Proceedings of the April Meeting of the Veterinary Cardiovascular Society
BSAVA Affiliated group

Wednesday 6th April 2016

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<tr>
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# Programme Veterinary Cardiovascular Society meeting

**Kingston Theatre, Austin Court, ICC, Birmingham**  
**Wednesday 6th April 2016, pre-BSAVA meeting**

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**Dr Roberto A. Santilli Dr MedVet PhD DipECVIM-CA (Cardiology)**

Roberto Santilli graduated from the Veterinary College of the University of Milan in 1990. He became a diplomate of the European College of Veterinary Internal Medicine - Companion Animals (Specialty of Cardiology) in 1999. In the period 2004-2006 he undertook a Masters degree in electrophysiology and electrical stimulation at the University of Medicine of Insubria. He obtained a PhD at the University of Turin – College of Veterinary Internal medicine in 2010. He directs the cardiology divisions of the Clinica Veterinaria Malpensa in Samarate, Varese (Italy) and of the Ospedale Veterinario i Portoni Rossi, Bologna (Italy). Since 2014 he has been Adjunct Professor of Cardiology at the Veterinary College of the Cornell University, New York – USA. His main research activities include the diagnosis and treatment of arrhythmias in dogs.

**Chris Booth BSc BVSc CertAVP(VC) MRCVS**

Chris graduated from Bristol vet school in 2004 and worked as a mixed practitioner in Somerset for four years. He then moved to the East Midlands and joined Oakham veterinary hospital, which is where he developed his cardiology interest. He received his CertAVP (VC) in 2014 and subsequently became an advanced practitioner in veterinary cardiology. The hospital is also a clinical associate of Nottingham University’s vet school and so integrates teaching students into his busy first and second opinion small animal and cardiac work.

**Xavier Navarro i Cubas DVM MRCVS**

Xavier graduated from the Cardenal Herrera-CEU University, Valencia, Spain, and went on to complete a general internship at the Autonomous University of Barcelona in 2008. He moved to the UK in September 2009, and worked in general practice for almost a year, before commencing a general internship at the University of Glasgow in September 2010. He then moved to Edinburgh and completed a cardiology internship at the University of Edinburgh. In 2016 he completed a Residency in Cardiology at the University of Liverpool and is currently working as a member of faculty as staff clinician.

**Dr Sonya Gordon DVM DVSc DipACVIM (Cardiology)**

Dr Sonya Gordon is board certified in cardiology by the American College of Veterinary Internal Medicine. She is currently an Associate Professor at Texas A&M College of Veterinary Medicine and Biomedical Science where she is part of a busy, progressive cardiology program. She is routinely an invited speaker at local, national and international veterinary meetings. Although she considers herself a clinician and teacher first, her research interests include canine chronic valve disease, dilated cardiomyopathy, cardiovascular imaging, interventional procedures and clinical trials. She has published numerous manuscripts and book chapters and, recently, co-authored a practical small animal clinical cardiology book entitled The ABCDs of Small Animal Cardiology. She considers her home College Station Texas, where she shares her life with her husband, four dogs and two cats.
Rachel James MA VetMB CertSAM DVC MRCVS

After graduating from Cambridge University in 1999, Rachel worked in small animal general practice for 4 years during which time she gained her certificate in Small Animal Medicine. She then undertook a residency in Veterinary Cardiology at the University of Liverpool, achieving her Certificate and then subsequently her Diploma in Veterinary Cardiology.

In 2007 Rachel established the Cardio-respiratory referral service at Nantwich Veterinary Hospital. From 2009-2013 Rachel worked at the University of Nottingham as an Associate Professor in Small Animal Medicine and achieved RCVS recognised specialist status in Veterinary Cardiology in 2009. Rachel is the current supplements editor for the Journal of Small Animal Practice and has previously been the secretary for the European Society of Veterinary Cardiology. Rachel currently works as a freelance cardiologist in the Northwest where she visits veterinary practices seeing cardiology referrals. Rachel still maintains close ties with Nottingham University participating in joint research projects: her research interests include syncope in dogs and feline cardiomyopathies.

Dr Amara Estrada DVM DipACVIM (Cardiology)

Dr Amara Estrada is an Associate Professor and Associate Chair at the University of Florida College of Veterinary Medicine who is board certified in veterinary cardiology by the American College of Veterinary Internal Medicine. She is also the Director of the Teaching Academy at the University of Florida, College of Veterinary Medicine. She graduated from the University of Florida before completing an internship at the University of Tennessee and a residency in comparative cardiology at Cornell University.

Dr Estrada is actively involved in all four years of veterinary student education and has won numerous teaching awards as well as awards for her clinical research both from the University of Florida and nationally. She has authored many peer reviewed research publications on veterinary cardiology and has contributed to numerous veterinary textbooks including Current Veterinary Therapy, Textbook of Veterinary Internal Medicine and the Clinical Veterinary Advisor. She is on the editorial board for the Journal of Veterinary Cardiology and frequently speaks at continuing education meetings both on a national and international level. She enjoys collegial collaborations with her colleagues both within the veterinary cardiology and paediatric cardiology disciplines.

When she is not working to advance veterinary student education or cardiology in clinical practice, Dr Estrada enjoys spending time with her husband and three incredible children.
Virginia is part of the Cardiology Service and is particularly interested in cardiomyopathies in cats and mitral valve disease in dogs. She has particular interests in progressing our understanding of the prevalence and progression of feline hypertrophic cardiomyopathy, and defining the key pathological changes in this disease. She is also interested in aortic thromboembolism, and in particular its pathogenesis and risk factors. Virginia also has interests in cross-sectional imaging techniques for the heart, such as cardiac MRI for mitral valve disease and CT angiography for congenital disease. Virginia hopes to improve the management of feline myocardial disease by participating in several prospective longitudinal clinical trials.
Inflammatory Cardiomyopathy in the Dog

Roberto Santilli
Clinica Veterinaria Malpensa, Varese, Italy
e-mail: rasantil@tin.it

In humans endomyocardial biopsy (EMB) is highly recommended in cases of unexplained left ventricular dysfunction associated with ventricular arrhythmias (VA) or high-grade atroventricular block (AVB). Despite the frequency of these conditions in dogs, histopathology data are lacking.

The aims of this study were to describe the feasibility of EMB in dogs and to investigate a possible role of viral myocarditis in case of unexplained dilated cardiomyopathy (DCM) phenotypes, high-grade AVBs, supraventricular arrhythmias (SVA) and VA. Twenty-five dogs of different breeds, M/F 1:5, mean age 5.95 ± 3.07 years, mean body weight 32.8 ± 11.52 kg, presented for third degree AVB 9/25, DCM 6/25, VA 6/25, SVA 2/25, and VA+SVA 2/25, underwent percutaneous right EMB under general anaesthesia throughout the jugular vein. For each dog clinical records were analysed. In all dogs at least one right ventricular sample (range 1-4) was collected for histopathology and immunohistochemistry; in 16/25 dogs at least one sample (range 1-3) for viral PCR was collected. All histopathologic samples were stained with haematoxylin and eosin, Masson's trichrome and red elastic picrocirus. In selected cases stains with monoclonal anti-CD3 and anti-CD79 were performed.

Nucleic acids were obtained after sample storage in RNA later solution, disruption with tissue Lyser and extraction with TRIzol; and tested for canine viruses (enteric and respiratory coronavirus, herpes virus, distemper virus, adenovirus 1 and 2, and parvovirus) and for West Nile virus and Bartonella spp.

Seven out of 25 dogs had non-specific signs of cardiomyopathy and 2/25 suggestive of arrhythmogenic right ventricular cardiomyopathy (ARVC). EMB gave normal samples in 6/25 dogs and were not diagnostic in 1/25 dog. Nine out of 25 samples were suggestive of myocarditis at different stages (3 third degree AVB, 5 DCM and 1 SVA). Two of these dogs resulted positive for virus (1 enteric coronavirus, 1 herpes virus). None of the dogs had positive immune-histochemical stains. Two dogs with cardiomyopathy were positive for herpes virus, and for herpes virus & parvovirus, respectively. Both of these dogs came from a kennel. No complication was noted in 24/25 dogs, one dog had a self-limiting pericardial effusion. This study showed, similarly to human cardiology, that EMB is a safe and useful technique that allows recognition and classification of unexplained myocardial and rhythm disorders, 25% of which were possibly associated with viral myocarditis. Further studies are needed to prove the relationship between viruses and myocarditis in a larger cohort of dogs.
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6- Kaneshige T, Machida N, Nakao S, Doiguchi O, Katsuda S, Yamane Y. Complete atrioventricular block associated with lymphocytic myocarditis of the atrioventricular node in two young adult dogs. J Comp Path 2007;137:146-150


9- Liu PP, Mason JW. Advances in the understanding of myocarditis. Circulation 2001;104(9):1076-1082

Heart disease is surprisingly common in the domestic ferret (*Mustela putorius furo*). In general the most common diagnoses are valvular disease, dilated cardiomyopathy (DCM) and arrhythmias (especially 2nd/3rd degree atrioventricular block). Hypertrophic cardiomyopathy (HCM) is rare. Neonatal heart disease is uncommon with occasional reports of Tetralogy of Fallot and ventricular septal defect in the literature. Neoplastic infiltration of the heart is unusual, although mediastinal lymphoma is fairly common. Myocarditis can be caused by bacteria, Toxoplasmosis and Aleutian disease. Aortic thromboembolism (ATE) is almost unheard of. *Dirofilaria immitis* can affect ferrets in parts of the world where this is endemic.

The normal ferret heart is heard between the 6th and 8th ribs, its rate being stated on average 180-250 beats/min in a leading ferret textbook, although several studies suggest values widely divergent from this (higher and lower). Some ferrets have a profound, clinically insignificant, sinus arrhythmia that needs to be differentiated from pathological arrhythmia. Murmurs can be difficult to localise specifically due to the ferret's small size, often a left parasternal holosystolic murmur is heard no matter which valve it originates from. S3 and S4 gallops can occur with DCM and HCM respectively as with other species.

Cardiac work-up requires a similar consideration to that of any other domestic species. Restraint can be challenging; many ferrets are wriggly, difficult to hold and can occasional exact a surprisingly strong bite. While chemical restraint may be preferable for radiography and the taking of a venous blood sample it may interfere with the results of an echocardiograph (echo) or electrocardiograph (ECG). Mild chemical restraint can be gained by using a combination of butorphanol 0.2mg/kg and midazolam 0.25mg/kg intramuscularly (IM), these medications are not licensed for use in ferrets and so consent should be obtained from the owner to use these under the cascade. However, the author has found the use of "Ferretone®" or a mixture of raw egg, lactose-free milk and olive oil given by an assistant to be an effective alternative. Ferrets seem to love this food mixture and will tolerate a number of interventions while lapping this up, the author has even achieved good quality conscious thoracic radiographs using this method. If chemical restraint is being used then fasting should be undertaken for no longer than four hours before sedation, as ferrets are prone to becoming hypoglycaemic after this point.

Radiography can be valuable, but there are important differences in ferrets compared to other domestic species because they have such a long trachea and thorax. At least two orthogonal views are taken - with right-lateral (RL) and dorso-ventral (DV) views the minimum required. The trachea should be parallel to T4/T5 and the heart should sit between ribs six and eight. The cardiac silhouette can often float in the thorax normally on RL view, giving the erroneous appearance of pneumothorax. Vertebral heart score (VHS) measurement is useful for gauging cardiac silhouette size, and can be done from a RL (preferable) or DV radiograph. To obtain the VHS the long axis and perpendicular short axis cardiac silhouette measurements are compared with the length of thoracic vertebrae T5-T8.
(cranial edge of T5 to caudal edge of T8)⁹. T5-T8 are, as a rule-of-thumb, about 1cm in length in most ferrets. Generally a RL VHS of 5-5.5 (i.e. 5-5.5cm) is considered usual, on a DV about 6 is considered usual. Radiographic signs to look for in cardiac disease are similar to those in dogs and cats.

For ECG measurement sedation should be avoided as this affects the ECG results and sedation will require some fasting. Fasted ferrets seem particularly prone to hypoglycaemia, which in turn can lead to AV block. Although RL recumbency may be the standard position for ECG in cats and dogs this position is difficult to maintain (and ECG clips difficult to keep attached) in a wriggly ferret. Having an assistant hold the ferret's scruff with one hand and supporting its bottom with the other (the so-called "hanging" position) is generally well tolerated and enables the ECG leads to be attached by a second individual – this technique is advocated by one study⁶. Generally the crocodile clips on standard ECG machines can be painful to ferrets so it is sensible to blunt the clips' teeth or alternatively use atraumatic clips (such as those recently given out by the manufacturer of a popular canine heart drug, which is what the author does). Ferrets dislike the smell and sensation of surgical spirit, so if it is to be used this should be applied to the clips before attaching them to the ferret. Ultrasound gel is a sensible alternative. The normal ferret surface ECG trace resembles that of a canine, although often with markedly taller R waves¹⁰. S-T segment elevation is not unusual in clinically healthy ferrets⁵. Table 1 contains a summary of ECG values in a study using conscious ferrets⁶, while other studies have measured these values in ferrets under sedation/anaesthesia⁷,¹¹.

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<th>Variable</th>
<th>Characteristic</th>
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<tr>
<td>P wave</td>
<td>Duration (seconds)</td>
<td>&lt;0.04</td>
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<tr>
<td></td>
<td>Amplitude (mV)</td>
<td>&lt;0.3</td>
</tr>
<tr>
<td>PR interval</td>
<td>Duration (seconds)</td>
<td>0.06-0.1</td>
</tr>
<tr>
<td>QRS</td>
<td>Duration (seconds)</td>
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</tr>
<tr>
<td></td>
<td>Amplitude (mV)</td>
<td>&lt;2.8</td>
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<td>QT interval</td>
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<tr>
<td>T wave</td>
<td>Amplitude (mV)</td>
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<tr>
<td>Mean electrical axis</td>
<td>Degrees</td>
<td>59-90</td>
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<tr>
<td>Heart rate</td>
<td>Beats/minute</td>
<td>Males 210</td>
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Table 1: Suggested ECG values for [ Einthoven's ] lead II based on the examination of 43 healthy conscious ferrets in the "hanging" position⁶
Echocardiography can be a rewarding imaging modality in ferrets, and can be done conscious wherever possible. The technique is similar to that adopted in a feline patient and the author finds that a high frequency probe and settings suitable for a feline patient will yield fairly good images in a ferret. The author has a GE Vivid 7®, and finds the 7S (at 8MHz) and 10S (at 10MHz) sector transducers useful. Scanning through a cut-out in an echo table gives optimal images in those ferrets that will tolerate this (judicious use of Ferretone® or eggy milk by an assistant can help with this). An alternative is asking an assistant to hold the ferret off the table in a horizontal orientation and scanning from the side. Recording a concurrent ECG trace is, in many cases, unrealistic in a conscious ferret, making echo measurements more difficult to standardise. Standard M-mode measurements of chamber and wall dimensions are usually taken, as are Doppler measurement of valves although this can be very tricky in a conscious patient. There are notable differences in echo findings between males and females and in both sexes the left atrial/aortic root diameter ratio often approaches 1.8 in normal patients (i.e. higher than cats and dogs). Table 2 summarises published echo values from normal conscious ferrets, not included are echo measurements that been recorded in ferrets under sedation, although a variety have been published.

**Table 2:** Suggested values for standard M-mode measurements from a right parasternal short axis view.

<table>
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<tr>
<th>Variable</th>
<th>Males (range)</th>
<th>Females (range)</th>
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<tr>
<td>LVIDd (mm)</td>
<td>13.0 (11.8-14.2)</td>
<td>10.4 (8.8-12.0)</td>
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<tr>
<td>LVIDs (mm)</td>
<td>8.9 (7.7-10.1)</td>
<td>7.1 (5.6-8.6)</td>
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<tr>
<td>LVWd (mm)</td>
<td>3.3 (3.0-3.6)</td>
<td>2.8 (2.4-3.2)</td>
</tr>
<tr>
<td>LVWs (mm)</td>
<td>4.4 (3.9-4.9)</td>
<td>3.6 (3.2-4.0)</td>
</tr>
<tr>
<td>IVSd (mm)</td>
<td>3.1 (2.8-3.4)</td>
<td>2.6 (2.4-2.9)</td>
</tr>
<tr>
<td>IVSs (mm)</td>
<td>4.1 (3.7-4.5)</td>
<td>3.4 (3.0-3.8)</td>
</tr>
<tr>
<td>LA (mm)</td>
<td>10.0 (9.1-10.9)</td>
<td>8.9 (8.0-9.4)</td>
</tr>
<tr>
<td>Ao (mm)</td>
<td>5.5 (4.9-6.1)</td>
<td>4.6 (4.2-5.0)</td>
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<tr>
<td>A. pulm (mm)</td>
<td>5.2 (4.7-5.7)</td>
<td>4.5 (4.1-4.9)</td>
</tr>
<tr>
<td>FS%</td>
<td>32 (25-39)</td>
<td>34 (28-40)</td>
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*LVIDd* - Left ventricular internal diameter end diastole, *LVIDs* - Left ventricular internal diameter end systole, *LVWd* - Left ventricular wall thickness end diastole, *LVWs* - Left ventricular wall thickness end systole, *IVSd* - Interventricular septal end diastole, *IVSs* -Interventricular septal end systole, *LA* - left atrial diameter end systole, *Ao* - Aortic diameter end systole, *A. pulm* - Pulmonary trunk diameter end diastole, *FS* - Fractional shortening.
Indirect blood pressure measurement via oscillometric or Doppler techniques is fraught with difficulty. One study found a size 1 cuff too large and so consistently underestimated systolic arterial blood pressure. Little information, therefore, has been published regarding what is considered normal in the ferret. Cardiac biomarkers, similarly, have not yet been validated for ferrets so their use is limited. The measurement of resting or sleeping respiratory rate (sRR) to monitor the advent and progression of CHF has been shown to be useful in dogs and cats. While the author could find no published records regarding these indices in ferrets the he has found sRR increases as CHF progresses and measurements above 40 breaths/minute are a cause for concern.

The most common myocardial disease in ferrets is DCM. Its cause is unknown, with no evidence of the taurine related issues reported in cats and some dog breeds. Genetic, viral and drug-related causes have been postulated - the author has had one case in a ferret with lymphoma that developed CHF due to DCM not long after doxorubicin therapy was initiated - begging the question whether the lymphoma or its treatment (or a combination of the two) had caused this. The author has had another case of DCM that developed about a year after the ferret in question had had an exploratory laparotomy to remove a ruptured intestinal abscess, whether that previous condition had any bearing on the development of DCM is unknown.

DCM can be difficult to diagnose in the pre-clinical stage, as only some patients have murmurs; some ferrets may only be diagnosed when they develop congestive heart failure (CHF). On thoracic radiography there can be cardiomegaly, dorsally displaced trachea, atrial enlargement and signs of left or right CHF. On echo fractional shortening is usually well below normal limits and there will be increased internal dimensions of left and right ventricles. The pathogenesis of DCM in ferrets is similar to that in the dog. Valvular regurgitation and heart murmur (as well as an S3 gallop) can occur with DCM, although regurgitant jets are usually small and central with DCM. The treatment of DCM depends on the degree of CHF. The most common signs of CHF are hyperpnoea/shortness of breath, weakness, exercise intolerance, pale or cyanotic mucous membranes, hind-limb weakness, pulse abnormalities and hypothermia. Pleural effusions, pulmonary oedema and ascites are particularly common manifestations of CHF due to myocardial disease. If pleural effusion is present this should be removed via thoracocentesis (butorphanol/midazolam sedation - see above for doses - can provide fairly safe chemical restraint). Ascites can seriously affect quality of life and so consideration should be given to draining this if it is excessive (the author has drained 1000ml of ascitic fluid a ferret with DCM, causing a substantial increase in quality of life and appetite).

Table 3 lists common heart drugs and their doses in ferrets. Without exception their use is off-label and via the cascade and so informed, written, consent is needed from the owners before use. Ferrets with DCM tend to respond to medical management at least as well as dogs and cats do. In DCM with CHF pimobendan, ACE inhibitors, furosemide and spironolactone can be useful. If DCM becomes refractory then the author has found adding in amiloride/hydrochlorothiazide at cat doses can help to stabilise the patient. Ferrets seem to be fairly resistant to the hypokalaemic effects of loop diuretics and so potassium supplementation of ferrets on furosemide is not usually necessary, when it is required "Kaminox", VetPlus® can be used -the author uses this in the presence of cachexia. Atrial fibrillation is a very rare complication of DCM in ferrets – when it occurs digoxin elixir can be used.

Valvular disease is common in ferrets. A clinical normal ferret can have small regurgitant jets from any of its valves. Valvular regurgitation is most common in the aortic valve (AV), followed by mitral valve (MV) with tricuspid (TV) and pulmonic valves (PV) a joint third. Generally AV regurgitation is insignificant unless there is concurrent MV regurgitation. CHF is much more likely to be associated
with MV regurgitation, and rarely with disease of any other valve. Radiographic and echocardiographic findings of MV disease are similar to those in the dog. Treatment is limited to individuals showing evidence of CHF and is similar to that given to dogs (see Table 3 for doses).

Arrhythmias are fairly common in ferrets. One source found second degree AV block and sinus arrhythmia the most common arrhythmias in a retrospective case series of 95 ferrets. Third degree AV block can occur, and can be debilitating - requiring pacemaker treatment. Tachyarrhythmias are incredibly rare, atrial fibrillation and supraventricular tachycardia have been reported.

HCM is rare in ferrets, the author has not seen a case. ATE has not (to the author’s knowledge) been reported so anti-thrombotics are not indicated as a preventative in left atrial enlargement.

The author has found cardiac cachexia as common in ferrets as it is in dogs. Supportive care is required during these stages to help slow weight loss. The use of eggy milk/Ferretone® to help increase calorie intake can be useful. Adding in a vitamin supplement, e.g. Kaminox®, VetPlus (at the low end of the cat dosage) and/or vitamin B12 injections, can help to maintain interest in food. Convalescent diets (e.g. Hills® a/d) can be added in and ferrets can usually tolerate some degree of syringe-assisted feeding.

Table 3: Commonly used drugs and doses for ferrets with cardiac disease. The amiloride/hydrochlorothiazide dose is the author’s empirical dose and is similar to the off label feline dose. Doses are taken from a number of sources.*

<table>
<thead>
<tr>
<th>Drug*</th>
<th>Indication</th>
<th>Dose</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiloride/hydrochlorothiazide</td>
<td>Refractory CHF</td>
<td>2-4mg/kg po</td>
<td>Q24h</td>
</tr>
<tr>
<td>Amlodipine²</td>
<td>Afterload reduction</td>
<td>0.1-0.4mg/kg po</td>
<td>Q24h</td>
</tr>
<tr>
<td>Atenolol²</td>
<td>SVT</td>
<td>3.125-6.25mg/ferret po</td>
<td>Q24h</td>
</tr>
<tr>
<td>Benazepril²</td>
<td>ACEi: CHF</td>
<td>0.25-0.5mg/kg po</td>
<td>Q24h</td>
</tr>
<tr>
<td>Digoxin (elixir)²</td>
<td>Atrial fibrillation</td>
<td>0.005-0.01mg/kg po</td>
<td>Q12h</td>
</tr>
<tr>
<td>Diltiazem²</td>
<td>SVT</td>
<td>1.5-7.5mg/kg po</td>
<td>Q12h</td>
</tr>
<tr>
<td>Furosemide oralsuspension¹⁶</td>
<td>CHF</td>
<td>1-2mg/kg po</td>
<td>Q12h</td>
</tr>
<tr>
<td>Furosemide injectable²</td>
<td>Acute CHF</td>
<td>2-4mg/kg iv</td>
<td>Q8-12h</td>
</tr>
<tr>
<td>Imidapril suspension¹⁶</td>
<td>ACEi: CHF</td>
<td>0.25mg/kg po</td>
<td>Q24h</td>
</tr>
<tr>
<td>Pimobendan¹⁶</td>
<td>Systolic dysfunction</td>
<td>0.5mg/kg po</td>
<td>Q12h</td>
</tr>
<tr>
<td>Spironolactone²</td>
<td>CHF/Aldosterone escape</td>
<td>2-4mg/kg po</td>
<td>Q24h</td>
</tr>
</tbody>
</table>

ACEi - ACE inhibitor, CHF - congestive heart disease, iv - intravenous, po - per os, SVT - supraventricular tachycardia. * All these medications are off-label and should be used with informed (written) consent via the cascade.
To summarise: a few tips the author would emphasise in the diagnosis and management of heart disease in ferrets

- The use of Ferretone® or eggy milk is a substitute for sedation in many cases
- Thoracic radiographs are quite different to dogs and cats, with a much lower VHS and the presence of the cardiac silhouette being lifted off the sternum is often considered normal (not to be confused for pneumothorax).
- ECG is easier and well tolerated by the ferret in the "hanging" pose rather than being held in RL recumbency
- DCM and valvular disease are most common, mitral valve disease is the most significant valvular disease, aortic valve regurgitation is usually insignificant
- Treat CHF due to valvular disease or DCM as you would in the dog, titrate doses up as required and as owners to monitor resting/sleeping respiratory rate. It can be difficult to know what to do when diagnosing pre-clinical DCM or valvular disease as acute CHF can occur with little warning. The author usually starts low dose CHF treatment in such cases.
- Drain all pleural effusions and any ascites that is affecting the ferret's ability to move or eat - then increase diuresis or add in amiloride/hydrochlorthiazide
- Ferrets generally respond very well to CHF treatment with life expectancies comparing favourably with clinically similar dogs

References

Can we do better than QUEST? Triple therapy plus for the treatment of degenerative mitral valve disease

Xavier Navarro | Cubas
University of Liverpool, United Kingdom
Let sleeping dogs lie: Anaesthesia in canine cardiac patients

Sonya Gordon
Texas A&M University, United States of America

The anaesthesia of dogs with heart disease is challenging. Most anaesthetic agents reduce contractility (negative inotropic properties) and heart rate (negative chronotropic properties) and cause arterial dilation (reduction in vascular resistance/afterload) leading to variable degrees of systemic hypotension. These effects are dose dependent and predictable and thus systemic hypotension and the resultant poor perfusion are expected possible/probable complications of anaesthesia in any patient. In cardiac patients the severity of these changes maybe more significant and approaches to mitigate them are limited when compared to those routinely employed in dogs with normal cardiovascular systems. For example boluses of fluids and routine use of prophylactic positive inotropes may be contraindicate in some dogs with heart disease. An understanding of the nature and severity of each dogs underlying cardiac disease, indication (what surgery will be performed) and duration of anaesthesia/sedation required and an appropriate monitoring plan can lead to rapid management of expected complications. Most anaesthetic catastrophes can be predicted and avoided with proper panning. Best approach is like that of a Boy Scout “be prepared’. This session will use a variety of clinical scenarios requiring anaesthesia to outline a practical clinical approach with emphasis on prediction and prevention of complications.

Anaesthesia Scenarios:

- Repair of a PDA
- Palliative procedure for severe PS or SAS
- Dental or lump removal in an adult dog with Stage B2/C chronic valvular disease

Anaesthesia Generalities:

- Ensure patient is as clinically stable as possible
- Patient specific premedication
- Pre-oxygenation
- Minimize use of agents that cause myocardial depression and hypotension
  - Balanced protocol
    - Local blocks when possible
    - Premedication
    - Intra-operative intravenous agent(s) can decrease inhalant MAC of inhalant (inhalant sparing agents) by 30-75% or 100% when a total intravenous anaesthesia (TIVA) protocol is used. Use of these agents will cause dose dependent reductions in HR and spontaneous ventilation and may require mechanical ventilation. In addition, whether you are using them as an intra-operative CRI for inhalant MAC reduction or as TIVA you will need to reduce the CRI by as much as 50% after 60 min to avoid excessive build up. Reversal agents should always be on hand when using these agents clinically.
<table>
<thead>
<tr>
<th></th>
<th>MAC with no intra-op CRI</th>
<th>MAC with intra-op CRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sevoflurane</td>
<td>2.3%</td>
<td>0.5%</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>1.3%</td>
<td>0.2%</td>
</tr>
</tbody>
</table>

- CRI of fentanyl & midazolam as part of balanced protocol
  - Fentanyl CRI 5-48 ug/kg/min
  - Midazolam 0.8 ug/kg/min
  - Ventilation may be required
- For TIVA
  - Fentanyl CRI doses atypically increased to approximately 48 ug/kg/min
  - Midazolam CRI dose rate unchanged
  - Ventilation typically required
  - Use sevoflurane instead of isoflurane if possible

<table>
<thead>
<tr>
<th></th>
<th>Contractility</th>
<th>Vascular Resistance</th>
<th>Systemic BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Halothane</td>
<td>Severe reduction</td>
<td>Minimal reduction</td>
<td>Dose dependent reduction</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>Mild reduction</td>
<td>Moderate reduction</td>
<td></td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>Mild reduction</td>
<td>Moderate reduction</td>
<td></td>
</tr>
</tbody>
</table>

Note: all cardiovascular affects are dose dependent.

- Avoid agents that cause increased myocardial work (O2 consumption)
  - Alpha 2 agonists, ketamine, anticholinergic
- Appropriate monitoring:
  - Pre operatively
    - HR and rhythm
    - Respiration rate
    - Anxiety/stress/pain level
  - Intra operatively
    - Blood pressure
    - Heart rate and rhythm (ECG)
    - Oxygenation (pulse oximetry)
    - Body temperature
    - Appropriate depth of anaesthesia for surgical stimulus
  - Post operatively
    - Respiration rate and effort
    - Heart rate and rhythm (ECG)
    - Body temperature
    - Body temperature
    - Anxiety/stress/pain level
Practical Tip:

1. *Most life threatening complications occur post-op, during recovery, and can be mitigated or prevented with monitoring*
2. *Extubation does not equal recovery!*

Sedation Scenarios:

- Echocardiogram
- Radiographs

Sedation Generalities:

- Avoid agents that cause increased myocardial work (O2 consumption)
  - Alpha 2 agonists, ketamine
- Opioids are good choices alone or in combination with other agents
  - Avoid those that cause excitement, panting, nausea or respiratory depression
    - Butrophanol & buphrenorphine are good choices
- Acepromazine is good in combination with an opioid if an opioid not enough alone
  - Low dose-0.02-0.03 mg/kg
- Midazolam is good in combination with an opioid

Take-home messages:

Know what to expect & plan accordingly

- What procedure/surgery is being done
  - Expected duration
  - Level of pain-opportunity for local blocks or epidural
  - Risk of arrhythmias-catheter stimulation
- Is the dog clinically stable
  - If not could we stabilize prior to anaesthesia?
- What if any chronic therapy is the dog receiving
  - Do we need to adjust or discontinue and chronic medications
    - Atenolol, sotalol, angiotensin enzyme inhibitor, pimobendan, furosemide etc.
- Is the dog at risk for CHF (cardiogenic pulmonary oedema)
  - Prior dx of CHF
  - Left to right shunt
  - Mitral regurgitation (+/- left atrial enlargement)
  - Myocardial systolic dysfunction
- Does the dog have pulmonary hypertension
  - Pre-oxygenation critical
  - Background/chronic medications
- What is the pre-op BP
- If the dog becomes hypotensive intra-op how can we stabilize
  - What is the specific plan?
- What is the pre-op HR and rhythm
  - e.g. does the dog have known/chronically managed arrhythmias
- What is the code/owners wishes if arrest occurs?
Be a 'Boy Scout' ....be prepared...plan

1. How will you respond to systemic hypotension and what is the threshold for Tx
   • Fluids, anticholinergics, dobutamine (dopamine), combination
2. How will you respond to bradycardia and what is the threshold for Tx
   • Maintain body temperature, anticholinergics
3. How will you respond to new or worsening arrhythmias
   • Ventricular- IV lidocaine bolus and CRI
   • Supraventricular (Afib)-acute-IV procainamide (15-25 mg/kg over 10 min)

Components of an optimum anaesthetic plan for a dog with cardiac disease

1. Induction with
   o Midazolam (or diazepam) + etomidate
2. Maintenance with
   o Sevoflurane
   o CRI of fentanyl + midazolam
3. Monitor!!!!
Arrhythmia induced cardiomyopathy

Roberto Santilli
Clinica Veterinaria Malpensa, Varese, Italy
e-mail: rasantil@tin.it

Introduction

The presence of uncontrolled tachycardia, either supraventricular or ventricular, can induce an impairment of left ventricular (LV) function defined tachycardia-induced cardiomyopathy (TICM). (1-6) Tachycardia-induced cardiomyopathy is characterized by different degree of chambers dilation with systolic and diastolic ventricular dysfunction and congestive heart failure. (1-6) Systolic dysfunction is usually transitory with complete recovery after controlling the rhythm disorder. (4) Lately the term TICM has been modified to arrhythmia-induced cardiomyopathy (AIC) to include atrial and / or ventricular dysfunction secondary to rapid, asynchronous or irregular rate that can be completely or partially reversible after treatment of causative arrhythmia. (6) In AIC should also be included cardiomyopathies caused by asynchronous myocardial contraction, resulting from premature ventricular contractions (PCV) or right apical ventricular pacing, that can lead to a completely or partially reversible myocardial dilatation and symptoms of congestive heart failure. (7-8)

Arrhythmia-induced cardiomyopathy may finally be divided in a pure form in which ventricular dysfunction is present in normal hearts and is characterized by a complete recovery, and in an impure form in which ventricular dysfunction is present in patients with underlining cardio-structural disease, for whose recovery is often incomplete. (9)

Rate-dependent changes

Several experimental studies have investigated the pathogenesis AIC during of rapid, asynchronous or irregular pacing. (10-11) These experiments have provided valuable information on the effects of rapid atrial or ventricular stimulation, resulting in systolic and diastolic dysfunction. The systolic and diastolic dysfunction was more pronounced when induced by ventricular pacing, while its severity was linked to the duration of tachyarrhythmias. (10-11) A pacing continues with more than 240 beats / min for 3 weeks induced a decrease in cardiac output, a dilation of the left ventricle, reduced systolic function and diastolic dysfunction, and neuro-humoral abnormalities similar to those present in the course of human dilated cardiomyopathy. (10-11) After the cessation of stimulation, there has been a rapid recovery of systolic function, and diastolic function remained abnormal. (10-12) Rapid, irregular or asynchronous pacing induces changes in myocardial structure and function, neuro-humoral disorders, and changes at the microscopic level, with depletion of energy reserves (creatine,
phospho-creatine and adenosine triphosphate) and myocardial ischemia. (11,13-15) They also highlighted lower levels of Na - K activity - ATPase, as a result of increased activity of the enzymes of the Krebs cycle. (11,13-15) Ischemia is induced by structural and functional alterations of the capillary network of the myocardium, with impaired blood flow reserve. (11,13-15) A form of myocardial hibernation may be the reason why these changes are partially or fully reversible after termination of the arrhythmia. Other assumptions include a decrease in the density of B-receptors, the occurrence of an oxidative stress, which contributes to myocardial damage through an imbalance between pro- and anti-oxidants and a decrease in the density of the T-tubules and L - Ca channels, which contribute to the coupling abnormality. (16) The myocardial hypertrophy in the course of rapid stimulation seems to be associated with activation of some mechanosensors (Icirc). Once developed AIC induces an electrical remodelling of ion channels (Ito, Ikr, Iks and Ca2 +) that promotes ventricular arrhythmias. (17)

**Asynchronous contraction**

The asynchronous contraction of the myocardium as in the case of bundle branch block or right ventricular pacing alters the normal activation of the myocardium by the His- Purkinje system. During asynchronous contraction workload is redistributed with loss of contractile strength. (18) During right ventricular pacing adrenergic innervation of the ventricle is altered in the vast majority (89.7%) of patients particularly in regional area of the lower portion (92.3%) and apical portion (38.5%) of the LV wall. (19) Myocardial perfusion defects have been proven in up to two thirds of patients with chronic right ventricular apical pacing. (20) The same mechanisms seem to be the cause of PVC-induced cardiomyopathy. The number of PVC, expressed as a percentage of PVC on the total number of QRS complexes or as a sum PVC per day are used as criteria to define cardiomyopathy PVC induced. (21) Many studies have identified the percentage of ectopics that can induce the PVC-induced cardiomyopathy (> 24% or > 20% if LV originating PCV and > 10 if right ventricular originating PCV), or the prevalence of cardiomyopathy related to the absolute number: 1000/24 hours (4%), 1000-10 000/24 hours (12%), and >10000/24 10 hours (34%). (22) Another cause of asynchrony is atrial fibrillation which induces a loss of atrial contraction and a reduction of 15 - 20% of the cardiac output with a concomitant alteration of LV filling times. It has been shown that even a sequence of irregular RR intervals has the hemodynamic consequences independently of heart rate. Implicated mechanisms are alterations of myofibres length and the force of contraction relationship. (23)
Atrial myopathy

During incessant tachycardia, the atrium remolds electrically and anatomically. During the high rates an alteration of calcium handling occurs, which induces a deficiency of L-type channels, together with phenomena of apoptosis, cell death and inflammatory infiltration. (24)

Diagnosis of arrhythmia-induced cardiomyopathy

The hemodynamic changes begin as early as 24 hours post-stimulation at rapid rates with a decrease in blood pressure and cardiac output and increased in right atrial and wedge pressure. These variations will last for at least 3 weeks, and the size of the left ventricle remain altered with a slow reverse remodelling which can last 3 months. (1-12)

A definitive diagnosis of AIC is difficult. The suspected diagnosis can only be confirmed by a normalization or improvement of impaired LV function after the control of tachyarrhythmias. However, it is also true that the tachyarrhythmia control does not always contribute to the improvement of left ventricular function in patients with AIC, and may simply reflect the irreversible stage of a pure AIC or an impure AIC. (9) Some authors have proposed the following criteria for the diagnosis of AIC: 1) dilation or heart failure, 2) chronic cardiac arrhythmias or very frequent with incessant behaviour emphasizing the concept that if chronic tachycardia is not constant but lasts more than 10-15% of the day, with an atrial rate of over 150% of that expected for age, may also cause AIC. (9)

The echocardiographic data obtained in humans with AIC have shown that LV cavitary diameters are smaller than in the case of dilated cardiomyopathy, and rhythm control induced an improvement of at least 15% compared to 5% in the case of dilated cardiomyopathy. (25)

Treatment

The basic concept for the treatment of AIC is heart rate and asynchrony control respectively with antiarrhythmic drugs, ablation techniques and re-synchronization therapy with biventricular pacemaker. The most common antiarrhythmic drugs used for supraventricular tachycardias are diltiazem, verapamil, digoxin and sotalol, for ventricular sotalol and amiodarone. The advent of radiofrequency catheter ablation in veterinary medicine allowed to control permanently many rhythm disturbances both supraventricular (focal atrial tachycardia, typical and atypical atrial flutter, and bypass tract-mediated tachycardia) and ventricular tachycardia. (26-33).
References

Spironolactone (SP) is an aldosterone receptor antagonist, registered in Europe for the treatment of congestive heart failure (CHF) caused by valvular regurgitation in dogs, in combination with standard therapy. In cats, cardiomyopathy (CM) is the predominant cause of heart failure. To evaluate the safety and efficacy of SP in cats with CM, a double blind, randomized placebo-controlled study has been conducted with cats receiving either SP (1.7 to 3.3 mg/kg PO once daily) or placebo for up to 15 months in addition to benazepril and furosemide (dose at clinician’s discretion). 20 cats (17 DSH, 1 ragdoll, 1 Siamese and 1 Burmese) with CM of various types (15 hypertrophic, 2 dilated, 2 unclassified and 1 Arrhythmogenic Right Ventricular) were enrolled.

The cats were randomized to either group A or B according to the presence of HCM or not and whether the cat required hospitalization due to clinical need or not. 9 cats were recruited to group A (SP) and 11 cats recruited to group B (placebo). The only significant difference between the two groups at baseline were aortic diameter (p=0.0077) larger in the SP group, and LA:Ao ratio (p=0.012).

The survival analysis showed a survival rate respectively of 78% and 71% in the intention to treat (ITT) and per protocol (PP) populations in the SP group and 12% and 14% in the placebo group. The difference between the two groups was significant (Log rank test: ITT population p=0.011; PP population p=0.033). The hazard ratio indicates an 84% (ITT) and 80% (PP) reduction in risk of an event occurrence in the SP group. The effect of covariates (age, weight, BCS, systolic blood pressure, ratio LA/Ao) was not significant.

Although this is a pilot study with small numbers of cats, this data would suggest that spironolactone is likely to be beneficial in the treatment of cats with congestive heart failure secondary to a cardiomyopathy.
Balloon Valvuloplasty: who, why, when and how?

Amara Estrada
University of Florida College of Veterinary Medicine, Gainesville, USA

Pulmonic stenosis and subaortic stenosis are both outflow tract obstructive congenital lesions which are often treated with balloon dilation, both in paediatrics and in veterinary medicine. Firm guidelines for deciding which animals need to be treated have not been developed. In paediatric cardiology, most interventionalists agree that treatment should be performed in any child with moderate stenosis, or a gradient greater than 50 mm Hg. This is not as clear for veterinary patients as we have no long term studies to tell us that patients do better in the long term following balloon dilation.

Pulmonic Stenosis (PS)

Firm guidelines for deciding which animals need to be treated have not been developed. In pediatric cardiology, most interventionalists agree that treatment should be performed in any child with moderate stenosis or a gradient greater than 50 mm Hg. Most veterinary cardiologists would agree that animals with gradients in the severe range should be treated and a recent study showed that the due to a greater risk of cardiac death, dogs with a gradient greater than 60 mm Hg may benefit from intervention. Several other factors can also be taken into consideration when making the decision for treatment, such as degree of RV hypertrophy, presence of clinical signs, and severity of concurrent tricuspid insufficiency.

Pulmonary balloon valvuloplasty is the first line of treatment for PS. Valvuloplasty should be performed in any symptomatic patient as soon as PS is diagnosed. Even asymptomatic patients should be treated as soon as possible. Delay in treatment can lead to progression of RV and infundibular hypertrophy, not only making the procedure technically more difficult, but also diminishing the immediate response to therapy. In fact, in some patients with significant infundibular hypertrophy, this subvalvular gradient can become acutely more severe following dilation of the stenosed pulmonary valve. This condition, termed *suicide right ventricle*, occurs immediately following relief of severely stenosed valves. Right ventricular pressures dramatically increase as the hypertrophied infundibulum creates subvalvular obstruction that can sometimes become worse than the valvular obstruction. Therapy with intravenous β-blockers (esmolol 0.05 to 0.5 mg/kg slowly IV as a bolus) and volume loading of the right ventricle with intravenous fluid boluses can improve or alleviate this subvalvular obstruction.

As described previously, subvalvular PS in English bulldogs and boxers can be caused by an anomalous left coronary artery. Recognition of this abnormality on echocardiographic or angiographic studies is imperative because balloon dilation in these patients has been reported to cause rupture of the artery and sudden death of the patient. Balloon dilation in these patients is risky, and surgical conduit placement around the stenosis may be the only alternative. Less aggressive balloon dilation with a balloon that is 0.8 to 1.0 times the size of the pulmonary annulus may also be performed in dogs with severe stenosis, right-sided congestive heart failure, or other clinical signs. This procedure carries the same risks, but has been reported to be safe in four English bulldogs with severe PS and an anomalous left coronary artery.
Interventional catheter-based procedures should only be performed by veterinarians with advanced training in these techniques. The technique for pulmonary balloon valvuloplasty involves placement of a dilation balloon across the stenosed valve. The entire procedure is performed under fluoroscopic guidance. A balloon that is 1.2 to 1.5 times the measured pulmonary annulus (from either echocardiographic or angiographic studies) is most often used. In the author’s practice, when the annulus diameter exceeds 18 mm, we recommend a double balloon technique, which allows the use of smaller introducer and balloon sizes. Since the entire outflow tract is not obstructed as the balloons are inflated in this technique, we find that it is easier to maintain the balloon position across the valve and this also allows better maintenance of cardiac output. Additionally, the double ballooning technique (versus one large balloon alone) allows for generation of more pressure to effectively dilate the stenosed valve and inflation and deflation of a single large balloon also takes a considerably longer amount of time than two smaller balloons. When using a double balloon technique, the effective balloon dilation size is 0.82 times the sum of the two balloon diameters.

The balloon dilation catheter is placed from either a jugular or femoral vein using a vascular cut-down or percutaneous approach. Once the balloon is positioned across the stenosed valve, it is inflated with fluid under pressure to stretch or break open the valve. The balloon is kept inflated for only a few seconds. Multiple inflation-deflations are performed until a satisfactory degree of dilation is achieved. When the balloon is inflated initially, there is an indentation in the balloon at the region of the stenotic valve. This is called a waist. Successful dilation is achieved with visualization of a loss of this waist during inflation. On successful balloon dilation, RV pressure and transvalvular gradient decrease. This decrease following dilation can be detected immediately with intra-cardiac pressure monitoring. While standard balloon dilation catheters have relatively low burst pressures (1.5-6 atm), higher pressure balloon dilation catheters (8-20 atm) are now available and may be useful for resistant or recurrent PS. These high pressure balloons require a specially designed inflation device with a pressure gauge for optimal use. The higher pressure generated by these balloons with an inflation device often results in successful dilation of the valve when a lower pressure balloon does not. Most recently, other dilation techniques, including the use of stents and cutting balloons (balloon dilation catheters with small blades mounted to the balloon), have been explored for the most severe and resistant PS lesions.

In pediatric cardiology, a good outcome is defined as a residual Doppler gradient of 35 mm Hg or less. In veterinary cardiology, reduction of the gradient to less than 50 mm Hg or 50% of the original value is generally thought of as successful. Complete resolution of gradients across the valve in these patients is not possible because most valves are somewhat dysplastic and always cause at least some obstruction to flow. Accurate assessment of results requires evaluation of Doppler-derived gradients or gradients indexed to stroke volume in the ensuing weeks to months after catheterization.

Serious complications that can occur during this procedure are rare, but include cardiac perforation causing pericardial effusion and tamponade, rupture of the pulmonary artery, suicide right ventricle, and fatal arrhythmias. Minor complications that occur with more frequency include damage to the tricuspid valve, creation of a right bundle branch block, temporary arrhythmias, and hemorrhage from vascular access sites. These latter complications are rarely clinically important, and most patients are discharged from the hospital the following day.

Intra-cardiac pressure measurements are usually taken immediately before and after balloon dilation to guide therapeutic decisions. If the gradient or RV pressure has not decreased satisfactorily and there is not dynamic obstruction to explain the gradient, a larger or higher pressure balloon may be...
placed, and the procedure repeated. Alternatively, stenting of the right ventricular outflow tract for particularly dysplastic valves can also be performed in select cases.

Echocardiographic studies are performed the day following the procedure. All measurements are repeated, with specific attention to transvalvular gradients and amount of pulmonary insufficiency and tricuspid insufficiency. Pulmonary insufficiency is virtually always increased following balloon valvuloplasty, but is well tolerated by the right ventricle. Tricuspid insufficiency is usually diminished because of the drop in the RV pressure created by the valvuloplasty procedure. Gradients measured the day after the procedure may not show as dramatic a drop as those measured immediately following the procedure. Pulmonary valve leaflets become swollen and edematous following balloon dilation and may temporarily increase the measured gradient until the swelling has subsided. Furthermore, stroke volume greatly influences gradients and is typically lower in dogs under general anesthesia.

Long-term follow-up studies in pediatric cardiology show excellent results. Several recent veterinary studies have also shown that pulmonary balloon valvuloplasty significantly reduces transvalvular gradients, clinical signs, and risk of cardiac death in dogs with severe PS. Recheck evaluations are typically performed 3 and 6 months after the valvuloplasty procedure and then annually. Gradients can continue to decline in the first 3 to 6 months and possibly even longer as resolution of valve edema and regression of RV/infundibular hypertrophy occurs; however, restenosis is also possible over the same time period. Therefore, determination of whether a valvuloplasty procedure has been successful should not be judged until at least several months following the procedure. Some cardiologists continue β-blockade in dogs with residual hypertrophy and dynamic RV outflow obstruction.

**Subaortic Stenosis (SAS)**

The pathophysiology of severe SAS involves concentric hypertrophy of the left ventricle and eventual left-sided congestive heart failure, myocardial ischemia, coronary arterial sclerosis, and cardiac arrhythmias that can result in sudden death, often before three years of age. Median survival dogs with severe SAS who are left untreated is only around nineteen months. Dogs with mild forms of the disease may only develop minimal ventricular hypertrophy, and the pressure gradient often remains below 50 mmHg. These dogs have a better chance at living normal lives. Therapy options, for dogs with severe SAS, have included atenolol therapy, low pressure balloon valvuloplasty, and open heart correction.

One study looked at a group of dogs treated with low pressure balloon valvuloplasty, along with a group of dogs treated with atenolol only. The results of this study showed that median survival time for the group of dogs receiving atenolol only was 56 months; this did not vary significantly from a median survival time of 55 months for dogs treated with low pressure balloon valvuloplasty alone. It also showed that low pressure balloon valvuloplasty can result in a significant decrease in the pressure gradient in dogs with severe SAS for up to six weeks post-procedure; however there did not appear to be any benefit in survival time for dogs treated with low pressure balloon valvuloplasty versus treatment with atenolol only. However, since the dogs were only evaluated echocardiographically at six weeks post-procedure, the reason for similar survival outcomes is unclear. No record was available to say that the dogs that underwent the balloon valvuloplasty re-stenosed (pressure gradients increased) or that their reduced pressure gradients were maintained over their lifetime. In addition,
adding a group of dogs treated with both balloon valvuloplasty and atenolol would have been useful to look for complimentary effects of combining treatments.

Open heart techniques often involve higher perioperative mortality rates when compared to catheter procedures. This rate was 18.2% in one study. The median survival time for the group of dogs undergoing surgery in this study was approximately 45 months. This study has shown that despite initial reductions in the systolic pressure gradient, a benefit for survival was not documented in dogs undergoing surgery versus just being treated with atenolol. Once again, in this study, pressure gradient was only evaluated right after surgery and not followed long term. Re-stenosis could have affected survival time; it was not stated if pressure gradient reduction was maintained over the long term. The authors of this study stated that long-term atenolol therapy was recommended, but they did not actually state which dogs received it. Again, concurrent treatment with atenolol could have an effect on survival. The author of these proceedings notes uses high atenolol doses (titrated to achieve heart rates of 80-100 bpm at rest) for both PS and SAS and has seen many dogs with a dramatic response/reduction in gradient for both SAS and PS.

Recently, our group at the University of Florida has been investigating the use of cutting balloons for dogs with severe SAS. The cutting balloon has the ability to score the vessel longitudinally thereby, allowing a second, high pressure balloon to stretch and dilate the vessel more effectively without causing vessel injury. High pressure balloon valvuloplasty has been shown to improve the success rate of vessel dilation in humans. Since, the fibrotic ridge or ring in SAS has the potential for re-stenosis, it is likely that SAS lesions will not be ameliorated by simple stretching as in the low pressure balloon valvuloplasty techniques.

The procedure is performed from a vascular cut down over the right carotid artery for placement of introducers, followed by catheters, guide wires and dilation balloons. The diameter of the cutting balloon is selected to be equal to the subvalvular width (approximately 1/3 the width of the aortic annulus in dogs with severe SAS in order to avoid damage to this region). Following inflation, the cutting balloon is removed and the high pressure balloon (approximately 80% of the aortic annulus) is placed across the subvalvular region. Following reduction of the subvalvular waste and pressure decline, the balloon catheter and guide wire are removed and the artery ligated. Dogs are typically started on atenolol therapy prior to the procedure and maintained on a 1.5-2 mg/kg BID dose after the procedure. The most common post-op complications seen are transient ventricular arrhythmias (VPCs and ventricular tachycardia). Many dogs develop post-op arrhythmias and are treated with sotalol therapy if needed based on the rate and complexity of the arrhythmia. Most are able to be successfully weaned off the sotalol and maintained on just atenolol therapy. We have had one dog, with severe SAS and a tunnel lesion, suffer a mitral valve tear following cutting balloon and high pressure balloon valvuloplasty. This dog developed acute fulminant pulmonary oedema and died within four hours of the procedure. Necropsy and histopathology showed a surgical cut in the anterior mitral valve leaflet and subaortic region.

This combination of cutting balloon valvuloplasty and high pressure balloon valvuloplasty has proven to be effective in reducing the systolic pressure gradient and maintaining it over a period of over two years in some dogs. Thus far we have performed this procedure in 28 dogs with severe SAS. The dogs in the study were enrolled at different time points, so follow-up evaluations of pressure gradient for dogs enrolled later in the study still needs to be done in the future. We also have not been able to perform evaluation of survival statistics compared to previous studies or compared to a population of dogs treated with atenolol alone as many of the dogs are still alive. This will be an important part
of determining whether performing this procedure actually translates to a survival benefit for dogs. We have also recently looked at the Aortoseptal angle (AoSA) and how this relates to the development and response to balloon valvuloplasty and have found that dogs with AoSA > 160° on right-sided parasternal long-axis view echocardiograms responded better to balloon valvuloplasty than dogs with AoSA < 160°. This suggests that AoSA is associated with long-term outcomes of CB/HPBV, and measurement of the AoSA could help in the evaluation of dogs with SAS that are candidates for CB/HPBV.
Interactive panel discussion: Approach to the treatment of heart disease

Chair: Malcolm Cobb
Panel: Amara Estrada, Sonya Gordon, Virginia Luis Fuentes, Roberto Santilli

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