bouvier des Flandres, Dogue du Bordeaux, Old English Sheepdog, Labrador Retriever, Samoyed, English Springer Spaniel, Standard Schnauzer, or other large breeds.

† A Holter monitor is indicated in many dogs with preclinical DCM and as a screening test in some high risk breeds, such as the Boxer and Doberman, regardless of echocardiographic findings.

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CPD Questions

AC/1447/16

1. Which one of the following is NOT consistent with an increased prevalence of DCM?
   a. Female dogs
   b. Male dogs
   c. Large breed dogs
   d. Great danes
   e. Spaniels

2. Which one of the following mechanisms (a – e) is NOT a compensatory mechanism in canine DCM?
   a. Activation of the RAAS
   b. Decrease in angiotensin secretion
   c. Catecholamines
   d. Increased heart rate
   e. Increased blood pressure

3. Catecholamines are increased in DCM. Which one of the following (a-e) is NOT caused by catecholamine release?
   a. Down regulation of Beta receptors
   b. Impaired systolic function
   c. Impaired diastolic function
   d. Exacerbation of arrhythmias
   e. Decreased heart rate

4. Which one of the following (a – e) is NOT due to the RAAS system?
   a. Vasoconstriction
   b. Supraventricular Arrythmias
   c. Sodium and water retention with increased thirst
   d. Cardiac myocyte hypertrophy
   e. Myocardial necrosis and fibrosis

5. Which one of the following clinical signs is (a-e) NOT typical in a case with DCM?
   a. Dyspnoea
   b. Weight loss
   c. Bradycardia
   d. Exercise intolerance
   e. Ascites

6. Which one of the following clinical signs (a-e) is NOT typical of pericardial effusion?
   a. Arrhythmia
   b. Ascites
   c. Exercise intolerance
   d. Tachycardia
   e. Jugular pulse

7. Which one of the following changes is NOT a typical radiographic finding in canine DCM.
   a. Prominent left atrium
   b. Prominent right atrium
   c. Pulmonary oedema
   d. Cardiomegaly
   e. Pleural effusion

8. Which one of the factors listed (a – e) is NOT a negative prognostic factor in canine DCM Negative variables?
   a. Breed - great dane
   b. Onset of clinical signs at a young age.
   c. Pulmonary oedema at presentation
   d. Presence of VPCs
   e. Higher plasma creatinine levels

9. Which one of the following echocardiographic criteria (a-e) is least reliable to make a diagnosis of canine DCM?
   a. Left ventricular dilation in systole
   b. Breed specific adjusted evaluation of fractional shortening
   c. Fractional shortening of < 20 – 25%
   d. EPSS >6.5 mm
   e. Left or bi atrial enlargement

10. Which one of the mechanisms listed (a – e) is not applicable to pimobendan ?
    a. Is a positive inotrope by increasing myocyte sensitivity to calcium
    b. Inhibits phosphodiesterase III
    c. Reduces pulmonary arterial pressures
    d. Decreases heart rate by slowing impulse conduction
    e. Increases stroke volume

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Shaping the future of animal health

Shampoo Therapy

Veterinary Dermatology

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INTRODUCTION

The use of therapeutic shampoos for the treatment of skin diseases has become an integral part of the treatment regimes of Veterinary Dermatology. Veterinarians should know details about the composition of shampoos, their mechanism of action, and when and how to use them. This will help in instructing owners and obtaining the best treatment results.

A shampoo is an aqueous solution, with added surfactants, cleansing agents and various therapeutic and/or cosmetic agents. In animals, the hair coat penetrating and cleansing effect of a shampoo has a great advantage over the use of other topical formulations such as creams and ointments. The pH of a dog's skin is different from that of a human. The pH of canine skin is 7.5 compared to 5.5 for human skin. Due to the alkalinity of a dog's skin, it provides a favourable environment for bacterial growth. Many skin diseases in small animals have a wide spread to generalised distribution. Shampoos are particularly useful for the treatment or management of such disorders.

GENERAL PRINCIPLES

Medicated shampoos work in more than one way. They allow application of specific therapeutic molecules to skin and hair, restore normal structure and function of the skin and cleanse the skin and hair of microbes and parasites, allergens and other harmful material. In this way they enable many specific and non-specific dermatological disorders to be effectively treated, managed or prevented.

The therapeutic molecules need to be absorbed and to penetrate to the deeper layers of the skin in high enough concentrations in order to achieve a therapeutic effect. The time needed to achieve this, is known as the contact time. Contact time can be defined as the time interval between the end of applying the shampoo and rinsing it off. Contact time is usually between 5 and 15 minutes. In most shampoos the active ingredients are removed when the shampoo is rinsed off. To be fully effective many medicated shampoos have to be repeated very frequently. This may lead to dehydration of the stratum corneum and loss of the protective barrier function and often leads to disappointing results. The correct contact time and correct frequency of the shampoo therapy are therefore crucial for success.

SHAMPOO THERAPY FOR SPECIFIC DERMATOLOGICAL DISORDERS

There are 6 categories of dermatological disorders which will be discussed with special focus on shampoo treatment of these disorders:

1. Keratoseborrhoeic disorders (KSD)

Keratoseborrheic disorders (KSDs) are those that alter the surface appearance of the skin. The epidermis of animals is constantly being replaced by new cells. The normal epidermal cell renewal time is approximately 22 days. Despite this high turnover rate, the epidermis maintains its normal thickness, has a barely perceptible surface keratin layer, and loses its dead cells invisibly to the environment. If the delicate balance between cell death and renewal is altered, the epidermal thickness changes, the stratum corneum becomes noticeable, and the normally invisible sloughed cells of the stratum corneum become obvious.

Keratoseborrheic disorders (KSD) are characterized by scaling, crusting, greasy or dry skin and alopecia and can be primary or secondary. Generally the primary KSDs are hereditary and are associated with a primary keratinisation defect. They usually manifest clinically by excess scale formation in which the primary pathophysiology involves a defect in the keratinising

epithelium or cutaneous glandular function. Clinical signs will usually appear during the first 2 years of life. Primary KSDs include primary idiopathic seborrhoea, epidermal dysplasia, Schnauzer comedo syndrome, canine ichthyosis and canine acne. These cases usually cannot be cured and very often need life long or long term treatment. Breeding with affected individuals should be discouraged.

At least 80% of KSD cases seen in general practice are secondary. The most common causes include allergies (flea allergic dermatitis, atopic dermatitis and cutaneous adverse food reactions), bacterial infections, immune-mediated disorders, endocrine disorders, parasitic disorders (scabies, demodicosis, and cheyletiellosis), fungal infections and nutritional disorders.

Shampoos containing keratomodulating and/or antiseborrheic agents, as well as essential fatty acids, are indicated for these disorders.

Keratomodulating agents

Keratomodulating agents have two mechanisms of action.

- Firstly they restore normal keratinocyte multiplication and keratinisation by exerting a cytostatic effect on the basal cells, thereby reducing their rate of division. Agents working in this way are called keratoplastic (keratoregulating).
- The second mechanism of action is the elimination of excess keratin of the stratum corneum, either by increasing desquamation, by reducing intercellular cohesion or by breaking up keratin. Agents working in this way are called keratolytic. Keratolytic agents therefore break down the keratin layer.

There are several types of keratomodulating agents, e.g. salicylic acid and sulphur.

- Salicylic acid (0.5 – 2%) is keratolytic. It causes a reduction in skin pH, which leads to increased hydration of keratin. These actions help to soften the corneal layer. It has direct effects on the intercellular junction system (desmosomes) with solubilisation of intercellular cement and facilitation of keratinocyte detachment (desquamation). It acts synergistically with sulphur and is often present in small quantities in shampoos, its efficacy varying with concentration.

- Sulphur (0.5 - 2 %) is mildly keratolytic (forms hydrogen sulphide in the stratum corneum), keratoplastic (has a direct cytostatic effect, interacts with epidermal cysteine to form cystine which is an important component of the stratum corneum), and has numerous antiseborrheic effects. It is gradually being replaced in topical products by more effective keratomodulating agents with fewer side effects (e.g. a rebound increase in scaling). It is not a good degreasing agent. Sulphur may be used in cats.

Antiseborrheic agents

Antiseborrheic agents inhibit or reduce sebum production by the sebaceous glands, and help clear the ducts. These agents act at the level of the sebaceous gland and its duct. Examples are sulphur and benzoyl peroxide.

- Sulphur is a classic antiseborrheic agent, but is drying and may trigger a rebound effect. It exerts a synergistic activity with salicylic acid. This synergism appears optimal when both substances are incorporated into the shampoo in equal concentrations.

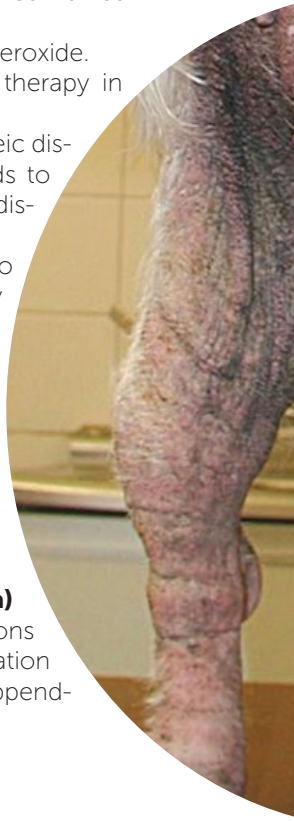
- Benzoyl peroxide is antiseborrheic by causing sebum hydrolysis and reduced sebaceous gland activity. It exerts a follicular flushing action which is useful when treating disorders associated with come domes and/or follicular hyperkeratosis. It is bactericidal and keratolytic. It is particularly useful in severe cases of greasy seborrhoea with or without pyoderma. Side effects such as irritations and erythematous rashes have been reported especially in concentrations higher than 5%. Long term use may lead to a dry skin. Emollients are always indicated after treatment with this agent.

Essential fatty acids

Essential fatty acids have been incorporated in to various veterinary shampoos for their softening and moisturising properties. These include linoleic acid, glycerine, lactic acid, ceramides and fatty acid polyesters.

Principles of shampoo therapy for KSDs

- Mild, dry scaling often responds to a moisturising, hypoallergenic shampoo.
- More severe dry scaling responds to sulphur, salicylic acid combinations, preferably combined with moisturisers.
- Greasy, oily scaling requires benzoyl peroxide.
- Guidelines for the use of shampoo therapy in KSDs
- Long haired dogs with severe seborrheic disorders should be clipped as this leads to more effective application and better distribution of the active ingredient;
- Shampoos should initially be used 2 to 3 times weekly. With time, frequency of application can gradually be reduced;
- Cases should be monitored frequently. The therapeutic agent often needs to be changed following the development of side effects or change in clinical presentation (e.g. transition from greasy to dry seborrhoea).



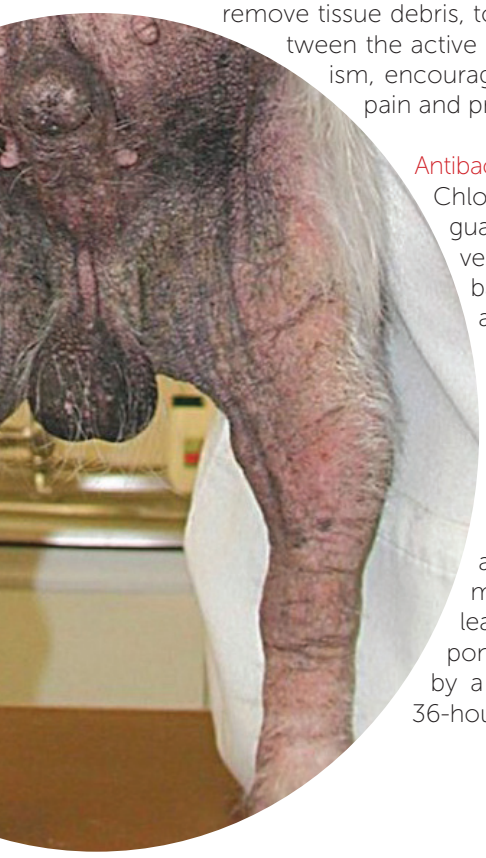
2. Bacterial skin infections (Pyoderma)

Bacterial pyodermas are pyogenic infections of the skin and occur due to the multiplication of bacteria on or in the epidermis and its append-

ages and with invasion of the dermis. Pyodermas are almost always secondary. Classification of pyoderma based on depth of bacterial involvement is clinically the most useful, because it provides information on diagnosis, differential diagnosis, underlying disease, prognosis and response to treatment. Pyoderma may be on the skin surface, affecting the stratum corneum and outer epidermis (e.g. intertrigo and pyotraumatic dermatitis); superficial, involving only the epidermis (e.g. impetigo) and the epithelial appendages in the dermis (e.g. folliculitis) or deeper, compromising structures in the dermis and deep, subjacent fatty tissue.

The development of pyoderma occurs in 2 stages. Firstly the microbes colonise areas of the body surface. This occurs commonly as many skin diseases are aggravated by microbial colonisation or invasion. The second phase occurs when the stratum corneum is invaded to cause impetigo and/or invasion of the hair follicles causing folliculitis. The latter two are classified as superficial pyoderma. When the infected hair follicle ruptures, the infection spreads into the dermis (furunculosis) or spreads more deeply along tissue planes (cellulitis). The latter 2 are termed deep pyodermas. Resolution depends on the non-specific defenses (neutrophils and macrophages) and on the specific defenses (cell mediated immunity and antibodies). Resolution is accompanied by an inflammatory response, which is responsible for the clinical signs of pyoderma.

Shampoos are used to decrease the number of microbes on the skin surface and rinse them off. This interrupts the microbial colonisation, thus sustaining the restoration of normal skin structure and function. Shampoos are also used in pyoderma cases to remove tissue debris, to allow direct contact between the active ingredient and the organism, encourage drainage and decrease pain and pruritus.



Antibacterial agents

Chlorhexidine is a synthetic biguanide, an antiseptic that is very effective against most bacteria (gram positive and negative), fungi and Malassezia pachydermatis. This molecule exists as various forms of salts. Chlorhexidine digluconate is the most used form in topical dermatology. It is bactericidal by action on the cytoplasmic membrane, which causes leaking of intracellular components. It is characterised by a rapid "kill" action, has a 36-hour residual activity and is non-toxic and non-irritant.

Povidone-iodine is an iodophore which slowly releases iodine to tissue. The titre able iodine is usually of the order of 0.2 to 0, and 4 %. It is an effective broad-spectrum antimicrobial and is useful for local lesions. It has a prophylactic effect because of its persistence on the skin. It is not advisable to be used repeatedly for generalised skin problems due to its irritant and staining properties.

Benzoyl peroxide is metabolised in the skin to benzoic acid and much of its microbicidal activity probably derives from the lowered skin pH. This disrupts microbial cell membranes. It is also an oxidizing agent, which releases nascent oxygen into the skin and produces a series of chemical reactions resulting in permeability changes and rupture of bacterial membranes. It has an excellent prophylactic effect. It is generally used in concentrations of 2 to 3%, which are well tolerated, but irritation can occur at higher concentrations (erythema, pruritus and pain). Benzoyl peroxide is toxic to cats. It may, however, be used sparingly/diluted on local lesions such as chin acne or stud tail.

Piroctone olamine is an antiseptic agents that has broad in vitro activity against major dermal veterinary pathogens, including dermatophytes and yeasts as well as gram positive (Staphylococcus) and gram negative (Pseudomonas) bacteria. Piroctone olamine is unrelated to other antiseptics used in Veterinary Medicine. Members of the "pirox" family are used in the human field to cure onychomycosis and Malassezia related skin disorders. No resistance has been documented to date. In addition, this antiseptic acts at low concentrations, has high affinity for keratin and is completely safe.

Quaternary ammonium compounds are surface acting agents. They have less effect and are only useful for limited bacterial involvement.

Guidelines for the use of shampoo therapy in bacterial pyodermas

- In general, most patients with bacterial pyoderma will be treated with systemic antibiotics, and topical therapy is used as an adjunct treatment. The exceptions would be those dogs that have surface or superficial pyoderma.
- It is important to pre-bath with a mild cleansing shampoo to remove grease, debris and dirt prior to using the medicated shampoo.
- Cases of deep pyoderma need to be clipped before using shampoos. This prevents the formation of sealing crusts and allows the active ingredients to come in contact with the lesions.
- The frequency of bathing will depend on the individual patient, but in general for active infections medicated baths should be administered 2-3 times weekly.
- A common indication for the long term use of

shampoos is in the dog suffering from recurrent folliculitis (idiopathic of secondary to endocrine or allergic skin disease). Here antibacterial shampoos may have a prophylactic effect if used regularly e.g. weekly or every two weeks.

3. Fungal skin infections

Malassezia dermatitis is a fungal skin infection. The pathogenic agent, Malassezia pachydermatis, is a lipophilic yeast belonging to the cutaneous microflora of the normal dog, along with *Staphylococcus pseudintermedius*. In certain conditions, related to cutaneous and/or immune-mediated factors, Malassezia may proliferate within the stratum corneum. The subsequent yeast overgrowth may then acquire a real pathogenic capacity and initiate a true dermatitis. It is recognised as a secondary pathogen in a large number of skin conditions, e.g. in dermatoses associated with dry or greasy scales, inflammatory and pruritic skin conditions and in various pyodermas. It may also be a primary pathogen.

Topical therapy is often used for the treatment of Malassezia infections and may also be beneficial when used routinely to help prevent recurrence of infection. Shampoo therapy is most useful for generalised infections, while localised infections may benefit from creams, lotions and sprays. Dogs with severe generalised Malassezia infections show the most rapid response where shampoo therapy is used in conjunction with systemic therapy (as compared to topical therapy or systemic therapy alone). Active ingredients in topical preparations that may be useful in managing Malassezia infections include: imidazoles (miconazole, ketoconazole, enilconazole), chlorhexidine, and piroctone olamine.

Imidazoles act by interfering with cell wall formation in fungal and yeast organisms, which increases cellular permeability, thus suppressing metabolic function and inhibiting growth. There has also been evidence that ketoconazole exerts an inhibitory effect on keratinocytes in culture. In a study by Jasmin and coworkers a 3% chlorhexidine shampoo was highly effective in the treatment of Malassezia dermatitis and also aided in the treatment of the concurrent bacterial pyoderma when present. The product had similar efficacy whether dogs were affected with concurrent pyoderma (treated with systemic antibiotic) or not, and whether an underlying dermatosis was present or not.

The use of a keratoseborrheic shampoo prior to applying the antifungal shampoo enhances the effectiveness of the antifungal agent.

Dermatophytosis is a fungal infection of the keratinised tissues, i.e. the hair, stratum corneum, nails and claws. It is caused by Microsporum spp. (70% of cases) and Trichophyton spp. (30% of cases) in dogs. Microsporum canis causes 98% of all cases in cats.

The best treatment protocol is a combination of topical treatment (to kill infective material and prevent its dissemination into the environment and to accelerate recovery), systemic treatment (to shorten the time of infection in the infected animal) and environmental treatment (to help prevent recurrence of infection or spread to other animals or people in the household).

Topical treatment may include the following: imidazoles (miconazole, ketoconazole, enilconazole), lime sulphur, chlorhexidine and piroctone olamine.

In a study by White-Weithers and Medlieu, seven commonly used, topical antifungal products (i.e., lime sulfur, chlorhexidine, captan, povidone-iodine, sodium hypochlorite, and enilconazole solutions, and ketoconazole shampoo) were evaluated for their antifungal activity on Microsporum canis-infected hairs from dogs and cats in an in vitro study. Hairs were soaked or shampooed in each product for five minutes twice a week for four weeks.

Of the seven products used in this study, lime sulfur and enilconazole solutions were superior in inhibiting fungal growth; no growth occurred on fungal cultures after two treatments with either product. Chlorhexidine and povidone iodine solutions were effective after four treatments, and sodium hypochlorite solution and ketoconazole shampoo inhibited fungal growth after eight treatments. Captan did not inhibit fungal growth during the test period.

Guidelines for the use of topical therapy in dermatophytosis

- Clipping the hair coat is recommended in generalised infections to decrease shedding of contaminated hairs into the environment.
- Owners should be warned that animals may have more lesions during the first week after clipping.
- Keratomodulating shampoos can be used before antifungal therapy when indicated as they are beneficial in removing infected scales and crusts.

4. Allergic skin diseases

Shampoo therapy often contributes a large part in the management of allergic skin diseases. Shampoos rehydrate the skin and result in the patient looking, smelling and feeling better. Shampoo therapy in cases with allergic skin disease helps to eliminate allergens from the skin surface, helps to restore the epidermal

barrier and helps to control the inflammatory process and secondary skin infections.
There are a variety of shampoos available that may be used in the management of an allergic skin disease: Shampoos with an antipruritic effect can improve the condition of allergic dogs, especially when they are used frequently (e.g. twice a week, at least initially). The most widely recommended topical antipruritic agent is colloidal oatmeal. It has both emollient (softens, lubricates, soothes) and hydroscopic (incorporated into the stratum corneum and attracts water) activities and has a direct anti-inflammatory and antipruritic action.

The exact mechanism is not clear.

- Antibacterial and antifungal shampoos e.g. chlorhexidine may be used to control secondary bacterial and /or yeast infections.
- Keratomodulating shampoos are indicated in cases with allergy induced keratoseborrhoea.
- Cleansing, non-irritating shampoos, fatty acid shampoos and ceramide containing shampoos may be used to restore the epidermal barrier.
- A shampoo specifically designed for canine atopic dermatitis has been recently developed (Allermyl®). It was designed to restore cutaneous integrity, maintain epidermal barrier function, control aggravating microbial proliferation, and limit immune and inflammatory reactions. It contains linoleic acid, gamma-linolenic acid, mono and oligosaccharides, vitamin E, and piroctone olamine.

5. Parasitic skin disorders

Antiparasitic shampoos, e.g. containing organochlorins, natural pyrethrins or synthetic pyrethrins, are not considered to be as efficacious as antiparasitic rinses and dips and other formulations (sprays, spot-ons), mainly because they are rinsed and cannot act during a sufficient time. Their main use is for quick removal of fleas in puppies, kittens and debilitated animals.

Colloidal oatmeal can be used to decrease inflammation and pruritus due to parasitic infestation. Benzoyl peroxide shampoos are recommended in the treatment of demodicosis because of their degreasing and follicular flushing effect. Many parasitic diseases such as scabies, cheyletiellosis and flea allergic dermatitis can cause a keratoseborrheic disorder and affected animals will benefit from application of keratomodulating shampoos.

6. Dry sensitive skin

Dry skin results from increased water loss through the skin. It occurs in many skin conditions, such as allergies, with excessive use of corticosteroids or due to treatment with inappropriate shampoos.

Water by itself has a strong hydrating effect if used properly. A contact time of 10 – 15 minutes should be allowed to properly hydrate the stratum corneum. If the contact time is too short or baths are given too frequently, the skin will dry further by evaporation of water.

Emollients are used to cover the surface of the skin, thus decreasing transepidermal water loss and keeping the skin soft and pliable. They are usually based on plant or mineral oils. Today emollients are rarely used in veterinary medicine, except for topically applied essential fatty acids. They reduce transepidermal water loss by being incorporated into the stratum corneum ceramides.

Humectants are oil free agents that work by being incorporated in the stratum corneum and attracting humidity from the lower layers of the epidermis. They are colloidal oatmeal, sodium lactate, urea, lactic acid, propylene glycol, glycerine or carboxylic acid. Humectants are used to rehydrate dry skin and may be applied in between baths.

This category includes products said to be "hypoallergenic" and "All-natural." Some of these shampoos, however, contain colorants, whiteners, deodorants, added colours or fragrances that can be potent irritants and sensitisers for allergies. True hypoallergenic shampoos should contain few substances that could cause an allergic reaction. They should be the least irritating shampoos on the market. "All-natural," which means that none of its components are manmade or synthesized, should not be confused with "hypoallergenic." Some natural ingredients, including oatmeal, aloe vera, melaleuca oil, tea tree oil, citrus extracts and eucalyptus, may be primary irritants or allergens. Others, such as eucalyptus, used for their moisturising and/or anti-inflammatory properties, can be potent allergens.

Conclusion

Therapeutic shampoos are often used as an adjunct to systemic, usually oral therapy, e.g. antibiotics and antifungals. They help to achieve better and faster results and prevent early recurrence by restoring normal structures and functions of the skin. Shampoos cause the patient to feel better and smell better and that is already very encouraging for the owners.

Advantages include direct therapeutic access to target organ, reduction in systemic absorption and adverse effects and better delivery systems of the new generation shampoos. Shampoo therapy must be carefully adjusted to the needs of the individual. Good communication with the client and evaluation of their compliance is crucial for successful treatment. The contact time and the correct frequency of the shampooing are crucial. Shampoo therapy increases the cost of therapeutic plan, but appropriate topical therapy may greatly reduce the need for systemic treatment.

References available on www.vet360.vetlink.co.za

Bad Hips and Knees: Hip Dysplasia or a Torn Cruciate Ligament?

By Jennifer L. Wardlaw, DVM, MS, DACVS
VETERINARY MEDICINE

It can be difficult to differentiate between these two orthopedic problems. Luckily, the sit test, among other diagnostic evaluations, can help.

Differentiating between a torn cruciate ligament and hip dysplasia can be tricky, if not frustrating. In one study, 32% of dogs referred to a surgeon for hip dysplasia treatment actually had a torn cranial cruciate ligament.[1] Let's review the differences between the two conditions and how simply asking a dog to "sit" offers great clues.

CRANIAL CRUCIATE LIGAMENT TEAR

The severity of lameness depends on the severity of ligament disruption. For dogs with stable partial tears, lameness can be subtle and noted only after periods of strenuous activity. For dogs with complete tears, lameness will initially be severe and non-weight-bearing. Then, moderate to severe weight-bearing lameness will occur.

Rupture of the contralateral cruciate ligament occurs in 37% to 48% of dogs within six to 17 months of the initial diagnosis.[1] However, ruptures can be bilateral on presentation, giving affected dogs what appears to be a neurologic, crouched walk.

In obvious cases, the keys to diagnosing cranial cruciate rupture are a positive cranial drawer sign and tibial thrust. But what about less obvious cases?

Physical examination

Orthopedic examination reveals various degrees of stifle pain with flexion and extension, variable crepitus, and possibly clicking associated with a meniscal tear.

In patients with partial tears, a pain response is elicited when the joint is in full extension. In patients with chronic cases, muscle atrophy is notable, and periarticular fibro-

Figure 1: Sit Test in a Labrador.

sis (medial buttress) is evident on the medial side of the stifle. Medial buttress is almost pathognomonic for a cranial cruciate rupture. The only other condition that may present with a medial buttress is a medial collateral ligament tear, which is usually seen with a deranged stifle, not with simple lameness.

Joint effusion is also a key finding. It can be palpated on the medial and lateral aspects of the patellar tendon.
In patients with a partial tear, the cranial drawer sign may or may not be present. An examination performed while the patient is sedated is needed to confirm the findings. Many patients that do not seem to have a cranial drawer sign while awake have one once they are sedated and relaxed.

Sit test

Dogs with a torn cruciate ligament sit abnormally. For example, notice how the patient in Figure 1 does not want to flex its right knee. Affected dogs often sit with the affected leg extending out to the side rather than sitting squarely, which they will do even with hip dysplasia. So noting how the dog sits is a critical part of an evaluation.

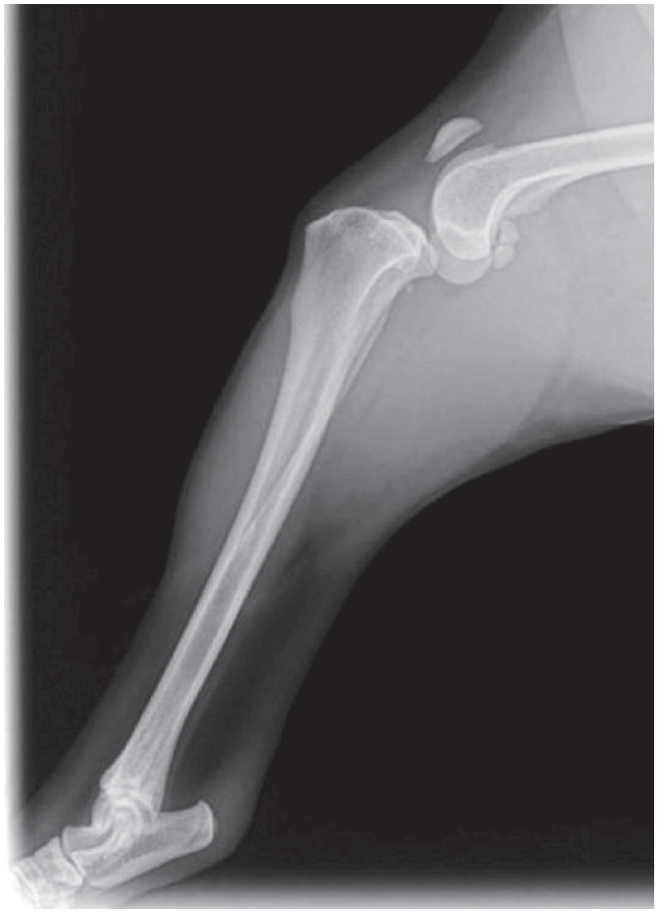


Figure 2a: Cranial tibial thrust is evident on this radiograph. The knee is subluxated.

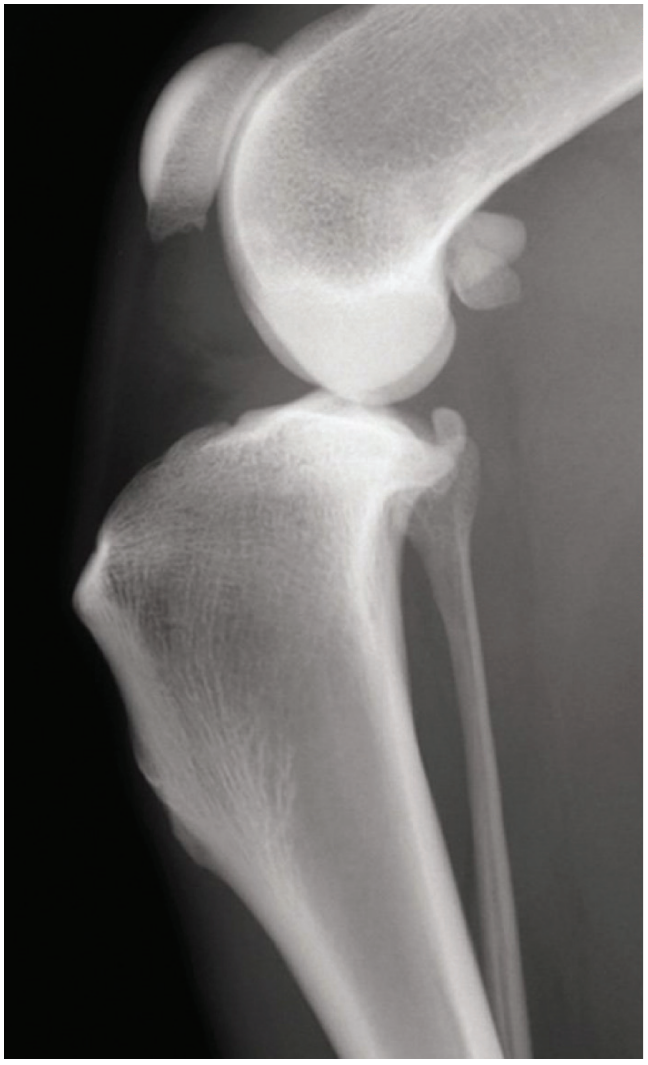


Figure 2b: Joint effusion is appreciated on this radiograph (loss of visualization of the infrapatellar fat pad shadow and caudal displacement of the gastrocnemius facial plane), but this tibia is positioned normally without subluxation.

This dog's abnormal sitting position--extending its leg rather than flexing its knee--indicates that it has a cruciate ligament tear, not hip dysplasia.

Imaging

Radiography is warranted in all suspected cases to document stifle arthritis, confirm pathology in challenging cases of partial tears and rule out other disorders (e.g. tumors). The earliest, most consistent finding is the loss of an infrapatellar fat pad shadow by a soft tissue opacity in the lateral view, which is consistent with effusion. Caudal displacement of the gastrocnemius fascial plane, located caudal to the joint capsule by a soft tissue opacity, is also consistent with synovial distention.

In many cases, you can see the cranial tibial thrust on a

radiograph (Figure 2a). Compare this with the position of the second radiograph, which also has effusion but the tibia is not displaced into a cranial position (Figure 2b). Another consistent finding is osteophyte or enthesiophyte formation in the region of femoral trochlear ridges and tibial plateau and at the base and apex of the patella.

HIP DYSPLASIA

Hip dysplasia causes joint inflammation and secondary osteoarthritis, which lead to variable degrees of pain. Clinical signs can vary from slight discomfort to severe acute or chronic pain. Although the disease onset has a linear progression over time, it can be divided into two forms.

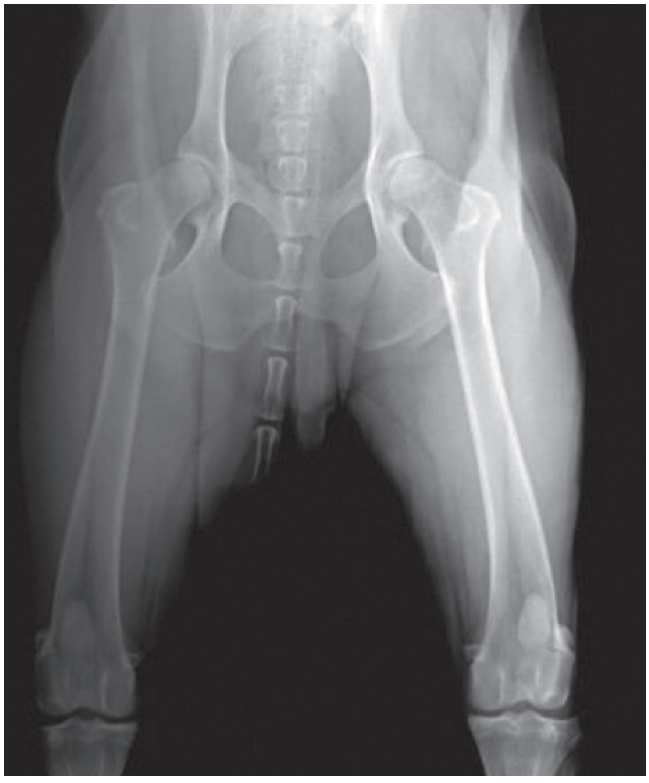


Figure 3a: Mild hip dysplasia often has incongruence, acetabular sclerosis and a thickened Morgan's line where the joint capsule inserts along the femoral neck.

The juvenile form typically affects dogs between 5 and 12 months of age. Affected dogs may present with unilateral or bilateral hindlimb lameness or pain on hip extension. Affected dogs may be bunny hopping at presentation, have difficulty rising after rest, exhibit exercise intolerance, or seem reluctant to walk, run, jump or climb stairs. These clinical signs are the result of joint laxity and resultant instability and inflammation.

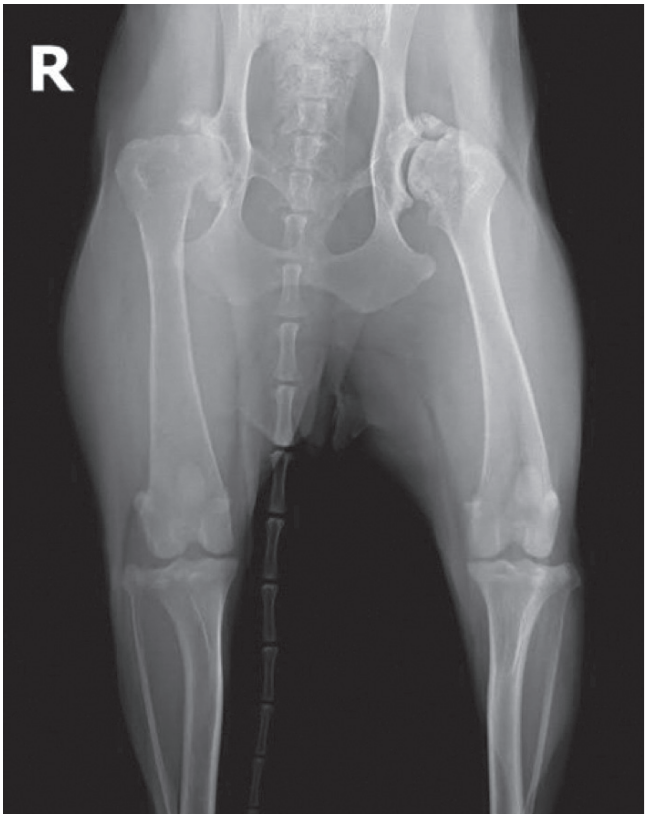


Figure 3b: Severe hip dysplasia is more easily seen and felt on physical examination. Radiographs confirm arthritic changes and help planning for surgery.

The chronic form of hip dysplasia has a highly variable onset of clinical signs in middle-aged to senior dogs.

Pain is most often related to degenerative joint disease and has a more severe presentation. Clinical signs are similar to the juvenile form. Pain is elicited most notably during hip extension. Patients tend to off load their hip joints by shifting weight forward onto their thoracic limbs. Because of weight shifting over a long period of time, muscle atrophy of the hindlimbs is common. In large dogs, you can often see muscular hypertrophy of the forelimbs as a result of the dogs' chronically hauling themselves up by their forelimbs.

Imaging

As the disease progresses, crepitus can be palpated with range of motion manipulation. An examination while the patient is sedated, followed by orthogonal radiography will further support the diagnosis of hip dysplasia. While the more chronic cases are much easier to diagnose on radiographs and physical examination (Figures 3a & 3b),

If you examine the patient and still have doubts about the diagnosis, sedate the dog and repeat your entire examination.

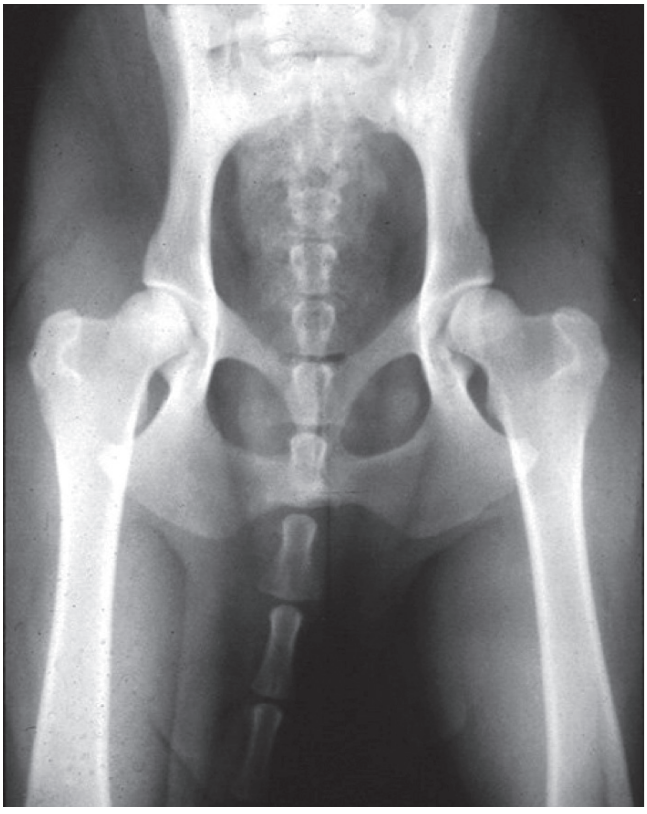


Figure 4a: This radiograph was scored as OFA Good with mild incongruence and slight acetabular sclerosis in a young dog.

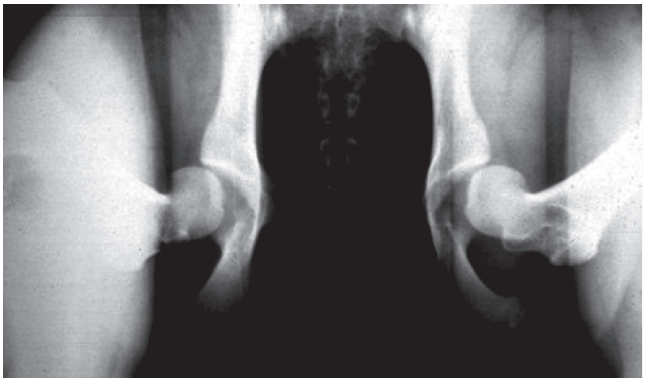


Figure 4b: This distracted frog-leg view is of the same dog in Figure 4A but more clearly illustrates the laxity that is present in this patient that was missed with the standard OFA-style radiograph.

younger dogs can often be challenging on radiographs. Since you may not have bony arthritic changes on these films, assessing for laxity in your physical examination and radiographs is vital for young dog diagnosis (Figures 4a & 4b).

Sit test

Dogs with only hip dysplasia (no concurrent cruciate ligament injury) sit normally, with both legs flexed symmetrically.

DOUBLE TROUBLE

Of course, both conditions can be present in a dog at the same time. In the study mentioned above, 32% of dogs referred to a surgeon for hip dysplasia treatment had a torn cranial cruciate ligament.[1] Interestingly, 94% of the dogs with a cruciate tear had concurrent radiographic signs of hip dysplasia.[1]

Thus, I think it is a great practice to radiograph the hips in patients with torn cruciate ligaments as well since it affects the pain and rehabilitation protocols. In my experience, 80% never need hip surgery, but your knee patients won't do as well if you aren't aware you are fighting two battles.

CONCLUSION

When evaluating an affected dog, it is imperative to do a thorough orthopedic and neurologic examination to accurately localize the clinical signs and provide an appropriate diagnosis and treatment.

Acknowledgment

The author would like to thank Dr. Phil Zeltzman for his input in this article.

Reference

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Anaesthesia

in Patients Suffering a Heart Murmur

Article review by Dr Lynette Bester

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Heart murmurs diagnosed on precordial auscultation are a significant finding that warrant further clinical investigation.

The clinical implications of the murmur requires consideration to determine the anaesthetic risk.[2] Patient signalment, presenting complaint, presence of concurrent diseases, exercise tolerance and current medication are important considerations to address during a pre-anaesthetic evaluation. A heart murmur per se is not a contraindication, but rather a caution for general anaesthesia in a patient not showing clinical signs. The drug selection to achieve general anaesthesia and analgesia may require a patient tailored approach, especially if the murmur is due to cardiac pathology.

- Murmurs are audible evidence that there is turbulent blood flow through the heart. The murmur is graded according to how loud the murmur is compared to the lub-dub sounds heard during the cardiac cycle.[4] The lower the grade of the murmur (III and less) the less reliable it is to indicate the severity of the pathology.[4, 9] Therefore, on detecting a heart murmur, further investigation is required to determine the severity of the pathology and if the murmur is cardiac in origin or not.

Post-murmur-detection clinical investigation

Two critical clinical investigation aims need to be achieved. First, is to determine if the pathology is due to cardiac or non-cardiac processes. Second, is to determine if the patient is otherwise clinically affected by the presence of the murmur.[1, 2]

- Cardiac related pathologies include valve stenosis or insufficiency (disease or traumatic), atrial or ventricular septal defects, or complex cardiac malformations (e.g. tetralogy of Fallot). Non-cardiac pathologies include vascular anomalies (e.g. patent ductus arteriosus), altered viscosity (e.g. anaemia), altered flow velocity (e.g. hyperdynamic flow), or obstruction to flow through the heart (e.g. thrombus, air emboli).[4]
- Exercise tolerance is considered an excellent marker to gauge cardiovascular fitness and risk for general anaesthesia, whereby, the more tolerant the patient is to exercise the lower the risk of general anaesthesia. Perceptive questioning could clarify the patient's exercise tolerance.
- The clinician should be suspicious if the patient presents with a murmur and tachycardia, delayed capillary refill time, pale mucous membranes and a history of being "sleepy" or mostly inactive during the day. These findings could suggest that the patient is not cardiovascular stable and thus of a higher anaesthetic risk. The resultant pathophysiology is invariably related to a decrease in cardiac output which translates into a decrease in perfusion of oxygen to metabolically active tissues.

Figure 1: Evaluation of blood pressure by doppler

Oxygen is chiefly transported to the tissue via haemoglobin and delivery is dependent on the cardiac output. Cardiac output is the product of the stroke volume per beat multiplied by the heart rate. The stroke volume is generally decreased in patients suffering cardiac murmurs. The heart rate is increased to preserve the cardiac output. Thus heart rate can be an indicator of clinical stability.

Arterial blood pressure (figure 1) is the product of cardiac output multiplied by the systemic vascular resistance. During low cardiac output states, systemic vasoconstriction is often present in order to preserve an adequate arterial blood pressure (activation of the sympathetic nervous system and the renin-angiotensin-aldosterone axis). Therefore, patients presenting with pallor and tachycardia with an audible murmur require pre-anaesthetic stabilisation.

Pre-anaesthetic stabilisation

A functional or innocent heart murmur in an otherwise healthy patient may not require any special pre-anaesthetic stabilisation.[4] Patients which are already on cardiovascular supporting drugs (pimobendin, digoxin, angiotensin converting enzyme inhibitors, diuretics etc.) but are otherwise stable and compensating may also not require stabilisation. Serum electrolytes (potassium, sodium and calcium) should be measured in patients undergoing long term diuretic or digoxin therapy, as they may present with low potassium or calcium concentration which would prolong the recovery. Sodium is important to maintain the intravascular fluid volume (water shifts according to the sodium gradient between the blood and tissue) of circulating blood. If the sodium level is high it could indicate that there is a decrease in the circulating volume and that fluid bolusing may be required. While a low sodium level may indicate caution with fluid management as it would be easier to inadvertently fluid overload these patients. Furthermore, routine haematology and serum creatinine and total serum proteins (albumin of most interest) should be measured for baseline readings in all patients. Collecting this data

Figure 2: Cat under general anaesthesia being monitored by pulseoximeter

prior to general anaesthesia may assist in managing patients suffering unexpected post-recovery complications.[6, 11]

- Patients presenting with cardiovascular instability should be managed medically prior to general anaesthesia (Table 1). Patients that are cardiovascularly unstable and requiring emergency intervention carry a high risk for general anaesthesia. The presenting pathology will indicate the appropriate emergency medical intervention to improve the cardiac output, blood pressure and tissue oxygenation prior to general anaesthesia.
- Murmurs due to anaemia have serious implications and will require pre-anaesthetic stabilisation. It is imperative to understand that oxygen transport is heavily dependent (97% of all transport) on the amount of functioning haemoglobin available. Pre-oxygenation with 100% oxygen prior to general anaesthesia in an anaemic patient is inadequate to compensate for a low haemoglobin

Table 1: Clinical signs associated with cardiovascular instability requiring stabilisation prior to general anaesthesia in patients presenting with a murmur

Clinical parameter	Parameter range	Cause; possible stabilisation treatments
Tachycardia	Heart rate > 160 beats/minute	Increased sympathetic tone, activation of the RAAS; isotonic fluids, inotropes, vasodilators, ACE-inhibitors
Mucous membrane colour	Pale pink to white (with or without a blue tinge)	Vasoconstriction due to increased sympathetic tone, RAAS or anaemia; isotonic fluids, inotropes, vasodilators, ACE-inhibitors, fresh whole blood (anaemia)
Capillary refill time	<1 second >2 seconds	Decreased cardiac output, hyperdynamic or hypodynamic cardiovascular states; ; isotonic fluids, inotropes
Mentation	Depressed, inactive	Decreased cardiac output; isotonic fluids, inotropes, ACE-inhibitors
Muscle tone	Weak, poor exercise tolerance	Decreased cardiac output or hypokalemia; isotonic fluids, inotropes, start potassium sparing diuretics

RAAS: Renin-Angiotensin-Aldosterone-System; ACE: Angiotensin-Converting-Enzyme

concentration, especially during low cardiac output states. The generally accepted guideline is to ensure a haematocrit level of greater than 0.2 to 0.25 (20 to 25%) in dogs and cats, even in patients suffering chronic anaemia (e.g. chronic renal failure in cats) prior to general anaesthesia.[6, 11]

Anaesthetic management

General anaesthesia should be kept to the shortest time period possible. Therefore, a decisive plan on all procedures that need to be completed must be decided on and prepared for prior to induction.

- A general principle for a cardiac safe general anaesthesia is to not change the heart rate or vascular tone from that in the pre-anaesthetic clinical exam in a stable patient. Therefore monitoring of the heart rate and blood pressure during general anaesthesia is paramount. Other indicators of peripheral perfusion such as capillary refill time and mucous membrane colour may be helpful in monitoring cardiac output. A pulseoximeter (figure 2) is useful to monitor peripheral perfusion (machine must detect a pulse) and oxygen haemoglobin saturation.[10] Capnography (measuring the end tidal carbon dioxide) should also be used to monitor these patients. The capnograph is a useful tool to detect respiratory depression and poor cardiovascular performance.[6, 11] Other indirect markers of perfusion are heart rate, mucous membrane colour and CRT.
- Patient anxiety and stress should be managed in patients with cardiac disease. Management interventions such as: 1) allowing the patient to be dropped off on the day of the procedure prior to the general anaesthesia, 2) having the owner present during the premedication, 3) keeping the patient in a quiet, warm cage where the patient can be regularly observed, 4) treating for existing pain (injured animals or those suffering osteoarthritis) will all decrease the level of anxiety and stress. Patients suffering from extreme anxiety during visits to the veterinarian may benefit from an oral dose of a benzodiazepine 15 minutes prior to leaving home. Oral alprazolam (0.01 to 0.1 mg/kg) or diazepam (0.2 to 0.5 mg/kg) usually suffices.
- Acepromazine, a phenothiazine derivative psychotropic drug, is a frequently used sedative drug. It causes a dose dependent reduction of the stroke volume and the cardiac output. Due to alpha1-adrenergic blocking effects on the vascular walls, vasodilation occurs and arterial blood pressure decreases.[12] Acepromazine desensitizes the myocardium to the potentially arrhythmogenic effect of catecholamines. Due to its effect on the myocardial alpha1-receptors, it prevents the development of ventricular arrhythmias which may be advantageous in certain cases.[13]
- Patients already on cardiovascular supportive medication should continue their medication as prescribed. Pre-anaesthetic fasting should be 6 to 8 hours. However, water should not be withheld until the administration of the premedication.
- A conservative cardiovascular sparing drug combination should always be used in a patient presenting with a murmur (Table 2). The most common cardiovascular sparing premedication drugs are the pure mu-agonist opioids and benzodiazepines (midazolam and diazepam). Histamine release that causes vasodilation is a common side-

Table 2: Cardiovascular sparing general anaesthetic protocols for diagnostic and surgical patients with precordial auscultated murmurs

Drug	Drug	Species	Notes
Premedication			
Opioids	Morphine 0.2 to 0.4	dog, cat	Histamine release and emesis are concerns
	Fentanyl 0.005 to 0.015	dog, cat	Bolus upwards in increments of 0.003 to 0.005 mg/kg every 5 to 10 minutes. Panting common in dogs
	Sufentanil 0.002 to 0.005	dog, cat	As for fentanyl
	Buprenorphine 0.02 to 0.03	dog, cat	Only use pre-operatively in mild to moderate painful procedures (i.e. not more painful than a gonadectomy in a healthy patient). Very useful post-operative analgesic once pain level is under control.
	Butorphanol 0.2 to 0.4	dog, cat	Useful to antagonise undesirable pure mu-opioid effects or in short non to minimally-invasive diagnostic procedures such as radiographs.
Benzodiazepines	Midazolam 0.2 to 0.5	dog, cat	Intramuscular or intravenous route, use lower dose range for cats
	Diazepam 0.2 to 0.5	dog, cat	Only intravenous route recommended, use lower dose range for cats
Phenothiazine derivatives	ACP 0.01 to 0.03	dog, cat	Excellent vasodilator that could be used in patients suffering valvular insufficiency. Decreases the regurgitation fraction and improves cardiac output. Administer 1 hour before induction of general anaesthesia to allow for clinical effect. Caution if the patient is already on vasodilators, Angiotensin-Converting-Enzyme inhibitors or hypovolemic (contracted intravascular volume)

effect of pethidine administration, and to lesser extent with morphine. Morphine induces vomiting (increases patient anxiety and stress) more commonly compared to fentanyl and methadone. Therefore, pre-emptive treatment with an antiemetic (e.g. maropitant) prior to morphine administration is advised. Buprenorphine, a partial mu-agonist, should only be reserved for minor painful procedures or as a post-surgical analgesic when pain levels have subsided to a mild to moderate pain level (e.g. gonadectomy procedures). Butorphanol, a mixed mu-antagonist-kappa-agonist, is usefully to partially antagonise undesirable effects of pure-mu opioids (e.g. dysphoria, delayed recovery, hypoventilation) while maintaining some analgesia.

- Induction agents that can be used include potent opioids of the fentanyl group (e.g. fentanyl, sufentanil), propofol or alfaxalone. Patients suffering a murmur of cardiac origin may have a decreased cardiac output and thus the perfusion to the central nervous tissue may be decreased. Therefore, careful titration of intravenous induction agents is always advised as there will be a delay in the onset of action. Volatile inhalation anaesthetics (isoflurane, sevoflurane) are the preferred maintenance agents because they provide good myocardial perfusion and rapid recovery.[6, 11]
- The routine use of alpha2-adrenoceptor agonists (xylazine, medetomidine, dexmedetomidine) are contraindicated in most cardiovascular cases and should only be used in cases where the pathophysiology of the disease process is well understood. These drugs cause a profound peripheral vasoconstriction, increase in blood pressure and reflex bradycardia during the initial phase of drug administration. Once the drug moves more centrally (central nervous system) there is a decrease in sympathetic tone which causes a decrease in vasoconstriction and relative increase vagal tone to the heart which maintains the bradycardia.[8]

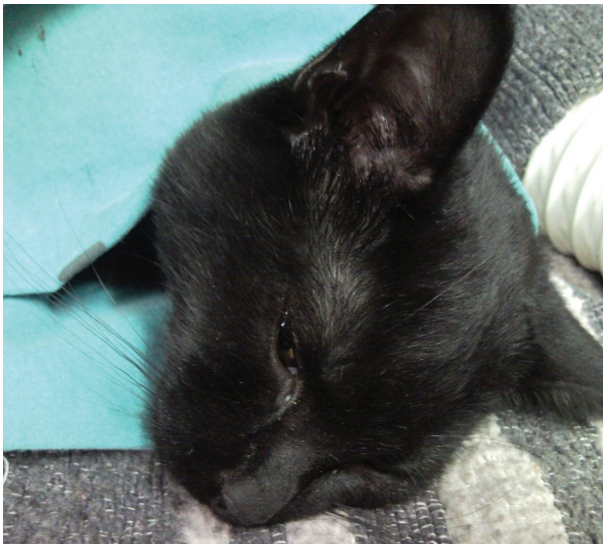

Figure 3: Maintaining normothermia with judicious use of blankets and heating devices.

Both the initial peripheral and delayed centrally mediated drug effects are not desirable in most patients suffering cardiac disease due to alterations in the systemic vascular resistance and heart rate. However, in certain cardiac pathologies where a murmur may be heard, such as hypertrophic cardiomyopathy in cats, medetomidine has been shown to decrease the dynamic outflow obstruction of the left ventricle and is useful in maintaining cardiac output.

- Making use of a familiar protocols to induce and maintain general anaesthesia is considered safer than using an unfamiliar protocol. However, there are physiological derangements which manifest from inappropriate drug selection which may be detected over time, such as an early cardiac patient presenting with acute renal failure, a stable patient rapidly deteriorating weeks after the general anaesthesia. Thus the immediate post- operative recovery for a poorly planned anaesthetic does imply that no "harm" has been done to the patient. Examples of less desirable anaesthetic protocols would include: xylazine-atropine premedication, thiopentone induction and maintenance, and protocols with no-premedication or analgesics.
- Always provide enough analgesia during the procedure using a multimodal approach where possible. Maintenance agents are designed to keep the patient asleep in an appropriate surgical plane (or lighter for diagnostic investigations such as radiographs). If the patient awakens often during surgical stimulation it is indicated to increase the analgesia and not the anaesthesia depth. In patients with cardiac disease a combination of opioids, local anaesthetics and judicial use of non-steroidal-anti-inflammatory (NSAIDs) drugs are preferred. Whereas healthy patients without other organ failure, a combination of opioids, local anaesthetics and routine use of NSAIDs should suffice.[7]
- Fluid management in patients with cardiac disease requires titration to individual needs. Ensuring that the intravascular volume is maintained throughout the general anaesthetic is important however fluid overloading is a real post-anaesthetic risk in patients suffering from cardiac disease. An early indicator of fluid overloading is an increase in the respiratory rate of at least 20% which is not due to surgical stimulation or depth of general anaesthesia. The effort of breathing will also be increased and referred breath sounds can be auscultated due to the increased sound carrying capacity of the lung tissue due to interstitial fluid accumulation. Late indicators of fluid overloading are jugular distension, increase in central venous pressure, auscultating for changes in respiratory sounds (crackles) and pleural effusions (especially in cats).
- A fluid rate of 10 mL/kg/hour could be started just after induction and titrated downwards to 5

mL/kg/hour during routine surgical procedures where minimal fluid loss and evaporation is anticipated.[5] Patients not on diuretic therapy often benefit from a low dose furosemide (1-2 mg/kg) twice daily for one or two days.

- Maintaining normothermia throughout the general anaesthetic and recovery is paramount and may be achieved by using blankets, forced warm air devices, warm water or electric (Hot Dog) blankets (figure 3). Recovery should be observed and be done in a quiet, warm environment where emergency drugs and equipment are at hand in case the patient crashes. The recovery period has the highest risk of death, especially in cats.[3]

During recovery from anaesthesia hypoxia is common and one of the main reasons for unexpected deaths and this necessitate vigilant monitoring until patient is fully recovered and conscious. The reasons for this are 1) functional residual capacity is reduced, 2) dampened ventilation response to carbon dioxide level, 3) decreased respiratory response to hypoxia at even sub- anaesthetic concentrations of anaesthetic argents (0.1MAC), 4) depression of the hypoxic pulmonary vasoconstriction reflex (this is the major reflex that matches blood flow to the ventilation of each alveoli), 5) CNS depression (residual drug in the system), 7) residual anaesthetic causes reduced strength in the diaphragm and intercostal muscles resulting in hypoventiliation, 8) reduction in compliance as a consequence of reduced FRC and the development of lung tissue atelectasis. 9) CNS depression (residual drugs) 10) Hypotension and decreased cardiac output, 11) hypothermia induced shivering which increases O2 consumption, 12) Pain, 13) Sympathetic nervous response and stress response both act to increase circulating catecholamines, 14) No sigh reflex (collapsed lung), 15) No voluntary changes in body position. [14]

6 "MUST KNOW" facts to consider in murmur patients undergoing general anaesthesia

1. A murmur is not necessarily a contraindication for general anaesthesia – it depends if the patient's cardiovascular system is stable. Differentiate between cardiovascular disease and cardiovascular failure.
2. The risk of general anaesthesia increases if the patient presents with concurrent disease processes and/or requires stabilisation prior to general anaesthesia.
3. Plan and prepare your procedures – this will keep your general anaesthesia to the shortest time possible.
4. Be patient with your patient – decreased cardiac output states delay the onset of action of the drugs, allow enough time for the drugs to reach clinical effect before administering additional boluses.
5. Fluid administration requires titration – even in stable patients.
6. Analgesia is paramount – Use opioids and local anaesthetics with judicial use of NSAIDs.

References available on www.vet360.vetlink.co.za

Dental procedures and older dogs .....

Exerpts from Senior Dental Care; Never too old for good dental health.
DVM 360 Dec 08, 2015. Heidi Lobprise.

The prevalence of periodontal disease increases with age and decreasing size of the animal i.e. older small breed dogs often require dental procedures. However older small breed dogs also often have heart murmurs due to mitral valve disease and may be on medication. Thus there is often a question regarding the safety of the anaesthesia in these patients.

It is only however in rare cases that the underlying cardiac disease would be so severe that anaesthesia should be avoided. However changes to standard anaesthetic protocols may be required as explained in Dr Zeilers article.

Maintaining the patients' body temperature during a dental procedure can be challenging as many patients are old and small, and the oral cavity is wet and being rinsed which makes the head of the patient also wet, causing heat loss. Geriatric patients may have exaggerated hypothermia resulting in a decreased metabolic rate. This can alter mentation, immune response and metabolism of drugs.

Keep the patient as dry as possible. Place patient warming devices under and on top of the patient.

Some dental procedures may be quite lengthy and in some cases it may be appropriate to stage the procedure.

Please Don't Leave me Alone!

Or I'll eat the house...

Jan 02, 2016
By dvm360.com staff
VETTED

Like mini-missiles set to mangle, pets with separation anxiety take it out on every floor and piece of furniture in sight.

Destruction, barking, whining, inappropriate elimination, excessive salivation—the clinical signs of this behaviour disorder are irritating at best. (At worst, of course, some sweet pet loses a home and never gets a second chance.) Veterinary behaviourist Dr. John Ciribassi offers these dos and don'ts to encouraging dogs' self-reliance, which helps keep the anxiety in check. Ideally, saving the banister.

Do: Have the owners ignore the dog upon arrival until he or she is relaxed. They shouldn't interact with or even acknowledge the dog.

Don't let owners further encourage the behaviour any more, ever. Owners should not respond in any way to a pet's attempts to get attention by such behaviours as barking, whining, jumping up and pawing. They should not look at, talk to or touch the dog when it is exhibiting these attention-seeking behaviours. Warn owners to expect the behaviour to initially get worse and more physical.

Do: Have the owners ignore the dog for 30 minutes before leaving the house to prevent inadvertent reinforcement of anxious behaviour as they prepare to leave. About five to 10 minutes before departure, the owners can give a toy stuffed with a treat to distract the dog from the act of the owners departing from the home.

Don't let punishment come into play. The owners should not use physical or verbal punishment in response to destructive behaviour or elimination. These behaviours are clinical signs of anxiety, so punishment, especially after the fact, will increase the dog's anxiety level.

Do: Work with the owners regularly on appropriate behaviour modification exercises involving indoor relaxation and graduated departures. Also prescribe anxiolytics as appropriate.

Do: Encourage the owners to provide consistent exercise in the form of walks and play, which can reduce anxiety by decreasing the dog's focus on the owner's departure from home.

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