



Proceedings of the Spring Meeting of the Veterinary Cardiovascular Society

Wednesday 1st April 2020

Digital Web-based Conference hosted by



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To reflect the lack of face-to-face contact with delegates, sponsors will also be supporting some individual lectures but please note the **content of lectures and abstracts provided remains the property and work of the authors themselves**, without sponsor involvement unless otherwise stated in the proceedings.

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Victoria Greet BVM&S PGDip VCP MRCVS

Vicki graduated from the University of Edinburgh in 2014. She then gained a place on the competitive PDSA graduate programme, remaining with the PDSA for an additional year as a full-time veterinary surgeon. In 2016, she was appointed as an intern at the Royal Veterinary College on their rotating small animal programme and completed her postgraduate diploma in veterinary clinical practice. She then moved to Southern Counties Veterinary Specialists to undertake a cardiology-specific internship in 2017 and started her ECVIM residency in cardiology in 2018

Pedro Oliveira DVM DipECVIM-CA (Cardiology) MRCVS

Pedro qualified from Porto University (Portugal) in 2005 and obtained the ECVIM-CA cardiology diploma in 2012. He worked in a number of European referral centres before joining Davies Veterinary Specialists (United Kingdom) in October 2012 where he heads the cardiology service. He has a particular interest in interventional cardiology and the study of arrhythmias. In 2015, he established an electrophysiology laboratory for the diagnosis and treatment of arrhythmias at Davies Veterinary Specialists. He co-authored a textbook that was recently released on canine and feline electrocardiography published by Wiley (*Guide to canine and feline electrocardiography*).

Katarzyna Smiejan DVM MRCVS AFHEA

Kat Smiejan graduated in 2014 from the University of Warmia and Masuria in Poland, where she developed her interest in Veterinary Cardiology. She spent her first years in first opinion small animal practice in Cornwall, before starting her Rotating Internship at the Royal (Dick) School of Veterinary Science, University of Edinburgh, in 2019. Kat is currently working towards her certificate in Advanced Veterinary Practice (Veterinary Cardiology). She is involved in multiple research projects, including the HR analysis and monitoring in canine patients, as well as, new techniques and multimodal approach to teaching cardiac auscultation.

Professor N. Sydney Moise DVM MS DipACVIM DipACVIM-CA

Dr. Moise is involved in teaching, clinical practice, and research. Her research has centered on arrhythmias in dogs. Currently, her research is directed toward the normal and abnormal patterning of cardiac rhythms during long-term Holter analysis. She also has orchestrated collaborative studies to understand the mechanisms of degeneration of the mitral valve in dogs. She was awarded the AVMA research award for arrhythmia studies in German shepherds and the Bourgelat Award for international contribution to the clinical practice of veterinary medicine. She was recently awarded the inaugural endowed C.V. Starr Professorship in Cardiology by Cornell University. Dr. Moise was Editor-in Chief (5 years) and Associate Editor (2 years) of the international *Journal of Veterinary Cardiology*. Previously,

she co-edited and authored with Dr. Philip Fox and David Sisson the textbook *Canine and Feline Cardiology*. In 2018 the textbook *Electrocardiography of the Dog and Cat* authored by Drs. Roberto Santilli, N. Sydney Moise, Romain Pariaut and Manuela Perego. She is currently working on her book (Moise NS and Flanders WH) *Heartbeats...Over Time (Long-term Electrocardiographic Monitoring in the Dog and Cat)*. She is extensively involved in the international aspects of academic veterinary cardiology including speaking and training.

Gwilym Morris BA BmBCh MA PhD Consultant Cardiologist Central Manchester University Hospitals NHS Foundation Trust

Gwilym Morris is a Consultant Cardiologist and Cardiac Electrophysiologist specialising in the treatment of heart rhythm disorders, especially sinus node disease, arrhythmias in athletes, and atrial fibrillation. He is a British Heart Foundation Intermediate Clinical Research Fellow and leads a research group at the University of Manchester investigating how electrical remodelling of the sinoatrial node complex may contribute to complex atrial arrhythmias including atrial fibrillation.

He trained in medicine at the University of Oxford, after completing an intercalated degree in Physiology he was awarded a Wellcome Trust scholarship to fund a period of research before commencement of undergraduate clinical training. His postgraduate Medical and Cardiology training was undertaken in Manchester. He advanced his skills in cardiac ablation and pacing through a clinical fellowship in Pacing and Electrophysiology at the world-leading cardiology department of the Royal Melbourne Hospital led by Professor Jonathan Kalman. During this time he received intensive training in advanced arrhythmia management and ablation, and performed research into mechanisms of atrial fibrillation. He trains doctors in the UK and internationally on cardiac ablation techniques.

Victoria Ironside VetMB MA CertVC MRCVS

Vicky qualified from Cambridge in 1989 and worked for 8 years in small animal general practice before passing the RCVS Certificate of Veterinary Cardiology in 1997. After a further 2 years in general practice she began working with different practices seeing mostly cardiology and internal medicine cases. This work grew and established over the years to become a regular cardiology clinic service at around 8 different practices in Sheffield, Derbyshire and more recently Bollington.

Francesca C F Worsman BVM&S MRCVS

Francesca graduated as a veterinary surgeon from the Royal (Dick) School of Veterinary Studies, University of Edinburgh in 2011. Initially she worked in mixed practice for 2 years in the North East of Scotland. Following this she completed an equine hospital rotating internship at the University of Liverpool. She then went on to work at a racing practice in New

Zealand. After this she completed a busy internal medicine fellowship at Hagyard Equine Medical Institute, Lexington, Kentucky in 2016. Francesca is now in her final year of an internal medicine residency back at the University of Edinburgh and is doing a Masters by Research as part of her residency, entitled 'Real-time three-dimensional echocardiography of the equine left atrium'. She is interested in all aspects of equine internal medicine, especially cardiology, neonatal and critical care, ophthalmology and gastroenterology. She is a member of the Association of Racecourse Veterinary Surgeons.

Jonathan Elliott MA VetMB PhD FHEA CertSAC DipECVPT MRCVS

Jonathan Elliott is currently Professor in Veterinary Clinical Pharmacology and Vice Principal for Research and Innovation at the Royal Veterinary College. A Cambridge Veterinary Graduate, his PhD was in vascular biology and post-graduate clinical training was undertaken at the University of Pennsylvania. He joined the RVC in 1990 as a lecturer in Veterinary Pharmacology and developed research interests in feline kidney disease and hypertension and equine laminitis. His research has resulted in a number of awards recognising the impact of his work on clinical practice, including the Pfizer Academic Award (1998), BSAVA Amoroso Award (2001), PetPlan Charitable Trust Award (2005), ESVNU Scientific Award (2007) and BSAVA Woodrow Award (2019). He is a board member of the International Renal Interest Society and is president of the European College of Veterinary Pharmacology and Toxicology (2018-2021). He has published more than 200 international peer reviewed original papers and reviews and supervised 30 PhD students to completion. He is editor of a number of text books, most recently (2020) Hypertension in the dog and cat published by Springer Nature Switzerland AG (ISBN 978-3-030-33019-4).

H Edward Durham Jr. CVT RVT LATG VTS

H. Edward Durham Jr is the Senior Cardiology Veterinary Technician at Southwest Florida Veterinary Specialists. He is a charter member of the Academy of Internal Medicine for Veterinary Technicians (AIMVT) and served on their executive board representing cardiology for 12 years. Previously, he spent 18 years at the University of Missouri in the cardiology service, and 3 and a half years at Ross University School of Veterinary Medicine in the anesthesia service. He is an international speaker on veterinary cardiology. He has a passion for teaching, especially veterinary technicians. He is the editor of the textbook Cardiology for Veterinary Technicians and Nurses published by Wiley-Blackwell and over a dozen peer-reviewed articles. He is an international speaker and educator on the subject of cardiology to veterinary technicians and veterinary students.

Emma Baggus RVN DipAVN

Emma started in primary practice and qualified as a RVN from Abbeydale Vetlink College in 2011. She started as a referral nurse at the Willows Veterinary and Referral centre in 2012

and joined the Cardiology department in October 2015. Since starting at the Willows, Emma has undertaken her Diploma in Advanced Veterinary Nursing and qualified from Harper Adam's university in 2017 and is currently studying for a VTS in cardiology and hopes to qualify in summer 2021. When Emma is not at work, she has a 3 year old daughter who keeps her on her toes, also Emma has a keen passion for dance and particularly enjoys teaching Ballet.

Sara-Ann Dickson RVN

Sara-Ann obtained her degree in veterinary nursing in 2005 from Edinburgh's Napier University. She spent most of her practical placements training at the R(D)SVS and enjoyed it so much that after some time spent in a general practice, she re-joined the nursing team at the R(D)SVS.

Sara-Ann has worked in a variety of roles throughout her career so far and has developed a love for all things cardiology. At the end of 2014 she was successful in becoming the first cardiology nurse at the R(D)SVS and is enjoying the challenge of seeing all the specialised cases. She really likes the interventional surgeries and is working towards her VTS in cardiology.

Charlotte Pace BA (Hons) RVN VTS (Cardiology) PG Cert (Vet Ed) FHEA

Charlotte Pace qualified as a veterinary nurse in 2003, whilst working in practice in London. In the same year she moved to the Royal Veterinary College to work as a medicine nurse. In 2006 she became the dedicated cardiology nurse for the Queen Mother Hospital for Animals. In 2010 she passed American veterinary technician exams and became the first nurse to hold the Cardiology qualification outside the US. Since 2012, Charlotte has taught veterinary nurses on both degree and diploma programmes, and also works in practice. She continues to write and lecture on veterinary cardiology.

Lauren Osborne RVN FdSC

Lauren graduated from the Royal Veterinary College in 2018 with a FdSc in Veterinary Nursing. In 2018 she joined Southfields Veterinary Specialists, initially as a multi-disciplinary nurse, before quickly realising her commitment to cardiology. She is now the dedicated cardiology nurse at Southfields. She has a particular interest in congenital heart diseases and interventional cardiology.

Description of the clinical features and outcome for dogs and cats with right-to-left shunting patent ductus arteriosus (r-PDA)



Victoria Greet MRCVS

Southern Counties Vet Specialists, Hampshire, U.K.

Introduction: Left-to-right shunting patent ductus arteriosus (PDA) is one of the most common congenital cardiac diseases in dogs, occurring less frequently in cats. If pulmonary vascular resistance is increased, blood flow through the PDA can reverse, resulting in a bidirectional or right-to-left shunting PDA (r-PDA). Although this complication is widely recognised, outcome studies regarding the clinical progression of animals with bidirectional or r-PDA are lacking. The aim of this study was to describe the signalment, presenting signs, echocardiographic features and survival time of dogs and cats with r-PDAs.

Methods: Retrospective multi-centre study. The medical records and echocardiographic findings were reviewed from animals diagnosed with a bidirectional or r-PDA. In cases where the PDA could be visualised from stored echocardiographic images, ductal morphology was characterised according to the Miller classification system. Kaplan-Meier survival curves were generated to assess the impact of ductal morphology, spectral Doppler flow profile, packed cell volume, sildenafil dose, severity of pulmonary hypertension, patient general anaesthesia (+/-surgery) and the presence of right-sided congestive heart failure (R-CHF) on all-cause mortality. Differences in survival between groups was compared using Mantel-Cox log rank comparison of Kaplan-Meier survival curves.

Results: Forty-six client-owned animals were included, comprising 43 dogs and 3 cats with r-PDA. Fifteen dogs and 3 cats had bidirectional flow through the ductus. The most common presenting signs in dogs were hind limb collapse (n=16) and exercise intolerance (n=9). Of the three cats, one each presented with tachypnoea and abdominal distension, whilst one cat was free of clinical signs. Three dogs (and the previously mentioned cat) had abdominal distension secondary to R-CHF. Ductal morphology could be assessed from 29 echocardiographic examinations. Type II and type III ductal morphologies were present in 12 cases (11 dogs and 1 cat), and 17 cases (15 dogs and 2 cats), respectively. Survival information was available for 38

animals: 12 were alive at the time of data collection, while 26 had died. The remaining 8 animals were lost to follow-up. Survival analyses were performed separately on data available for dogs. Median survival time was 626 days (range 1 - 3628 days). Ductal morphology, sildenafil dose, packed cell volume at presentation, general anaesthesia +/- surgery and severity of pulmonary hypertension did not influence survival. Dogs with R-CHF at presentation had a worse outcome (median survival time 58 days vs 1839 days, $p=0.028$).

Conclusions: Dogs and cats with bidirectional or r-PDA have a variable clinical presentation and prognosis. Dogs with R-CHF at presentation have a worse overall prognosis.



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EP studies and radiocatheter ablation in dogs – our experience so far

Pedro Oliveira MRCVS

Davies Veterinary Specialists, Hitchin, UK

Electrophysiology studies and radiocatheter ablation procedures (EP/RFA) allow the definitive diagnosis and treatment of cardiac arrhythmias via cardiac catheterization. In humans, these procedures have been available for decades and are now considered the first line of treatment for many cardiac arrhythmias - supraventricular or ventricular in origin. This is in contrast with veterinary medicine where only a small number of referral centres worldwide currently perform these procedures on a regular basis. In dogs, it is mostly used to treat supraventricular tachyarrhythmias such as atrioventricular reciprocating tachycardia (tachyarrhythmias involving an accessory pathway - AP), atrial flutter or ectopic atrial tachycardia.

Atrioventricular reciprocating tachycardia (AVRT).

In humans, RFA has become the first-line treatment for AVRT in humans, even in asymptomatic patients. It provides definitive resolution with a high success rate (89-97%) and very low complication rate (<1%).^{1,2} In dogs, a recent large retrospective study has reported a success rate of 98.8% with recurrence and complications in 0.03% and 0.06% of cases respectively.³ Another similar study reported an acute success rate of 100%, with recurrence in 7.7% of cases within 18 months, and a major complication in 2.6%.⁴ At Davies Veterinary Specialists we have treated 21 dogs with AVRT with a total of 25 accessory pathways. Procedural success was achieved in all but one case. In this case, 2 APs had been identified. One was successfully ablated but the other was too close to the atrioventricular node. During ablation, intermittent atrioventricular node block was observed and a decision was made to not attempt further RFA. Fortunately, this appeared to be enough to cause significant clinical improvement for this patient. He has been well since on anti-arrhythmic medication. One case of recurrence was observed in a dog with a left-sided pathway. All the other dogs treated have been well after the procedure and without anti-arrhythmic medication as far as we know, except for the very first case we treated. This was a young Rottweiler that had developed significant structural cardiac changes and atrial fibrillation by the time of RFA. Even though clinically he was better for a while after RFA, he did not show signs of recovery from the structural point of view and eventually developed signs of congestive heart failure that lead to euthanasia. It was too late for this dog and unfortunately this is a problem we have

been encountering often. In our experience, EP/RFA is often only considered after drug therapy fails and persistent tachycardia has resulted in significant structural damage. It is worth highlighting that this is a curative procedure and that even dogs with significant structural damage can recover fully. A recent report showed that 46.1% of cases undergoing EP/RFA for ablation of APs had developed tachycardia induced cardiomyopathy (TICM) and showed either complete resolution or marked improvement after only 1 month.³ Hopefully in the future any dog with documented tachycardia mediated by an AP will undergo EP/RFA as soon as possible, even if asymptomatic, as has become common practice in humans.

Atrial tachycardia

In humans, RFA is recommended for patients with symptomatic FAT as an alternative to medical treatment with a success rate of 69-100% and recurrence in 4-27% of cases.^{1,5} However, in experienced centres success rates consistently above 90-95% are observed with low complication rates (<2%).¹ In dogs, it is normally only considered in symptomatic patients after drug therapy fails. A success rate of 79% with recurrence in 2% of cases has been reported.⁶ We have performed EP in this setting in 5 dogs: two presented with multifocal atrial tachycardia (MAT) and the other three with junctional tachycardia (JT). RFA was performed successfully both dogs with MAT and in one of the dogs with JT without atrioventricular block. In another dog with JT, partial ablation of the atrioventricular node was performed and successfully controlled the heart rate without complete heart block and need for a pacemaker. RFA was not attempted in the remaining dog with JT. Recurrence was not observed and only a minor complication occurred in one dog – the grounding skin patch detached slightly causing a skin burn during RFA.

Atrial flutter

Ablation of atrial flutter is common in humans. In typical flutter RFA is performed at the cavoatrial isthmus resulting in success rates of 90-97% with large (8-10 mm) or irrigated tip catheters, and recurrence rates of approximately 7-10.6%.⁷⁻⁹ RFA is considered useful in patients that are either symptomatic or refractory to pharmacological rate control.¹⁰ In veterinary medicine, a success rate of 100% with recurrence in up to 15% of cases has been reported.⁶ We have only performed RFA in one case with suspected typical atrial flutter. The procedure was successful, without complications, and the dog has been well since.

Atypical flutter cases provide a bigger challenge as a well-known and well-defined re-entrant circuit is not known in advance. In these cases, electro-anatomical mapping with a 3D navigation system is helpful as it allows visualization of the re-entrant circuit on a 3D reconstruction of the atrial anatomy. With this type of atrial flutter, RFA is considered useful in patients that are recurrently symptomatic and in which at least 1 antiarrhythmic agent failed to provide appropriate rate control.¹⁰ In dogs, EP and RFA of non-isthmus dependent atrial flutter has been reported in a case series of 5 dogs.¹¹ In all dogs, the re-entrant circuit was located on the right atrium and ablation was performed successfully in all cases. One dog showed signs of recurrence after 15 days. Another dog presented with atrial fibrillation 2 months after RFA. The remaining 3 dogs were still free of arrhythmia after 18 months.

Conclusion

EP studies and RFA provide a safe and effective option for the definitive diagnosis and treatment of cardiac arrhythmias. Hopefully these procedures will become increasingly available in veterinary medicine and timely referral will contribute to a better outcome for dogs with supraventricular tachyarrhythmias.

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Use of low-cost heart rate monitors in canine patients

Katarzyna Smiejan MRCVS

Royal (Dick) School of Veterinary Studies, Edinburgh, UK

Introduction

Successful management of many cardiac diseases frequently requires monitoring of mean heart rate (HR) over prolonged periods of time. Repeated ambulatory electrocardiographic analysis (Holter) is variably tolerated by dogs and their owners, and can be financially prohibitive. In the human market, new HR monitors that use existing digital platforms, and record HR during exercise, are available. They are both, affordable and user-friendly. Until now, no information has been available regarding their use in canine patients. Smartphone-based low-cost HR monitors, used by owners at home, could improve the tolerance and affordability of HR analysis in canine patients with cardiac conditions such as atrial fibrillation, thus aiding diagnosis and management strategies.

Hypothesis:

We hypothesised that low-cost smartphone HR monitors can accurately record the HR in ambulatory canine patients in both, health and disease.

To investigate this hypothesis, our first objective was to validate the use of smartphone HR monitors, by comparing the HR recorded by two systems (Polar® and MyZone MZ-3), with femoral pulse rates simultaneously obtained from healthy ambulatory dogs. For this study, femoral pulse was used as a surrogate of HR. The influence of cardiac and non-cardiac disease was also determined. Subsequently, our second and major objective will be to compare mean HR, derived from multiple HR monitor recordings, with mean HR derived from Holter recordings over the same 24-hour period.

Methods:

Data obtained over one minute from Polar® H10 and MyZone MZ-3 devices were uploaded onto three mobile applications: Polar Beat, SelfLoops HRV, and My-Zone. Following assessment of normality (Anderson-Darling), correlations between HR and simultaneous femoral pulse rate were made with Pearson's or Spearman's test. Limits of agreement (LOA) and bias were assessed by Bland-Altman plots. Multiple comparison tests assessed the influence of body weight (BW), body condition score (BCS) and disease status.

Results:

The correlation between the HR obtained by pulse rate measurement and the different devices was excellent for the Polar Beat ($R=0.944$, $p<0.001$), Self-Loops ($R=0.908$, $p<0.001$) and My- Zone ($R=0.911$, $p<0.001$). Bland-Altman plots demonstrated lack of bias, with lower LOA for Polar Beat and My-Zone than for Self-Loops. There was no influence of resting heart rate, body weight, BCS or disease status.

Conclusions and Clinical Significance:

Recording HR with non-invasive, low-cost HR monitors, such as Polar® H10 and MyZone MZ-3, appears feasible and accurate over one-minute periods in ambulatory healthy dogs, and canine patients with cardiac disease. Further evaluation in a larger cohort of subjects, and in patients with different disease status, is necessary to complete validation. The clinical usefulness in assessing HR in canine patients with complex tachyarrhythmias over more prolonged periods remains unknown.

Conflict of interest and funding:

The authors declare no potential conflicts of interest and no financial support with respect to the research, authorship, and/or publication of this abstract.

Sinus node dysfunction: the details of mechanisms, electrocardiography, Holter monitoring and pacing

Professor N Sydney Moise
Cornell University, Ithaca, USA



TODAY. NOT ONE DAY.

The following notes are provided to accompany the Veterinary Cardiovascular Society presentation on April 1, 2020

N. Sydney Moise, DVM, MS

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The following manuscript provides a background in the dog for the heartbeat patterning with normal sinus node function over 24 hours:

<https://www.frontiersin.org/articles/10.3389/fphys.2019.01548/full>

Sinus node dysfunction is a spectrum of arrhythmias that include sinus bradycardia, sinus pauses, inappropriate sinus tachycardia and atrial arrhythmias. Sick sinus syndrome is diagnosed when clinical signs (symptoms in humans) accompany sinus node dysfunction.

The following characterize the patients with sinus node dysfunction.

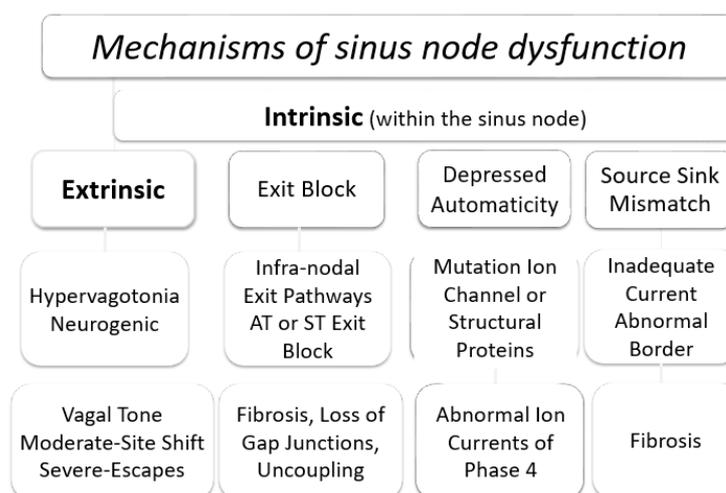
1. Small breeds (West Highland White Terrier, Miniature Schnauzer, Cocker Spaniel), Boxer
2. Often female middle age to older
3. Bradycardia, lethargy, near syncope, syncope

The following characterize the electrocardiographic findings with sinus node dysfunction.

1. Bradycardia only
2. Tachy-brady syndrome
3. Brady-tachy syndrome
4. T-waves enlarged or abnormal T-waves
5. QT interval prolonged
6. AV nodal conduction normal or abnormal
7. It is estimated that at least 50% of dogs with sinus node dysfunction have accompanying atrial arrhythmias in addition to sinus bradycardia

A critical observation with regards to the atrial rhythms in dogs with sinus node dysfunction concerns the origin of P waves associated with different supraventricular rhythms. The following would be under consideration.

1. Changing P wave morphology with rates that are similar to a normal sinus node discharge
2. Often retrograde P waves following junctional or ventricular escape beats. It is important to closely examine the ST segment of escape beats. Commonly when a pacemaker is placed sinus node dysfunction dogs have retrograde P waves that can result in echo beats because of normal AV nodal conduction. Retrograde AV nodal conduction (VA conduction) is common in dogs with sinus node dysfunction. Escape beats may originate from the sinus node, atria, junctional region or ventricular tissue (Purkinje fibers). Escape beats are often considered late or at rates less than the usual spontaneous depolarization of the subsidiary pacemakers. Another observation is that retrograde conduction through the AV node is not seen with sinus or atrial complexes; however, they are often seen with junctional or ventricular escape beats.
3. Atrial rhythms often have P wave morphology that is similar to that of sinus beats. It is hypothesized that these beats are originating from the region of the crista terminalis or ectopic beats from in the sinus node complex. Dogs with sinus node dysfunction may in fact have micro-sinus node reentry or macro-sinus node reentry. Micro – sinus node reentry is hypothesized as reentry within the sinus node complex where is macro – sinus node reentry involves either part of the atrial tissue and sinus node complex or involves the sinoatrial pathways connecting the sinus node proper to the atrial myocardium.
4. Atrial tachycardias with varying morphology can also be identified indicating multiple sites of origin or conduction.



Dogs with sinus node dysfunction may have atrial tachycardias that develops without warning and then results in overdrive suppression of the normal functioning sinus node cells or exit pathways. This results in prolonged pauses in atrial depolarization activity. Therefore, the sinus node is being overdriven and so are subsidiary atrial and junctional escape pacemakers. Often a lack of ventricular escape beats is also evident or the rate of spontaneous discharge is slower than what would be expected. These findings suggest that not only is the sinus node

spontaneous discharge abnormal in dogs with sick sinus syndrome, but so are the subsidiary pacemakers.

It is important to differentiate sinus node dysfunction from other disorders that are affecting the sinus node. These may include drugs or electrolyte imbalances. Hyperkalemia can cause a sinus bradycardia. The very flat P waves of hyperkalemia may be misdiagnosed as 'no' P waves. In such cases a misdiagnosis of sinus node dysfunction can be made. Closely observing the T wave (tented or pointed T wave) and the condition of the patient (urinary obstruction, Addisonian crisis) will give more indication that a secondary problem of atrial depolarization due to hyperkalemia exists. Another differential is the uncommon inherited condition of persistent atrial standstill.

Other electrocardiographic findings in dogs with sick sinus syndrome include varied P wave morphology, abnormal T waves, or prolonged QT intervals. In boxers that have sinus node dysfunction in addition to atrial arrhythmias ventricular arrhythmias may also be present because of arrhythmogenic right ventricular cardiomyopathy.

The summary below illustrates the complexity of the mechanisms behind sinus node dysfunction. It is important to realize that each of these is not mutually exclusive. That is, a dog can have primary sinus node dysfunction but also have it influenced by hypervagotonia.

Can a dog with sinus node dysfunction or sick sinus syndrome still have a response to atropine or exercise? Yes. But, it is important to remember that there is variation and that we need additional studies with regards to the chronotropic response of the sinus node in the dog. Currently, the definitions of a positive response are not based on comparisons to normal dogs in the clinical setting concerning age and condition.

You may read or hear that if a dog responds to atropine it does not have sinus node dysfunction or sick sinus syndrome. This is not true. The vast majority of dogs will have some response to atropine. We do not know what a normal response to a vagolytic drug in dogs of different ages should be. Additionally, a variability in the response will occur depending on the underlying mechanism for the sinus node dysfunction. Moreover, having a response to atropine that seems to be appropriate can be a good sign when judgments must be made about anesthesia and treatment decisions. If it is not an emergency, a good point of advice is the following. If a dog is suspected of having sinus node dysfunction and must undergo a procedure that requires anesthesia or sedation during which it is possible that a vagolytic drug will be required, a 24 hour Holter can be of value.

Most often sinus node dysfunction dogs are identified because of a bradycardia. If the dog has sinus node dysfunction and only has a bradycardia than the use of atropine is likely not a high risk. However, if the dog has tachy-brady or brady-tachy syndrome then the use of atropine may induce an atrial or sinus tachycardia that overdrive suppresses subsidiary pacemakers. This rapid heart rate in dogs with sinus node dysfunction may stop suddenly resulting in prolonged periods of asystole. By performing a 24 hour Holter monitor this permits the identification of any abnormal atrial tachycardias or sinus node complex tachycardias. If either of the previous are identified, this alerts the veterinarian that the use of atropine might be high risk. In these cases, transcutaneous pacing really should be available.

Atropine may be administered subcutaneously, intramuscularly or intravenously. Doses may range from 0.02 mg/kg to 0.04 mg/kg. Often almost all dogs (perhaps more often with intravenously administered atropine) will develop for a short time 2nd degree heart block. This is likely because the sinus node chronotropic effect occurs before the AV nodal dromotropic effect. Consequently, because of the decremental conducting properties of the AV node when the sinus rate increases the AV node cannot keep up with the rate and prolongation of the PR interval and complete block in the AV node can occur. It is best advised to use a continuously recording electronic ECG during this test. This will provide the most information about the dog's response.

The benefit of performing atropine response test is that it may help differentiate between a dog with hypervagotonia versus one with sinus node dysfunction. This differentiation is possible; however, it must be remembered that we do not have normal response characteristics for dogs of different ages in dogs may have a combination of problems. A positive response to atropine as judged by the degree that the heart rate increases supports the likelihood that if a vagolytic is required during anesthesia, it will work to increase the sinus node rate without an excessive supraventricular tachycardia causing overdrive suppression of the necessary subsidiary pacemakers. It may also indicate the likelihood that the dog will respond at least for some time to medical treatment so that if necessary a pacemaker implantation can be delayed.

Can dogs with sinus node dysfunction also have atrioventricular nodal block? Yes, they can and this is known as *binodal dysfunction*. This may be manifested as a prolonged PR interval or actual 2nd or 3rd degree heart block. The AV nodal conduction can be influenced by autonomic tone. It is very important not to confuse atrioventricular nodal block with physiologic AV conduction block due to a high rate from a supraventricular tachycardia. Approximately 50% of dogs with sinus node dysfunction have atrial tachycardias or inappropriate sinus tachycardia. Atrial tachycardias are illustrated with P' and the intervals as P'-P'.

Is a Holter recording necessary for the diagnosis of sinus node dysfunction? No, but it can help to understand the complexities that may need specific management during pacemaker implantation. It also may help determine if the patient has sinus node dysfunction as a primary problem or secondary to excessive vagal tone. Specific information from Holter recordings include the rate of bradycardia during sleep-wake cycles, the duration of pauses, the number of pauses greater than 2 seconds, and whether the beat patterning is consistent with sinus arrhythmia or sinus node dysfunction. Sinus node dysfunction dogs may or may not have a pattern identified on the Poincaré plots and tachograms RR interval plots that are characteristic of exit block.

What are the components that determine the sinus rate? It is important to know that there is an intrinsic/inherent heart rate of each species. This rate is the one, which is the spontaneous depolarization rate of sinus node cells and exit from the sinus node complex without autonomic influence. In the dog, this rate is usually between 90 and 100 bpm. This corresponds to 666 – 600 ms intervals. When intrinsic/inherent heart rate is present the PP interval is constant. In the dog parasympathetic and sympathetic influence on sinus rate result in dramatic wide ranged heart rates (30 – 300 bpm). Although the sinus rate can go as fast as 300 bpm in a young dog, this is not a sustainable rate. Such rapid rates last only a few seconds. For the most part, a sinus rate is usually below 250 bpm in the dog. During sleep, dogs can have a heart rate that goes down to 30 bpm. But, during a 24 hour period the majority of dogs

have a heart rate less than 50 bpm for less than 30 minutes. Some breeds (brachycephalic breeds) may have slower heart rates for a longer period of time.

Importantly, determining if a rate is too slow or too fast in the dog must take into account the physiologic response. How is the ECG recorded? Is it at home with a 24-hour Holter recording or is it an ECG in the hospital? How is heart rate judged? Is it judged by the average heart rate, the minimum or maximum heart rate, the amount of time above or below certain rates or the number and duration of sinus pauses. The influence of the circadian and sleep cycle, breathing frequency and depth, and central effects must always be considered.

The following data are provided as suggestions for a normal dog (median, range). This is based on 24 hour Holter data from 130 dogs of different breeds. For the most part, breed does not determine the rate in the dog; however, there are likely exceptions to this general statement. Influences may be whether or not the dog is a brachycephalic breed or of nervous behavior. And although there do not seem to be breed differences with regards to the average heart rate, how the heart rate distribution occurs over 24 hours may be different and demands further research.

- AVERAGE HEART RATE: 80 (65 – 120) bpm
- MINIMUM HEART RATE: 40 (30 – 72) bpm
- TIME HEART RATE < 50 BPM: 10 (0 – 350) min
- NUMBER OF PAUSES > 2 seconds: #75 (0 -1500)
- LONGEST PAUSE: 3.0 (1.5 – 5.5) seconds
- NUMBER OF PAUSES > 4 seconds in 24 HOURS: < 3 (majority of dogs have < 3 pauses > 3 seconds)

When trying to differentiate a normal dog from a dog with sinus node dysfunction, evaluation of the average heart rate during a 24-hour Holter can be of value. The median heart rate of normal old small breed dogs (n = 25) was 85 bpm (66 – 120 bpm range) and for dogs with sinus node dysfunction, which were also small old dogs, the median heart rate was approximately 65 bpm (49 – 116 bpm range). Despite the differences there is a lot of overlap in individual dogs are evaluated.

In the evaluation of sinus pauses in the dog, the older small breed dog has a median of approximately 30 pauses that were >2s in duration. Dogs with sinus node dysfunction/sick sinus syndrome had greater than 7000 pauses > 2s.

Therefore, a general suggestion to define sinus node dysfunction in the dog would include a collective assessment of several factors from 24 hour Holter monitoring. The following are suggested based on evaluation of 50 dogs with sinus node dysfunction. Not every dog with sinus node dysfunction will have each of the following characteristics; however, they will likely have 4 of the 6.

- AVERAGE HEART RATE: < 60 bpm
- MINIMUM HEART RATE: < 30 bpm
- TIME HEART RATE < 50 BPM: > 350 min
- NUMBER OF PAUSES > 2 s: > 1500 #
- LONGEST PAUSE: > 5.5 s
- NUMBER OF PAUSES > 4 s 24 HOURS: > 3

The heart rate from the electrocardiographic evaluation as described above will differentiate sinus node dysfunction from other problems for the most part; however, other tools are important in securing the proper diagnosis. These other considerations include:

1. Chronotropic incompetence.
2. The presence of a normal nocturnal dip or one that is exaggerated.
3. The beat to beat patterning of normal sinus arrhythmia versus sinus arrhythmia with altered intervals associated with sinus node dysfunction. The latter are judged by 24-hour tachograms and Poincaré plots.

Chronotropic incompetence has been studied extensively in humans. Different patterns of the inability of the heart rate to accommodate need have been identified. In humans that have heart failure chronotropic incompetence is common. These include:

1. Submaximal peak heart rate during exercise (in humans it is the inability of the heart rate to reach 80% of the age predicted maximum heart rate)
2. Delay in achieving maximum heart rate
3. Heart rate instability during exercise
4. Inadequate heart rate recovery
5. Inadequate heart rate reserve (the latter is defined as the peak heart rate minus the resting heart rate).

A nocturnal dip in humans is a difference in the average heart rate of 10% or more during the sleep hours compared to the awake hours. The nocturnal dip in the dog has not been determined and must be matched with a diary due to the sleep patterns of the dog. Preliminary information indicates that the nocturnal dip may be larger in the dog than the human. Heart rates of 15 to 20% slower have been appreciated.

Patterning of the heart rhythm in the dog must be understood to identify abnormal sinus node function. The dog has a unique pattern of beating that we recognize as sinus arrhythmia; however, when the heart rate speeds and slows in the human it occurs along the line of identity when beat-to-beat plots (Poincaré plots, Lorenz plots) are constructed. The dog does not do this but instead has a bifurcation away from the line of identity when the heart rate approaches the inherent rate of approximately 90 to 100 bpm.

The patterns of long and short P-P/R-R intervals is different between normal dogs and dogs with sinus node dysfunction. Most characteristically, sinus node dysfunction dogs have longer long intervals with shorter short intervals giving a more clustered appearance.

When the time of day is plotted on the x-axis and the RR interval as a surrogate for the P-P interval is plotted on the y-axis, normal dogs have a different pattern than humans. They have a region where there is a paucity of beats (zone of avoidance). The lower range of RR intervals (faster heart rates) is at the inherent sinus node rate during sleep when very little sympathetic tone is present. At longer intervals a band of varying density is seen. In dogs with sinus node dysfunction they may have an exit block pattern or one that is varied from normal. The exit block pattern has bands on the tachogram that tend to be fairly even because of the ratio of block from the sinus node complex. This type of pattern also is seen in dogs that have atrioventricular conduction block through the AV node because the intervals from 24 hour Holter's are based on R-R intervals and for the evaluation sinus node dysfunction this is a surrogate for the PP intervals. Obviously therefore, it is important to ensure normal AV nodal conduction in the evaluation of sinus node function from Holter patterns.

Why does a dog have sinus arrhythmia? It is a balance of autonomic input between the parasympathetic and sympathetic systems. This balance can be assessed by heart rate

variability. It is an oversimplification to think that sinus arrhythmia is only related to inspiration and expiration. There are stretch receptors in the lungs and the thoracic wall. When stretched the pulmonary C- fibers are stimulated. This stretching inhibits the vagal activity and this inhibition can be strong. Efferent neurons from the respiratory and circulatory systems modulate cardiac vagal pre-ganglionic neurons. These are strongly inhibited during inspiration. The depth of inspiration is important in the degree of stimulation.

Baroreceptors influence sinus arrhythmia. These are high pressure receptors that when the pressure is increased a decrease in heart rate is stimulated. Venous return also changes heart rate. Changes in right atrial pressure during inspiration affect the Bainbridge reflex. These receptors are low pressure receptors. With increased venous return during inspiration (inhalation drops thoracic pressure and increases venous return) atrial receptors are stimulated and this stretch activates the medullary center to activate the sympathetic system which then causes an increased heart rate. Therefore, the Bainbridge reflex and baroreceptor reflex have opposite effects on heart rate. The former slows the rate and the latter increases rate. If drugs are given that increase blood pressure a reflex bradycardia will occur due to the baroreceptor reflex.

Deeper breathing results in a heart rate that follows respiration closer such that during inspiration rate increases and during expiration it slows.

Sinus arrhythmia in the dog can have a variety of patterns. The patterns developed as a result of the ratio of long intervals to short intervals. Specific ratios such as 1:1 (long-short), 1:3 (long – short – short – short) or 2:4 (long – long – short – short – short – short) are a few which can be identified. Mixtures of these ratios can occur.

How does a sinus arrhythmia change with aging? Aging likely effects the following:

1. Central respiratory center in the medulla,
2. Intrathoracic pressure and compliance of the thoracic wall
3. Compliance of the venous and arterial system
4. Ability of lungs to fill and activate stretch receptors
5. Integrity of reflexes including the Bainbridge and baroreceptor
6. Central brain effects of aging

All of the above must be considered in the evaluation of heart rate and pattern. Numerous alterations occur with aging that have the potential to change the input to the rhythm system.

Studies in human beings reveal that sinus arrhythmia decreases with age. Baroreceptor sensitivity decreases with age, but it is not the only factor. Mechanostructural and normal components are also altered. There is a decreasing effect of acetylcholine on the sinus node with age. Beta-adrenergic function declines with age. Right atrial muscarinic receptor density and function also decrease. These have all been shown to occur with aging in human beings and likely play a role in the way that the heart rate and rhythm is determined in the aging dog.

During aging specific genes are required to maintain sinus node function during stress; however, with age these ion channels and genes are repressed. *Na1.5*, *Cx43*, *Ca 1.2*, *RYR2*, *HCN1*, *HCN4*, *Popdc1* and *2* have been identified as less in the age. Mice deficient in the latter 2 genes have been shown to develop stress-induced bradycardia that is age-related.

Rethink the total evaluation of the electrocardiogram. It is not just about P-QRS-T and the single beat and what it is, it's about the rhythm of all the beats

Sinus node dysfunction can be caused by intrinsic (problem within the sinus node) or extrinsic (problem outside of the sinus node complex). Importantly, some dogs may have an intrinsic problem that is exacerbated by extrinsic factors. The extrinsic factors involved hypervagotonia (high vagal tone that is the consequence of some input). With an extrinsic problem there can be a shift in the impulse origin/exit from the sinus node or with extremely high vagal tone escape beats from the atrial muscle, junction or ventricular tissues can occur. Intrinsic problems of the sinus node may be the result of (1) exit block, (2) depressed automaticity resulting in slowed oscillator in sinus node or (3) source sink mismatch. Within the sinus node there are exit pathways. In the dog 2 to 4 of these pathways have been identified. The pathways are located at the cranial dorsal region of the sinus node, mid sinus node and caudal ventral region. Fibrosis, loss of gap junctions or uncoupling of cells can cause exit block. A change in the oscillatory speed of the pacemaker cells within the sinus node can occur because of mutations in ion channels or structural problems of the cells. Specific genetic mutations have not been identified in the dog, but they have been identified in human beings such as HCN4. The abnormal automaticity of the sinus node is due to then the abnormal ion current function during phase 4 of spontaneous depolarization. A source sink mismatch occurs when there is inadequate amount of current coming out of the sinus node through the exit path due to an abnormal border or junction with the atrial tissue. This is often due to fibrosis

The clinical question of course is when do we worry about a patient who has sinus node dysfunction? Just as with any disease there is variation in severity and this of course dictates the variation in treatment. For example, a dog is at greater risk demanding more aggressive therapy if clinical signs are present of syncope or unrecognized signs are accompanied by a heart rate of < 50 bpm or numerous pauses > 6 seconds without vagal tone. Less concern is warranted in elderly dogs with mild bradycardia or pauses that are more often associated with high vagal tone.

What are the treatment decisions and options for dogs with sinus node dysfunction or sick sinus syndrome? It is important to realize the general statements concerning the prognosis of the dog with sick sinus syndrome may not apply to an individual dog that you are examining. Adequate study not been done in the dog to assess conclusions regarding treatment with specific criteria. No treatment may be needed in dogs that have a normal but high vagal tone, geriatric dogs that do not have clinical signs, dogs that are near the end-of-life, or dogs with life-threatening coexisting disease. Some dogs that have sinus node dysfunction have primary extrinsic causes and in such situations, eliminating the cause of hypervagotonia is ideal. An example would be upper airway problems. Some underlying diseases do not have solutions such as some brain disorders that cause the elevation in vagal tone. A nonselective antagonist of the adenosine receptor, theophylline, may be a short-term option in some cases. Some dogs that respond more to atropine without tachycardias coexisting may find the best benefit from this treatment with theophylline. The dosage is 10 mg/kg twice daily.

Unfortunately, most of the time dogs with syncope caused by sinus node dysfunction require pacemaker implantation. Pacemaker implantation may involve only an atrial lead, dual lead in the atria and ventricles or only a ventricular lead. Decisions revolve around the size, age and availability of varying equipment.

Is important that when a dog is sedated or anesthetized more pacemaker implantation to be able to support that patient if complete sinus arrest or complete exit block from the sinus node occurs. Atropine may work, but it may not. The ideal situation is to have transcutaneous

or transvenous temporary pacing available. When using transcutaneous pacing it is vital to ensure that an impulse is resulting in a blood pressure. This can be done by careful arterial palpation or ideally from an intra-arterial line. Because the artifact of transcutaneous pacing is large, it can be confused with an actual ECG and this is why verification of the pulses is mandatory.

Sinus node dysfunction can at times be confused with normal or exaggerated cardiovascular reflexes.

The following are neurocardiogenic reflexes associated with hypervagotonia.

1. Bezold-Jarisch reflex
2. Vasovagal syncope
3. Situational disturbances
4. Coughing, vomiting, defecation, micturition, sleep apnea
5. Intracranial hypertension (Cushing's reflex)

Neurocardiogenic syncope may result in cardioinhibitory action (slow rate) or basal pressure action (low blood pressure).

A low heart rate can be associated with a low or a high blood pressure depending on the cardiovascular reflex.

Examples of cardiovascular reflexes causing syncope can be discerned from Holter monitoring or implanted REVEAL devices. This is an example of a dog suffering from an anxiety disorder that caused it to have syncopal episodes after excitement. Tachycardia preceded electrical asystole. Before asystole developed the heart rate gradually slowed indicating increasing vagal tone until the parasympathetic tone was extreme resulting in no sinus node rhythm or AV nodal conduction.

A broad grouping of reflexes that may cause syncope is known as vasovagal syncope. In this situation enhancement of the parasympathetic nervous system and withdrawal of the sympathetic nervous system occurs. The hemodynamic responses can be due to cardioinhibitory response (negative chronotropic effect) or vasodepressor response (drop in blood pressure), or a mixed response of cardioinhibitory and vasodepressor response. The following are some descriptions of those, which have been noted in the dog.

The following are situations that must be differentiated from sinus node dysfunction.

Accentuated antagonism and vasovagal syncope (neurocardiogenic syncope)

Numerous triggers can elicit vasovagal syncope. The following have been noted: micturition, defecation, severe coughing, standing up quickly, stress, pain, and trauma. All triggers involve the activation of the nucleus tractus solitarius of the brainstem which then activates the parasympathetic nervous system with concomitant withdrawal of the sympathetic nervous system. This then causes a cardioinhibitory response in which the heart rate slows and contractility is reduced. Also, the blood pressure drops because of the vasodepressor response causing vasodilation. Immediately before vasovagal syncope there is a marked reduction of muscle sympathetic activity in this is believed to be the cause of severe hypotension. In humans, patients with vasovagal syncope have been found to have an increased resting amount of muscle sympathetic activity but with a blunted activation during orthostatic stress.. The arterial baroreceptors are located in the carotid sinus and the aortic arch and the cardiopulmonary baroreceptors (low pressure receptors are located in the great veins, atria (Bainbridge reflex) and ventricles (Bezold-Jarisch reflex). These are the receptors

that are stretch activated ion channels, which influence sympathetic outflow. A dysfunctional reflex of the arterial system has been implicated in the pathophysiology of the loss of consciousness known as vasovagal syncope. Some believe that a failure of vasoconstriction of the venous system can also play a role in vasovagal syncope. Accentuated antagonism can occur in some instances of vasovagal syncope. During such an event, marked sympathetic stimulation occurs followed by a vagal stimulus. In such situations, the vagal response is exaggerated because of the preceding sympathetic stimulation. In dogs, such a vagal response may not only result in sinus arrest or AV nodal block, but also induce atrial fibrillation. The atrial fibrillation induced in such situations is usually temporary. High vagal tone causes atrial fibrillation because it shortens the action potential duration of the atrial myocardium, which increases the propensity for reentry, which is the underlying mechanism for atrial fibrillation.

A special type of vasovagal reflex that is complex: the interplay of the parasympathetic and sympathetic system is that a Valsalva maneuver. There are 4 stages to the Valsalva maneuver. In phase 1 the systemic blood pressure elevates with an increased intrathoracic pressure, but the heart rate does not change as the blood pressure rises. In phase 2 the high intrathoracic pressure during straining results in a decrease in venous return, which then causes a reduced stroke volume. The decrease in stroke volume continues with a decrease in pulse pressure and straining continues. Now, the heart rate increases and the blood pressure decreases. When the strain is released, phase 3 results because of a decreased intrathoracic pressure and the pulmonary blood flow returns to normal. During phase 4, the blood pressure will overshoot as the heart rate returns to normal.

Bezold-Jarisch Reflex

This may be a form of vasovagal reflex. This reflex, which causes severe bradycardia, was postulated by Jarisch as a dysfunction of the stretch receptors in the left ventricular wall that served as a trigger of vasovagal reaction. When a very rapid tachycardia results in asystole this is caused by the activation of the cardiac afferent C fibers, or at least this is one hypothesis. When the heart rate slows in such a situation it has been called the Bezold-Jarisch reflex. Although usually thought to result in sinus arrest, AV nodal block has been documented in the dog (even those with normal atrial size). This reflex has a triad of hypotension, bradycardia and coronary artery dilatation. It is both a result of chemoreceptor and mechanoreceptor stimulation. In fact, there is a lot of controversy as to the real mechanism causing this reflex. The cardiac C-fiber afferents (nonmyelinated parasympathetic) travel via the vagus nerve and they act as tension receptors within the walls of the heart. The C fibers are stimulated mechanically with increases in left ventricular end-diastolic filling pressure with increased volume. Chemical substances can also stimulate them. Under physiologic conditions, they are in a tonic stimulation of the parasympathetic system and the sympathetic system output is decreased. With heart failure, the response of the C fibers can be altered. Furthermore, with severe volume depletion in which the baroreceptors are stimulated for sympathoexcitation, there is a second phase that causes a sudden drop in sympathetic release with a sympathoinhibitory response. This latter situation is a paradox as the mechanoreceptors that should be activated with more volume are activated with severe low volume. In the past, this response was believed due to the contraction of the heart with an empty chamber; however, more recently this has been called into doubt because of oversimplification. Instead it likely consists of different mechanical factors to include left ventricular end-diastolic pressure, coronary flow and perfusion pressure, smooth muscle contraction of coronary vessels and cardiac contractility. Another possible reason for the

extreme slowing after a tachycardia maybe the stretching of the sinus node during such an event because of the inability of the atria to empty. Of additional interest is the point that the Bezold-Jarisch reflex is activated with posterior left ventricular wall infarctions in humans. Is it possible that with extreme tachycardia, ischemia develops to cause this response in dogs? With concurrent hypotension and myocardial ischemia the cardiac vagal afferent fibers inhibit the arterial baroreflex.

Triggers that cause vagal mediated syncope include the following:

- Postural
- Coughing
- Stress
- Dehydration
- Vomiting
- Defecation or urination
- Extreme emotions
- Excessive heart rates
- Diseases increasing vagal tone
- Apnea during sleep caused by upper airway disease (yes, owners have reported syncope during sleep in dogs)

Cushing's response

Bradycardia may develop with head trauma, neoplasia of the brain, or certain drugs that evoke a similar response. This is most often the result of a Cushing's response/reflex. In general, a triad of clinical events that include bradycardia with systemic hypertension, irregular breathing and strong pulses due to a wide pulse pressure. The bradycardia is the response to increased intracranial pressure. The mechanism for the Cushing's response is as follows:

- Hydrostatic pressure of the CSF exceeds mean arterial pressure
- Then, the cerebral arterioles are compressed resulting in less blood to the brain
- With less perfusion the central chemoreceptors in medulla sense \downarrow pH and \uparrow pCO₂
- This activates the sympathetic nervous system causing peripheral vasoconstriction and \uparrow peripheral resistance

This \uparrow in blood pressure which triggers the baroreceptors in the carotid arteries and stimulates the parasympathetic system resulting in a bradycardia

A summary of the cardiovascular reflexes that may result in syncope follows:

- Excess vagal tone triggers a bradycardia and hypotension

VASOVAGAL REFLEX

- Abdominal press and release time dependent mixed response

VALSALVA MANEUVER (REFLEX)

- Tachyarrhythmia triggers a bradycardia and hypotension

BEZOID JARISCH REFLEX

- Atrial volume triggers tachycardia

BAINBRIDGE REFLEX

- Brain trauma triggers hypertension and bradycardia

CUSHINGS REFLEX

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1. Boswood, A. et al. Effect of Pimobendan in Dogs with Preclinical Myxomatous Mitral Valve Disease and Cardiomegaly: The EPIC Study—A Randomized Clinical Trial. *J Vet Intern Med* 2016;30:1765–1779

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**Sinus node function and disease in human; what do we know from
Homo sapiens and what have we inferred from other animals?**

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A study in general practice on risk indicators in cats with preclinical hypertrophic cardiomyopathy

Victoria Ironside MRCVS
Hallam Veterinary Centre, Sheffield, UK



In 2011 Phil Tricklebank and myself were awarded a Petsavers grant to carry out a prospective study following a population of cats with preclinical hypertrophic cardiomyopathy (HCM) in general practice. We aimed to identify indicators of the risk of progression of preclinical HCM.

Cats serially underwent physical examination, blood pressure measurement, blood sampling and echocardiography. Development of congestive heart failure (CHF), aortic thromboembolism (ATE) or sudden death (SD) were considered as cardiac related events. Associations between factors recorded at baseline, and on revisit examinations, and the development of a cardiac-related event were explored using ROC analysis.

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Assessment and intra-observer variability of equine left atrial volume using 4D manual LVQ algorithm

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Please note: due to technical difficulties which precluded completion of the previously planned study, this presentation is different from the published programme. VCS are delighted to welcome the following abstract, originally presented at the European College of Equine internal Medicine Congress (ECEIM) in Valencia, November 2019, where Francesca and colleagues were awarded the ECEIM Luis Monreal Best Resident Presentation award. The abstract is also scheduled for presentation at ACVIM Congress in June 2020.

Assessment and intra-observer variability of equine left atrial volume using 4D Manual LVQ algorithm.

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Volume estimation by two-dimensional echocardiography (2DE) relies on geometric assumptions; three-dimensional echocardiography (3DE) does not rely on such assumptions and may allow more accurate assessment of left atrial volume.

To determine the intra-observer variability of equine left atrial volume measurement using a 3DE software analysis package.

Graded datasets of the left atrium from athletic Thoroughbreds horses (n=22; 4-9yrs; 411-534kg), using a Vivid E9 with 3V transducer (GE Healthcare) were retrospectively analysed. Selection criteria excluded horses with grade >3/6 cardiac murmurs. Random generated order measurements were obtained by a single observer on 4 occasions. Real-time three-dimensional maximum and minimum left atrial volumes were measured using the 4D Manual LVQ function v. 202 (EchoPAC, GE Healthcare). Intra-observer variability was assessed via calculation of 1 - the intra-class correlation coefficient (ICC) from random-effect linear models on EDV and ESV with horse added as the random effect ($1-ICC_{\text{horse}}$) in R (v 3.5.2) using the *lmer* and *sjPlot* packages.

Average EDV was 593.1ml (range 349-1029ml) while ESV was 381ml (range 200-695ml), n=22. Lower observer variation ($1-ICC_{\text{horse}}$) for ESV measurements was observed (16%) compared to EDV (23%). There was good agreement between measurements ($1-ICC_{\text{horse}} < 25\%$).

4D Manual LVQ software is a quick, effective and practical tool for obtaining left atrial volume. Error contributing to variation may include EDV/ESV time points, endocardial marker positioning and manual adjustment of the semi-automatic surface recognition. Results may improve with refined measurement guidelines.

Clinical pharmacology of torasemide – how does it compare with furosemide?

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Loop diuretics are the most efficacious of all the groups of diuretic we have available for use in veterinary practice. When dogs with congestive heart failure stop responding to these drugs, the further therapeutic options are limited. Resistance to the tried and tested loop diuretic furosemide is recognised in both human and veterinary clinical practice. The authorisation of torasemide, a more potent and longer lasting loop diuretic provides another option but should torasemide be reserved for resistant cases? What are the mechanisms by which resistance to loop diuretics occurs and how do the pharmacological properties of torasemide and furosemide compare? What is the safety profile of the two drugs? These are the questions I will address in this presentation.

Effects of loop diuretics on the healthy dog kidney – pre-clinical studies

The pharmacology of the loop diuretics has not been studied in great detail. Furosemide and its structural analogues (e.g. ethacrynic acid) were discovered in the 1950s when structure activity relationships of sulphonamides were being explored to exacerbate their effects on the kidney and endocrine systems. As a highly effective diuretic it was authorised for clinical use in human medicine in 1962 (FDA) and has been used in veterinary medicine before full data packages were required for product authorisations. Thus, detailed study of its pharmacology in the dog has not been undertaken. We do know that its main site of action is on the sodium, chloride and potassium co-transporter (NKCC-2) in the loop of Henle. It binds to this transporter from the luminal surface of the epithelial cell (i.e. accesses its target from within the tubular fluid) and inhibits its activity. Because the loop of Henle reabsorbs 2 to 2.5 times as much sodium as the distal tubule, drugs acting in this part of the nephron can stimulate the highest loss of sodium of all the diuretic classes, hence they are sometimes referred to as high ceiling diuretics.

The NKCC-2 transporter moves sodium, potassium and chloride from the tubular fluid into the epithelial cell using the electrochemical gradient for sodium to power this transport. Potassium diffuses back into the tubular fluid whereas sodium and chloride are moved into the interstitial fluid with the sodium pump maintaining the low concentration of sodium within the cell. This transport of sodium and chloride ions by the thick ascending limb of the loop of Henle is important to create a sodium (and chloride) concentration gradient in the

medulla of the kidney and is part of the counter-current multiplier system that does this. The increasing salinity as you move deeper into the medulla is essential for the kidney's ability to produce concentrated urine and so conserve water.

Following single doses of furosemide or torasemide to dogs, urine flow increases rapidly and, as would be predicted from the above mechanism of action, loss of sodium and chloride also increases rapidly. Loss of potassium is much less marked than sodium and chloride. Published studies in the 1980s and 2000s demonstrated that torasemide was 10 to 20 times more potent than furosemide. In addition, the duration of diuretic effect of torasemide is longer than that of furosemide. The dose response curve relating dose to urine volume was steeper for torasemide than for furosemide such that the dose required to produce an equivalent effect to 0.1 mg/kg of torasemide was 1 mg/kg of furosemide (10 fold difference) whereas 8 mg/kg of furosemide was needed to produce an equivalent effect to 0.4 mg/kg of torasemide (20 fold difference).

With repeated dosing for 14 days the effect on the volume of urine produced persists and with high doses actually increases. However, the amount of sodium and chloride excreted diminishes over time. Detailed preclinical studies undertaken by Ceva Animal Health showed that after 4 days of continuous dosing of both furosemide and torasemide to healthy experimental dogs, the amount of sodium excreted per day had reduced towards the baseline level whereas the volume of urine had plateaued for low doses (0.1 and 0.2 mg/kg torasemide) but continued to increase for the high doses (0.4 and 0.8 mg/kg). Thus, in healthy dogs, neither torasemide nor furosemide show tolerance when diuretic (volume of urine) effect is assessed but both show tolerance when their natriuretic effect is studied.

Other effects noted in these pre-clinical studies include (i) activation of the renin angiotensin aldosterone system (RAAS), (ii) plasma urea and creatinine concentrations increase (urea to a greater extent than creatinine) and (iii) glomerular filtration rate (GFR) is reduced by both furosemide and torasemide. These effects are dose-dependent and reversible on cessation of dosing. The dose dependency appears to be the same as the diuretic and natriuretic effects of these two drugs with torasemide being more potent and having a steeper dose response curve.

The mechanisms behind the natriuretic resistance / tolerance to furosemide and torasemide have not been extensively investigated. Activation of the RAAS system will lead to increased sodium reabsorption by the nephron and so acts to physiologically antagonise the action of these drugs. Angiotensin II increases sodium reabsorption from the proximal tubule and stimulates aldosterone secretion from the adrenal gland which increases sodium epithelial transport in the late distal tubule. Other mechanisms proposed include upregulation of the early distal tubule sodium chloride co-transporter (target of thiazide diuretics) and increased expression in the loop of Henle of the NKCC-2 protein, thus increasing the number of transporters present. The fact that diuresis continues whereas tolerance develops to natriuresis suggests this latter mechanism is not very effective. Blockade of NKCC-2 in the loop allows the sodium chloride concentration gradient in the medulla of the kidney to dissipate, preventing the action of ADH on the collecting duct from leading to reabsorption of

water. Thus, if the mechanism of natriuresis tolerance was via restoration of NKCC-2 mediated transport of sodium and chloride by the loop of Henle, both diuresis and natriuresis would be affected to the same extent. When torasemide and furosemide dosing of dogs is stopped after 10 days of daily administration, urinary sodium excretion reduced below baseline for 24 h and then increased back to or overshoot the baseline for the next 24 hours as the physiological antagonistic mechanisms to natriuresis reversed.

The mechanism by which GFR reduces following repeated doses of furosemide and torasemide has not been investigated in any detail. Activation of the RAAS system maintains GFR in the face of hypotension and hypovolaemia and so is unlikely to be responsible. Loop diuretics activate RAAS very quickly following their administration, more quickly than could be explained by a reduction in circulating fluid volume. This is thought to be because the chloride sensor in the very start of the distal tubule (within the macula densa) is blocked by furosemide and torasemide, preventing the sensing of chloride and thus stimulating the signal (a prostaglandin) for renin to be secreted from the afferent arteriole of the glomerulus. The reduction in GFR is reflected in the increase in plasma creatinine concentration. This takes several hours to occur as creatinine has to accumulate within the body fluids after its elimination from the blood is reduced. Because of the exponential relationship between GFR and plasma creatinine, the concentration of creatinine only increases by a relatively small percentage change for proportionately larger change in GFR. The change in urea is disproportionate to the increase in creatinine. This reflects the role urea plays in the urinary concentrating mechanism. Urea transporters (as well as aquaporins) are inserted in the luminal membranes of cortical collecting tubule and the collecting ducts in response to ADH. Urea recycles back into the medullary interstitium and is part of the concentrating gradient generated here. Thus, in response to ADH, the body retains more urea and this is reflected in an increase in its concentration in the blood.

Relevance of pre-clinical data to the clinical use of torasemide and furosemide

Veterinary patients treated with loop diuretics often show an increase in creatinine and urea. This is likely to be related to the reversible reduction in GFR noted in experimental dogs discussed above (for creatinine) plus the action of ADH on the renal tubule leading to retention of urea (in the case of blood urea). In clinical cases the situation is complicated by pre-existing kidney disease which will be present in some dogs. Chronic activation of RAAS by diuretic use could exacerbate the progression of pre-existing kidney disease which is one of the reasons why cardiac disease might contribute to kidney disease (so called cardiorenal syndrome). However, in the majority of patients relatively mild increases in urea and creatinine are not accompanied by other adverse effects (such as inappetance or vomiting) which would be characteristic of clinical evidence of significant kidney dysfunction.

In the pivotal registration clinical trial, which was designed as a non-inferiority trial, ISEMID™ treatment led to a 53% (20 to 72%) reduction in risk of cardiac death/euthanasia or premature study withdrawal when compared to furosemide as a first-line diuretic for the treatment of congestive heart failure. At the same time, ISEMID™ treatment was associated with a higher number of dogs having adverse renal events (which included any elevations of blood urea or

plasma creatinine recorded above the laboratory reference range). The majority (90%) of these renal adverse events were non-serious, asymptomatic and did not require treatment. One interpretation of this difference between the two groups is that the greater changes in BUN and creatinine in the ISEMID™ group is consistent with its higher diuretic efficacy at the dose rate used. Overall, serious adverse events occurred with equal frequency between the two groups.

Whether torasemide has additional effects (not seen with furosemide) in the heart failure patient which reduce progression of cardiac disease to intractable heart failure and cardiac death has been much debated. It was initially suggested that this drug had anti-aldosterone effects based on early in vitro experimental evidence but these results have not been possible to verify by others. Antifibrotic properties by other mechanisms related to collagen metabolism have been suggested with some supporting evidence. Whatever the mechanism, a number of studies in the human literature suggest torasemide has beneficial effects when compared to furosemide which seem likely to be explained by mechanisms that are additional to the diuretic action of this drug. It is possible such effects occur in dogs with heart failure and contribute to the reduced risk of cardiac death/euthanasia or premature study withdrawal.

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Tubes and wires for the heart

H Edward Durham Jr.
*South-West Florida Veterinary Specialists,
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INTRODUCTION

Interventional cardiology refers to several cardiac procedures that involve inserting catheters or pacemaker leads into the internal chambers of the heart and vascular system. Pacemakers will be covered as a separate topic. This presentation will focus on the equipment utilized for intracardiac catheterization. The two most common procedures performed in small animal medicine are balloon valvuloplasty for pulmonic valve stenosis (PS) and closure of the patent ductus arteriosus (PDA); but this information will be the same for other procedures such as right ventricular catheterization to diagnose constrictive pericarditis, cutting balloon valvuloplasty for sub-aortic stenosis, transcatheter septal defect closure, endomyocardial biopsies, or any procedure in which a catheter is inserted into the heart or great vessels for angiography. Diagnostic angiography, once commonplace, has largely been replaced by echocardiography, magnetic resonance imaging, or computed tomography scans. In human medicine, cardiac catheterization of coronary arteries is still a widely utilized diagnostic for coronary artery blockages.

Cardiac catheterization is considered a surgical procedure. All infection alleviation protocols common to any surgical theater are required; including cap, mask, sterile surgical gown, sterile gloves, sterile instruments and drape, and surgical antibiotics. Personnel involved must be competent in the sterile theater. Cardiac catheterization is performed with fluoroscopy, a real-time moving radiograph. Radiation safety protocol must be used for personnel safety, including lead protection for everyone in the theater, even under surgical gowns, lead embedded sterile surgical gloves, reduced exposure time, and proper distance from the radiation for staff not directly involved with the surgery. Video loops can be recorded and replayed to reduce radiation exposure. Transesophageal echocardiography may be added as an adjunct to the imaging modalities. Transesophageal echocardiography has been reported as a sole imaging modality for PDA occlusion^{1,2}.

The steps for any cardiac catheterization can be divided into groups; vascular access, diagnostic imaging, and intervention. Vascular access is the portion of the procedure in which entry into a vessel is achieved to allow the passage of catheters into the target chamber or great vessel. Diagnostic imaging will encompass a ventriculogram, or angiogram to delineate to blood flow, identify the lesion to be corrected, and create a "roadmap" to guide the surgeon with the intervention. This is accomplished by injecting a radiopaque contrast into the bloodstream to highlight the internal structures of the heart and vessels. The

intervention portion will require specialized equipment. This may be balloon dilation catheters, occlusion devices, or stents that can be placed with a structure.

GENERAL INFORMATION

Cardiac catheters come in a variety of diameter sizes, length, and curvature shapes. Guidewires are used to help direct the catheters into the proper location and provide a degree of safety in avoiding perforations. Catheters are sized using the French (Fr.) scale, where 1 Fr is equal to 0.331 mm; thus a 3 Fr catheter is 1 mm in diameter. Unlike the Birmingham gauge scale used for hypodermic needles where increasing numbers equal a smaller diameter (18 g vs 25 g), the greater the French size the larger the diameter. Catheter size refers to the outside diameter (OD) and vascular introducers are referred to by their internal diameter (ID). While this may appear confusing, it allows one to know that a 6 Fr. or smaller catheter will fit inside of a 6Fr. introducer sheath. An introducer will accept any catheter smaller than its reported size. Most catheters are in the 100 cm range, but it should be remembered that all cardiac catheters and wires are designed for human medicine, and consequently, veterinary medicine uses a variety of pediatric and adult length catheters depending on the patient's size.

The other commonly used device for cardiac catheterization is catheter guidewires. Guidewires are utilized to provide stiffness to catheters, be advanced ahead of a catheter to help guide the catheter into the proper location, and secure placement in a location so one catheter may be removed and a new one replaced in its place. Guidewires also provide a degree of safety in advancing catheters inside the vasculature. Catheters can be stiff unyielding to tissue. A guidewire is configured to be soft and pliable at the tip or shaped in such a way to prevent the catheter from abrading the endothelium or perforating a vessel or cardiac chamber. Guidewires are sized by their OD in thousandths of an inch. A wide range of diameters guidewires are available; some as small as 0.010 inches to 0.038 inches. The length may vary from 45 to 260 cm. Specialized wires may be coated to make them easier to pass, be coated with heparin, have variable flexibility in the tip, moveable cores, or have "J" shapes. These features are designed to solve the unique problems experienced in cardiac catheterization.

Injection of radiopaque contrast may be performed by hand injection for small volumes. Larger volumes of contrast may be injected with the aid of a power injector. Power injectors are mechanical devices that hold specialized syringes which can be load with large volumes of contrast and injected through diagnostic catheters at a very fast rate and controlled pressure. Power injectors are often required for good imaging of large chambers such as the ventricles since the contrast is viscous and difficult to inject quickly by hand. Additionally, ventricles empty very quickly and one cardiac cycle may pass before all the contrast can be injected by hand.

VASCULAR ACCESS

Vascular access has been called the most important part of any procedure simply because, without it, nothing can be done. The vessel accessed with be determined by the area of the

heart needed to be reached. For the PS balloon valvuloplasty, typically the femoral or jugular veins are accessed. This allows passage of catheters through the venous system to the right atrium, passing through the tricuspid valve into the right ventricle (RV), and out the pulmonic valve. An approach from the femoral or carotid artery is commonly used for closure of the PDA.

An “introducer” is used to gain vascular access. The introducer is similar to a large-bore intravenous catheter, but has the added feature is a hemostatic valve that allows passage of small diagnostic catheters through it without continual blood loss. Many introducers will have a sidearm with a 4-way stopcock included allowing for flushing the introducer with catheters in place. The introducer may be inserted into the desired vessel percutaneously or via a surgical cut-down. Percutaneous placement is often accomplished with the modified Seldinger technique in which a needle is inserted into the artery or vein. Once blood flows from the needle, a short (45cm) guidewire is inserted through the needle into the vessel. The needle is then removed while holding the guidewire in place within the vessel. The introducer and its vessel dilator are inserted over the guidewire to “guide” it into the vessel. Once the introducer is securely in the vasculature, the guidewire and dilator are removed. Catheters and wires may now be passed into the vasculature.

DIAGNOSTICS

Once vascular access has been obtained, imaging of the vascular lesion is typically performed. Many types of catheters have been specially designed for individual procedures. It is beyond the scope of this presentation to discuss all of the catheter types. A potential problem with high-pressure contrast injection into a vessel is endocardial/endothelial staining in which the contrast stream is injected directly against tissue and infiltrates the cells and stain the tissue rather than flushing away with the blood pool. Catheters have been specifically designed to prevent this by using closed ends with side holes for contrast to escape rather than a sharp stream leaving an open end catheter. They come in various curves and bends to facilitate placement. The NIH and Berman catheters are examples. These catheters are commonly used for ventriculograms. Pigtail catheters, commonly used for injections in the great vessels, are open-ended but are curled in a full 360 degrees to prevent contrast from shooting against the endothelium by directing it into the center of the bloodstream. Many styles of pigtail catheters will also have side holes to increased safety and improved performance. Open-end catheters may be used for small volume low-pressure contrast injection as used for highlighting and tracking a catheter through small arteries. Guidewires are always used when advancing catheters to protect the internal structures and simplify directing the catheter to the correct location.

Open-end catheters are used to measuring intracardiac pressure. They may have a small latex balloon at the tip (distal end) which when inflated will be pulled along with the blood flow (flow directed Swan-Ganz style) to help guide the catheter through the heart and great vessels, even without the aid of fluoroscopy. Once in place, any guidewires used can be removed and pressure recorded. The catheter is then retracted out of the patient slowly

stopping to each chamber along the way to record pressure. The “bare” catheter is never advanced into a chamber to record pressure due to the risk of intimal damage or perforation. Other multi-lumen catheters are made for measuring cardiac output (thermodilution), and monitoring of pulmonary capillary wedge pressure which equates to left atrial pressure as well as sampling of blood at various locations along the length of the catheter. After the contrast is injected, the fluoroscopy unit will typically be able to save the contrast injection as a video loop or still image. The surgeon can then superimpose this image on the screen to create a “roadmap” to guide the next steps in the procedure. The contrast will be washed out of the heart, diluted in the bloodstream making it invisible through subsequent cardiac cycles, then excreted by the kidneys within hours.

INTERVENTIONS

The two most common interventions in veterinary small animal cardiology are the closure of the PDA with the Amplatz® Canine Ductal Occluder (ACDO) and balloon valvuloplasty for PS. The ACDO is a nitinol mesh device shaped somewhat like an hourglass. The device is attached to a braided steel cable that can be advanced through a catheter or long introducer into the PDA. Once in place, the ACDO encourages a thrombus to form in its structure occluding the PDA.

Balloon valvuloplasty catheters are specially designed catheters with a balloon at the distal end. The balloon comes is sized by its diameter and length. Sizes range from a couple of millimeters diameter up to 3 cm, and lengths from 2 cm to 11 cm. The selection is based on the size of the patient’s valve to be dilated. The balloon is made of a tough micro-thin noncompliant material to allow it to expand to dilate the valve without rupture. The catheters have a dual lumen; one centrally located to carry the guidewire, and a second lumen that allows for balloon inflation. These catheters are stiff and have tapered ends, therefore to navigate them into pulmonic valve a guidewire MUST be used.

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Pacemaker therapy for symptomatic bradyarrhythmias

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Pacemakers are electronic devices that stimulate the myocardium with electrical impulses to maintain or restore cardiac output and a stable paced rhythm. The most common indicators for permanent pacemaker implantation are sinus node dysfunction (SND) and atrioventricular (AV) block (high-grade second or third degree) causing bradycardia or intermittent prolonged sinus arrest.

Bradyarrhythmias

Clinically significant bradyarrhythmias are those that result in outward clinical signs and/or put the patient at risk of sudden cardiac death. Not all bradyarrhythmias require pacemaker implantation. Some occur due to an underlying systemic disease that only requires primary treatment with subsequent resolution of the bradyarrhythmia. Diseases of the brain, eye, thorax or abdomen and various drugs can cause pronounced bradycardia, even to the degree of AV block through the stimulation of the vagal nerve.

Primary bradyarrhythmias that tend to require pacemaker implantation include:

- Atrioventricular block: the problem is at the level of the AV node. There is a delay or even complete blockage of the electrical impulse as it is conducted from the atria to the ventricle through the AV node.
- Sinus node disease /sick sinus syndrome: the problem is most commonly at the level of the sinoatrial (SA) node, but other areas of the conduction system can be affected, resulting in bradycardia. The episodes of marked bradycardia along with sinus arrest or block tends to lead to rescue escape beats.
- Atrial standstill: this is a condition where normal atrial muscle has been replaced with abnormal tissue and cannot propagate the electrical impulses. This results in bradycardia.
- Sinus arrest: this is a condition whereby the SA node ceases to generate an electrical impulse that normally stimulates the myocardial muscle to contract.

Bradyarrhythmic patients are classed as cardiovascular unstable and result in being haemodynamically compromised.

Signs & Symptoms

Sinus bradycardia is defined as a heart rate (HR) lower than 60 beats per minute. Symptoms of bradyarrhythmia can vary from mild to major, from simple lethargy to regular syncopal

episodes. A descriptive and complete patient history and detailed clinical assessment of the patients is vital to treat, stabilise and resolve HR derangements. Weakness, collapse, exercise intolerance, syncope, ascites, pulmonary oedema (congestive heart failure), and even sudden cardiac death can occur with bradyarrhythmias. However, it is not uncommon for veterinary patients to appear outwardly normal despite having a bradyarrhythmia.

Diagnosis

The first physical exam finding is bradycardia, the cause of which should be confirmed with an electrocardiogram (ECG). An ECG is the best diagnostic test to identify and diagnose the specific type of bradyarrhythmia. A Holter monitor (home monitoring device) is an ECG device that is attached to the patient and records the cardiac rhythm over a 24-48 hour period (or longer) and can determine the frequency and severity of the arrhythmia, if an intermittent arrhythmia is suspected but not presented during clinical assessment. A loop recorder is an implantable ECG recorder and is another tool that can help to identify very intermittent arrhythmias. Additional tests such as blood work should be used to screen for conditions that might be contributing to the bradycardia, such as an infection, hypothyroidism or an electrolyte imbalance. An atropine test may be necessary to help differentiate if the bradyarrhythmias are primary or secondary to a systemic disease (high parasympathetic or vagal tone).

Pacemaker Therapy

Pacemakers consist of two main components: the pulse generator/battery and the pacing lead. The pulse generator delivers an electrical impulse through an electrode in the pacing lead to stimulate the myocardium, causing the heart to contract. All pacing systems require two electrodes for an electrical circuit to be maintained and cardiac pacing to occur. Single chamber pacemakers, stimulate the atrium, but more commonly the ventricle to contract, and a dual chamber pacemaker, stimulates both the ventricle and the atrium to contract in a rhythmic fashion. Pacemaker implantations are more commonly performed in dogs as they are more likely to develop bradyarrhythmias and have a naturally slower escape (safety) rate, which makes them more symptomatic and unstable. Although cats may develop third degree AV blocks, their escape rate is often over 100 bpm or they commonly have an underlying cardiac condition and therefore a pacemaker is generally either not necessary and/or indicated.

Types of Pacing

Temporary pacing

Temporary transthoracic pacing uses external electrode pads that attach to the lateral aspects of the thorax over the area of the heart. This type of pacing can be painful, as it results in skeletal muscle stimulation and requires anaesthesia and potentially a neuromuscular blocking agent to reduce the degree of muscle stimulation and therefore movement of the

patient. This type of pacing tends to be used in emergency pacing situations, where the patient needs emergency pacing due to a severe life threatening bradycardia.

Temporary transvenous pacing uses a temporary pacing lead introduced via a peripheral vein, usually the saphenous, but occasionally the jugular, This lead will be guided towards the heart and positioned into the right ventricle. This technique can be performed under sedation or premedication. This can be performed prior to a permanent pacing solution (stabilizing the heart rate, which would make the required general anaesthetic safer) or potentially even as a temporary solution for specific cases of transient bradyarrhythmias.

Permanent pacing

Endocardial transvenous pacing is the most common type of permanent pacing, where the pacing lead is passed from the jugular vein, through the vena cava and into the right ventricular apex of interventricular septum. Either jugular can be used, but the right is typically used in an attempt to avoid any undiagnosed congenital anomalies such as persistent left cranial vena cava. Once in an appropriate location, the lead is either actively screwed into place, or passively secured with 'fish hook' like projections that will attach to the walls of the ventricle. This procedure is performed under fluoroscopy to assist with lead placement. The lead will then be connected to a pulse generator located in the neck.

Epicardial pacing systems consist of a pulse generator with a pacing lead that is surgically attached to the left ventricular epicardium; this can be sutured to the myocardium or a self-tapping screw can also be used. This system is implanted via an abdominal incision with a transdiaphragmatic approach to the right ventricular apex. Epicardial pacing is rare and is more commonly implanted in multiple lead dislodgement patients, those with prothrombotic conditions or skin disease, but also in smaller sized patient (cats /ferrets) when vascular access is restrictive. The lead will then be connected to a pulse generator located within the abdominal wall musculature.

Post Procedure Patient Care

Transvenous endocardial pacing is the most commonly used pacing method. The surgical site is a small incision over the jugular for the insertion of the pacing lead and another small incision dorsal to the latter for the insertion of the generator; therefore the post-operative care pain is reduced compared to epicardial pacing. Epicardial pacing is typically similar management to any abdominal surgery. Management of all patients following pacemaker implantation involves minimising the risk of lead dislodgement in the short term and avoid any damage to the pulse generator and lead in the lateral aspect of the neck. Post implantation care can include opiate/sedation/analgesia to keep the patient quiet and to reduce the risk of lead dislodgement. A bandage is placed around the neck to protect the surgical site, reduce patient interference with the wound, reduce neck movements and reduce seroma formation. Once the pacemaker has been implanted, it is mandatory that blood sampling is not performed from the pacing jugular vein used for pacing and the owners should be informed that their pet should never wear a neck collar as either could result in

catastrophic damage to the lead resulting from lead fracture or dislodgment. Antibiotic therapy should be continued post operatively for 7-14 days to reduce the risk of infection. Post operatively there is a variety of tests that can be performed to confirm lead placement: right lateral and dorso-ventral radiographs can be used to confirm lead location and an ECG can be used to verify the pacemaker function, assess the paced complexes morphology and the pacing rate. Pulse generators are programmed to a base setting at the time of discharge and clients are asked to return for regular programmer pacing checks to ensure sufficient battery life, complete system checks and occasionally the endocardium develops a reaction with the pacing lead causing increased resistance to the pacing impulse.

Complications

Pacemaker complications are well documented and can range from mild to serious. Irrespective of the lead type, lead dislodgement is the most serious complication and this can typically occur days after the pacemaker implantation, however, it can also occur years after implantation. Longer term, congestive heart failure from pacemaker syndrome or from concurrent cardiac disease may develop. Post implantation infections occur infrequently and can arise months after implantation but can be fatal. In the event of pulse generator infection, aggressive antibiotic therapy should be pursued, ideally after culture results, however, removal of the pulse generator and lead may be necessary and a new pacing system may have to be implanted. The most common minor complication is seroma/haematoma formation around the generator. Seromas do not generally require drainage, as they tend to self-settle with time. A bandage is recommended to reduce or prevent the reoccurrence and antibiotics are prescribed.

Summary

Cardiac pacemaker implantation within the veterinary field is becoming more popular for the treatment of symptomatic bradyarrhythmias. Prognosis following a pacemaker implantation depends on any concurrent structural or functional cardiac disease, however, an excellent outcome can be expected for patients with primary bradyarrhythmias without any underlying cardiac disease. Recognising clinical signs early and being able to identify for bradyarrhythmias can achieve a quicker and potentially lifesaving treatment. Understanding the indicators for pacemaker implantation and the importance of post-operative care can alleviate potential problems with pacemaker systems.

What do you need to know for your first intervention?

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Imagine that you come to work in the morning, you look at the diary and see an appointment booked with the words “suspicion of PDA” and it’s a dog, ME, 7 week old poodle. As a cardiology nurse what can you do to aid the smooth running of the clinic? Which additional information do you need in order to ensure this?

1. You will need confirmation of the diagnosis. For this, echocardiography is essential.
2. Ensure the patient is a good candidate for treatment.
 - a. Owner has the funds.
 - i. The patient is clinically stable for a general anaesthetic.
 - ii. General blood profile results are available.
 - iii. The patient is not in congestive heart failure.
 - b. The patient has no other congenital cardiac abnormalities or systemic disease present.
3. Start planning for the intervention.
 - a. Pre-book theatre space.
 - b. Arrange anaesthesia.
 - c. Arrange a cardiologist or surgeon to perform the surgery.

To prepare for the intervention, start by checking that all the potential consumables required for surgery are available. It is a good idea to ensure you have enough and if you are requiring to reorder items.

How do you know what items you’ll need? Once the diagnosis of PDA has been confirmed you will need to find out the technique that will be used to occlude the PDA.

The most common techniques available are surgical ligation and interventional (keyhole) techniques. Examples of the latter are Amplatzer Canine Duct Occluder (ACDO) and the Amplatzer vascular plug II. Each technique requires a slightly different set up for theatre layout, equipment selection and patient preparation and positioning.

These small pieces of information allow the nurse to pre-empt the veterinary clinicians’ needs. It will seem like we are psychic, when in reality our nifty crystal ball gazing and mind

reading abilities are simply our exceptional abilities to gather key pieces of information from our clinicians' and use our foresight to ensure our services are efficient. This allows our clinicians' the luxury to concentrate on dealing with their cases.

Mind the Gap: Closure of the patent ductus arteriosus

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INTRODUCTION

Patent ductus arteriosus is one of the most common congenital heart defects found in small animal medicine. This defect is the patent remnant of the ductus arteriosus, one of three physiological shunts in fetal circulation. The ductus arteriosus shunts blood from the pulmonary artery to the aorta in the fetus bypassing the lungs. Shortly after birth, the ductus arteriosus closes and this shunt vanishes. If this fails to happen the patient will have a patent ductus arteriosus (PDA).

Because after parturition, the pulmonary vascular resistance drops below aortic pressure blood will move from the aorta to the pulmonary artery (PA). This is referred to as a left-to-right shunt because blood goes from the systemic circulation (aka left heart circulation) to the pulmonary circulation (aka right heart circulation). Left to right shunts volume overload the left atrium and ventricle and will eventually lead to heart failure in about two-thirds of patients. Closing the PDA is the most effective form of treatment and dogs with PDA's that have been closed as puppies will continue to live normal lives.

Traditionally open thoracic surgery and ligation of the PDA have been the most common treatment. In recent decades, transcatheter occlusion devices have become popular to avoid the risk and discomfort of thoracic surgery. Vascular plugs and embolization coils were used to close PDA's. In 2007 a specially designed device was reported for use in dogs the Amplatz® Canine Ductal Occluder (ACDO)¹. A similar device had been created for human PDA, but dogs demonstrate slightly different morphology in the ductal shape and the human device did not fully suit the need. The ACDO has simplified the transcatheter closure of PDS significantly. Only very small patients that have arteries too diminutive to accept catheters and very large PDA's require open thoracic ligation. The ACDO is a nitinol (a nickel and titanium alloy) mesh device with basically an hourglass shape; one side being a flat disc, and the other somewhat cup-shaped. The flat disc side is generally deployed into the PA and the cup portion into the ampulla of the ductus. The narrow "waist" is centered in the ostium emptying into the PA. The ADCO come from the manufacturer collapsed inside of a small tube or "straw", with a braided cable threaded onto the ACDO. Nitinol has excellent shape memory, and once outside the straw, the ACDO will spring to its hourglass shape. The device is released in the PDA by unscrewing the cable from the ADCO once positioned.

PROCEDURE

Vascular access is typically performed transarterially¹. The procedure has also been described with an approach from the venous approach^{2,3}. The femoral artery allows for relatively easy access to the descending aorta where the PDA intersects the aorta. The anatomy of a PDA most often extends slightly caudally and dorsally from the distal main PA to the aorta in a right lateral projection. Thus advancing a catheter from the caudal portion of the patient allows it to track easily into the ampulla of the ductus. Vascular access may be done percutaneously or via surgical cut-down to expose the femoral artery. The size of the introducer is based on the size of the catheter necessary to deliver the ACDO. The ACDO is sized by measuring the ostium of the PDA where it enters the main PA. The ACDO chosen will have a waist approximately 1.5 to 2 times the diameter of the ostium. The ostium measurement is usually achieved during the initial diagnostic echocardiogram. The various size ACDOs require delivery catheters of increasing sizes depending on the size of the ostium. The size of the ostium can generally be related to the size of the patient and adequate catheters may be introduced into the artery of the patient. In other words, a larger ostium appears in larger patients who have larger arteries and can accept larger bore catheters. Should a large ostium occur in a small patient then surgical correction may be a better option; however, techniques have been described for placing an ACDO in patients as small as 1.5 kg⁴.

After arterial access is established an aortogram is performed. Radiopaque contrast is injected in the aortic arch through a diagnostic angiographic catheter. The contrast will fill the aortic arch, the proximal descending aorta, flowing through the PDA, and into the main PA. A pigtail ventriculography catheter, Berman, NIH style, or other closed-end catheters can be used. The resultant image can be used to confirm the echocardiographic assessment of the ostium size and create a roadmap to guide the placement of the ACDO.

Once the anatomy has been delineated, a catheter or long introducer sheath with a sufficient internal diameter to accept the collapsed ACDO is advanced in the aorta. To advancement of the delivery catheter a straight of angles floppy tip guidewire across the PDA from the aorta side through to the PA side. The soft floppy tip of the wire is often carried with the blood flow through the PDA to simplify the advancement of the delivery catheter. Once the delivery catheter has traversed the PDA the guidewire is withdrawn. The ADCO on its delivery cable is advanced through the catheter until the first disc opens within the PA. The entire assembly (ACDO and delivery catheter) is then withdrawn until the disc seats up against the PA side of the PDA ostium. Transesophageal echocardiography (TEE) is useful to confirm placement. To deploy the cup portion within the ductal ampulla, the delivery wire is held still, and the catheter withdrawn back over the delivery cable until the cup springs into shape. A Branham reflex of decreased heart rate may be noted.

With the device correctly positioned and deployed blood flow will cease in approximately 10 minutes. Confirmation of occlusion can be done with TEE or transthoracic echocardiography if available. If a long introducer sheath with a sidearm flush post is used to deploy the ADCO, contrast medium can be injected around the delivery cable to check

for residual blood flow through the PDA. The cable is then unscrewed from the ADCO after confirming full occlusion, and the device is left in place. The cable is removed, and a final contrast injection is performed to verify the occlusion of the PDA. A very small amount of residual flow may be noted. This most often will fully resolve within 24 hours. The catheter is removed, then the vascular access introducer and the surgical cut-down sutured closed. Most often the femoral artery is ligated with no ill effect to the patient, but repair of the arteriotomy can be performed.

The patient may be sedated during recovery, and be exercise restricted for 2-3 weeks to keep arterial blood pressure low to prevent dislodgement of the device into the pulmonary vasculature. An echocardiogram the following day will confirm full occlusion or quantify any residual flow if present. A lateral radiograph can be used to verify ADCO placement for future reference should complications arise.

Complications are infection of the surgical cut-down, hemorrhage of the arteriotomy if repair is elected, infection of the ADCO⁵, or dislodgement of the ADCO. If the ADCO dislodges during the procedure, and a second larger device can be placed and the first one has been reported to be removed via transvenous access afterward⁶. The device will achieve full endothelialization within several weeks and become permanently embedded in the tissue. Therefore the first few weeks are the critical time for post-procedure dislodgement. Should dislodgement occur in the days following the procedure a second procedure must be performed; either open thoracic repair or a second ADCO.

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An update on the classification, diagnosis and management of feline heart disease



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The American College of Veterinary Internal Medicine (ACVIM) is about to publish a consensus statement which will provide guidelines for the diagnosis, management and treatment of the cat with heart disease. These recommendations will help veterinary nurses develop their practice, based on the best of current thinking and understanding of feline heart disease.

Disease definitions

The new ACVIM guidelines accept that it has been challenging for veterinary surgeons to define and classify feline heart disease. The recommendations also acknowledge that if there is not a trained cardiologist available, then skilled techniques such as echocardiography prove difficult. The new guidelines have changed the way that the disease is classified, but importantly also, put the emphasis on a new staging system which can help guide treatment, changing focus to a clinical approach of the patient.

The new approach to defining feline cardiomyopathy is based on phenotypical features rather than the underlying cause. The consensus statement has been adapted from the European Society of Cardiology classification system, where four phenotypic categories have been identified for use in cats. These are hypertrophic cardiomyopathy (HCM), restrictive cardiomyopathy (RCM), dilated cardiomyopathy (DCM) and arrhythmogenic cardiomyopathy (ARVC). It is to be noted that phenotypic group can change with time however, either because of disease progression, or due to concurrent disease processes such as hypertension or hyperthyroidism.

Staging system

The new staging system is based on a combination of classification systems, the human American Heart Association system, and the ACVIM system already in use for dogs with chronic valvular disease. A summary of the classification system is listed below.

Stage	Description
A	This group includes cats that are at risk of, or are predisposed to, developing cardiomyopathy but currently have no evidence of disease.

B	Cats in this group have been diagnosed with cardiomyopathy, but have no clinical signs as yet. This group is divided in to two separate categories:-
	B1 – Cats at low risk of developing congestive heart failure (CHF) or aortic thromboembolism (ATE) imminently.
	B2 – Cats at higher risk of developing CHF or ATE imminently.
C	Cats that currently have, or have had, signs of CHF or an ATE.
D	Cats that have become refractory to conventional CHF treatment.

Diagnosis

The ACVIM consensus statement also makes recommendations on diagnostic techniques. Genetic testing is still recommended for Maine Coons and Ragdolls that are being considered for breeding, but the statement makes clear the limitations that exist with this test, asserting the value of physical examination and echocardiography for stage A cats. Recommendations for physical examination remains much the same, with emphasis on the fact that some cats will have disease present, but no auscultatory changes. It states that certain prognostic indicators such as a gallop rhythm or arrhythmias are more likely to be associated with cardiomyopathy. Echocardiography is still considered the gold standard method of cardiomyopathy diagnosis, although the guidelines acknowledge the difficulty in achieving gold standard measurements. Instead, it recommends for the unstable patient, or if no specialist is available, to perform a focused point-of-care exam, using ultrasound to assess for abnormal fluid accumulation, such as pulmonary oedema with the presence of B lines, or pleural or pericardial effusions. It can also be used to estimate left atrial (LA) size and left ventricular systolic function.

The value of radiography is questioned. It used to be regarded as gold standard for the diagnosis of pulmonary oedema, but the guidelines emphasise that radiographs should not be performed if the procurement of them would add to the stress of the patient. Furthermore, the statement highlights that the radiographic pattern associated with cardiogenic pulmonary oedema is highly variable in cats, and evidence of left atrial enlargement and recognition of distended pulmonary vessels is inconsistent. Additionally, the classic 'valentine' shape of the heart is now not thought to be specific for a diagnosis of HCM.

Recommendations are also made about the usefulness of biomarkers, in particular the use of point-of-care tests. It is now suggested to monitor thyroxine levels in cats over the age of 6, if they have auscultatory abnormalities, even if there is no suggestion of concurrent left ventricular hypertrophy. Hypertension is a common finding in cats, and it is advised that any cat with increased left ventricular wall thickness, should have blood pressure measured.

Treatment and nursing guidelines

A brief overview of the treatment and nursing guidelines is listed below.

Stage	Treatment guideline	Nursing recommendation
A	No treatment recommended. Suggested that queens of high risk breeds are assessed regularly by cardiologist. Genetic tests performed where applicable.	None.
B1	No treatment recommended. For cases diagnosed with severe left ventricular outflow tract obstruction (LVOTO), atenolol may be used if it can be given consistently to the cat.	Annual screening recommended to owners.
B2	Clopidogrel recommended for cats at risk of developing ATE. Suggested indications are moderate to severe LA enlargement, low LA fractional shortening, low LA appendage velocities and the presence of spontaneous echo contrast. Other anti-thrombotics may also be considered in cats at high risk. Progression of disease should be monitored, but weighed against the risk of stress to the cat. For cats with ventricular ectopy (either HCM or ARVC), treatment with atenolol or sotalol may be recommended.	Owners to monitor resting respiratory rate.
C	<u>Acute decompensated failure</u>	<u>Acute decompensated failure</u>

	<p>Diuresis, oxygen, sedation if necessary, thoracocentesis if appropriate.</p> <p>In cases presenting with low cardiac output, consider pimobendan if no obvious LVOTO. If no improvement with pimobendan, consider continuous rate infusion of dobutamine.</p> <p>It is recommended to send the patient home as soon as the patient has been stabilised.</p> <p>Re-examination recommended 3-7 days later to check for the resolution of CHF, renal function and electrolytes.</p>	<p>Minimise stress, provide oxygen, and prepare thoracocentesis equipment as required.</p> <p>Monitor blood pressure, respiratory weight, body weight, body temperature and estimated urine output.</p> <p>Owner to monitor respiratory rate at home. Therapeutic goal to maintain rate to under 30 breaths/minute at home.</p>
	<p><u>Chronic heart failure</u></p> <p>Furosemide - After initiation of furosemide, examine in 3-7 days to check renal and electrolyte parameters.</p> <p>Clopidogrel - Recommended for any cat with moderate to severe LA enlargement.</p> <p>Pimobendan - Recommended in cats with no significant LVOTO</p> <p>Re-examination in 2-4 months, but level of stress should be considered. Presence of concurrent disease may warrant more frequent checks.</p>	<p><u>Chronic heart failure</u></p> <p>Owner to monitor respiratory rate at home. Therapeutic goal to maintain rate to under 30 breaths/minute at home.</p>
D	<p>Torsemide is recommended if the patient has become refractory to furosemide.</p> <p>Spironolactone may be added to aid diuresis.</p> <p>Pimobendan may also be added.</p>	<p>High salt foods are to be avoided, unless cardiac cachexia is a concern. If this is the case, calorie intake should be prioritised.</p> <p>Regular body condition scoring and body weight should be recorded.</p>

	<p>Potassium or taurine supplementation as indicated.</p> <p>However, owner/cat compliance need to be considered when adding additional medication.</p>	
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Management of ATE

Euthanasia is still the most common outcome for an ATE in general practice. The ACVIM statement outlines that poor prognostic indicators include hypothermia, more than 1 limb effected, and the presence of CHF. However, if treatment is to be initiated, effective pain management is crucial, especially within the first 24 hours. It is recommended that analgesics such as fentanyl or methadone are used.. Until the cat has been stabilised and can be given oral medication, either a low molecular weight heparin, unfractionated heparin, or an oral Xa inhibitor (such as rivaroxaban) should be administered. Clopidogrel is recommended as soon as oral medication is tolerated. Oral Xa inhibitors can replace heparin. Post ATE management recommendations are that the patient be re-examined 3-7 days after discharge, and regular checks post event to monitor for necrosis, electrolyte imbalance, appetite and treatment compliance, as well as for neuromuscular improvement. However, this needs to be assessed in conjunction with the additional anticipated stress to the cat. Resting respiratory rate should be monitored at home.

Case Report: Care of a cat with idiopathic pericarditis and subsequent myocardial disease, congestive heart failure and atrial fibrillation

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This case report focuses on a 5-year-old, male (neutered), domestic short hair feline, referred for dyspnoea and tricavitary effusion. Investigations revealed a pericardial effusion with a thickened pericardium causing cardiac tamponade, pleural effusion and hypertrophic cardiomyopathy phenotype. Pericardial effusion leading to cardiac tamponade returned despite medical management and pericardiocentesis. The patient therefore went for an pericardiectomy. After histopathological analysis and infectious disease screening, the final diagnosis was idiopathic pericarditis. The patient recovered well and was discharged from hospital in December 2018.

Over the course of the next year, the myocardial disease severely progressed. There was now moderate left ventricular hypertrophy and severe left atrial dilatation. Risk of thromboembolism was evident as “smoke” (slow moving blood) was observed in the left auricle. When the patient returned in November 2019, he presented in left sided congestive heart failure (CHF) and atrial fibrillation. Treatment of CHF with medical management was initiated. 1.7mg/kg of furosemide was prescribed twice daily as well as clopidogrel and rivaroxaban as antithrombotic therapy.

One week after initiating treatment of left sided CHF, the patient returned for re-examination. There was a good response to medical treatment of CHF with no further evidence of pulmonary oedema. Atrial fibrillation remained and the patient remained tachycardic with a mean heart rate of 280bpm. At this point it was decided that heart rate control should be initiated.

The patient began on 1mg/kg of diltiazem three times daily as well as 0.25mg/kg of pimobendan twice daily. The patient was then hospitalised for 24 hours of holter placement after a week of diltiazem to assess the success of rate control. The mean heart rate remained high and the diltiazem dose was increased to 2mg/kg three times daily in order to achieve a better rate control. The patient was discharged and remains at home with a good quality of life. The patient is monitored closely by the owner, using a sleeping respiratory rate diary and monitoring for clinical signs of congestive heart failure.

Up, up and away: The technique of balloon valvuloplasty

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Balloon valvuloplasty (BVP) is an interventional cardiac procedure to reduce the right ventricular (RV) outflow obstruction of pulmonic valve stenosis (PS). This procedure does not typically cure RV obstructions but reduces them sufficiently to keep the patient symptom free. Pulmonic stenosis is dysplasia of the pulmonic valve and may appear in various forms¹. The two most common groups are a hypoplastic valve annulus, with or without malformed valve leaflets and a normal annulus with fused but otherwise normal leaflets. The hypoplastic annulus is narrowed creating restricted blood flow out of the RV, increasing RV pressure. Cardiac remodeling of concentric RV hypertrophy is related to the degree of obstruction. Fused or poorly mobile valve leaflets exacerbate the obstruction. If the valve annulus is normal size however, BVP is a good option for improving leaflet excursion and reducing the RV obstruction. The third type of PS can be seen primarily in bulldogs and boxer dogs. This type of PS is created by a lack of a right coronary artery ostium leading the left coronary to branch over the pulmonary artery (PA) causing an obstruction²⁻⁴. Balloon valvuloplasty is contraindicated in these patients due to the risk of coronary arterial rupture and sudden death.

Catheters for BVP are specially designed catheters with a balloon at the distal end. The balloon is sized by its diameter and length. Sizes range from a couple of millimeters diameter up to 3 cm, and lengths from 2 cm to 11 cm. The selection is based on the size of the patient's valve to be dilated. The balloon is made of a tough micro-thin noncompliant material to allow it to expand to dilate the valve without rupture. The catheters have a dual lumen; one centrally located to carry the guidewire, and a second lumen that allows for balloon inflation. Overall catheter length is usually 100 cm with a shaft size between 5 and 11 French depending on balloon size. These catheters are stiff and have tapered ends, therefore to navigate them through the RV and across pulmonic valve a guidewire MUST be used.

PROCEDURE

Vascular access is achieved via the jugular or femoral vein. The route from vena cava requires the catheters and wires to traverse the right atrium (RA), tricuspid valve (TV), the RV and out the RV outflow tract to the pulmonic valve (PV). From the femoral approach, the catheter needs to dip ventral from the caudal vena cava into the RA then back dorsal to cross the PV. From the jugular approach catheters and wires make a large 360-degree loop to cross the TV and exit the RV outflow tract. The introducer size will be dictated by the interventional balloon chosen, and this information is included with the balloon

catheter packaging. For this reason, the jugular vein is often chosen for large catheters due to vessel size. Patient size is correlated to the size of the catheter required in most cases. The balloon catheter chosen will be 1.2 to 1.5 times the maximal annular diameter⁵. Vascular access can be performed percutaneously with a modified Seldinger technique or via surgical cut-down. Care should be taken not to use an introducer in small patients that extends into the RA. This may cause an RA perforation or difficulty passing catheters across the TV.

Depending on the technology available a pressure gradient is measured between the RV and the main PA. This may be performed by transesophageal echocardiography and/or direct cardiac catheterization. Direct cardiac pressure measurement requires the placement of an end-hole catheter into the main PA. A suitable guidewire is chosen (J tipped or soft angled wire approximately 150 cm long), and typically, a balloon-tipped flow-directed catheter and advanced through the introducer. This type of catheter has a balloon just proximal to the end-hole. The guidewire will be extended out the end of the catheter to prevent perforations in the cardiac structures while the catheter is advanced. Once the catheter tip has passed through the TV, the small balloon at the tip of the catheter can be inflated allowing it to be carried with blood flow toward and out the PV. After crossing the PV, the catheter can be advanced out into the pulmonary vasculature. With the balloon inflated, the catheter can be advanced until it wedges in a small pulmonary artery. When the wire is removed, the pressure recorded is known as Pulmonary Capillary Wedge Pressure and reflects left atrial pressure. The end-hole is isolated from the main PA flow by wedging the balloon in a pulmonary arterial pre-capillary vessel effectively sealing it away pulmonary arterial pressure. Then the balloon is deflated and withdrawn into the main PA and pressure recorded. Next, the catheter is withdrawn into the RV and the pressure recorded there. The PV pressure gradient is the difference between the RV pressure and the main PA pressure. The pressure gradient will be measured at the end of the procedure to assess the effectiveness of the intervention. Finally, the RA pressure can be recorded as the catheter is further withdrawn from the patient.

A ventriculography catheter is then advanced in the RV. The catheter for ventriculography will have a closed-end to prevent myocardial staining such as a Lehman, NIH or Berman catheter. A pigtail catheter can be used but it presents an increased risk of myocardial staining due to its end-hole. The Berman catheter is preferred in many instances. This specialized catheter has multiple side-holes and a balloon at the very tip of the catheter distal to the holes. This configuration allows for flow-directed advancement and protection of myocardial injury by encasing the catheter tip in a soft balloon. A power injector is commonly used to provide adequate filling of the RV lumen between heartbeats with iodinated radiopaque contrast to brightly delineate the RV outflow tract and PV. This angiogram is used to create a roadmap to guide the intervention and to make more measurements of the PV annulus to compare to those acquired during the diagnostic echocardiogram. An intervention balloon can then be selected. The ventriculography catheter is removed from the patient.

The interventional catheter, as mentioned previously, is quite stiff and tapered at the distal tip to facilitate pushing it through the hemostatic valve of the introducer. Consequently, they are very dangerous to advance in the patient without a guidewire. They are also sufficiently stiff that without a wire to track along, the catheter would not follow the bends through the RA and RV to reach the PV. The technique for positioning the intervention catheter involves the “full-length exchange” technique. To perform a full exchange, the initial flow directed end-hole catheter is repositioned in the PA as previously described. The guidewire used this time, however, will be much longer, 180 to 260 cm in length. This allows for the flow-directed catheter to be fully removed while maintaining the position of the guidewire across the PV. It is critical that an operator ALWAYS has control of the guidewire to control its position. Therefore the extra length of the wire provides sufficient wire outside of the patient to have the catheter completely out of the patient, but still have the wire in the patient in position. Once the flow-directed catheter is fully removed from the patient and off of the wire, then the interventional balloon catheter is fed over the wire, and through the introducer into the patient. It will track along the prepositioned wire until it can be centered across the PV. The catheter has radiopaque markers at each end of the interventional balloon to assist with positioning. After the balloon is correctly centered, the balloon is rapidly inflated with a 1:1 dilution of contrast media and saline. As the balloon is inflated the narrowed stenosis can often be seen as a “waist” in the balloon that will rapidly expand to the full balloon diameter once sufficient pressure is reached within the interventional balloon. The inflation is held for approximately 10 seconds to allow for the maximal separation of the valve leaflets. During inflation, all cardiac output ceases. The balloon is then rapidly deflated and the heart allowed to recover. Additional inflations are sometimes required.

The interventional catheter can then be removed and the flow-directed catheter replaced along the same wire into the main PA. The guidewire is removed and another pressure gradient is measured again and compared to the initial measure. A successful procedure will produce a significant decrease in the pressure gradient implying improved RV cardiac output. The introducer may be removed from the patient and any surgical cut-down repaired. The jugular vein is commonly ligated with no ill effect to the patient, but surgical closure of the venotomy may be performed.

Complications include ventricular arrhythmias during the procedure due to catheters in the RV, hemorrhage post-procedure, and infection at the venipuncture/venotomy site. The pressure gradient may rise slightly once anesthesia has been removed. Restenosis of the valve is an uncommon complication that may be addressed with a second BVP, medical management, open thoracic surgery with a patch graft and valvulotomy, or conduit placement to bypass the stenosis as required in bulldogs with an anomalous coronary artery².

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