



Proceedings of the April Meeting of the Veterinary Cardiovascular Society

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Wednesday 3rd April 2019, pre-BSAVA meeting
Hall 8b, International Convention Centre, Birmingham

8:30 -9:00	REGISTRATION	
9:00-09:45	Sudden cardiac death in dogs with atrial fibrillation <i>Kieran Borgeat, Langford School of Veterinary Sciences</i>	7
09:50-10:20	Cock-up! The perils of too much Viagra! <i>Chris Little, Barton Veterinary Hospital & Surgery, Canterbury, UK</i>	9
10:25-10:45	Optimal positioning of the CAM device in dogs <i>Dineke Rybak, Dick White Referrals</i>	11
10:45-11:15	COFFEE BREAK & SPONSORS' EXHIBITION	
11:15-12:05	Gene transfer therapy in veterinary cardiology: overview & early results of an ongoing pilot study <i>Sonya Gordon, Texas A&M</i>	17
12:10-13:00	Myocarditis in the UK <i>Kieran Borgeat, University of Bristol, Langford Vets</i>	18
13:00-14:00	LUNCH BREAK & SPONSORS' EXHIBITION	
14:00-14:20	Heart rhythm during episodes of collapse in Boxers with frequent or complex ventricular ectopy: a cross-sectional study <i>Marina Domingues, Dick White Referrals</i>	22
14:25-14:50	Can VHS predict EPIC echo inclusion criteria? <i>Sonya Gordon, Texas A&M</i>	26
14:55-15:25	Intra-cardiac conversion of atrial fibrillation <i>Geoff Culshaw, Royal (Dick) School of Veterinary Studies, University of Edinburgh</i>	27
15:30-16:00	COFFEE BREAK & SPONSORS' EXHIBITION	
16:00-17:00	In hospital management of canine CHF: strategies & challenges <i>Sonya Gordon, Texas A&M</i>	30

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Kieran Borgeat BSc BVSc MVetMed CertVC MRCVS DipACVIM DipECVIM-CA (Cardiology)

Kieran graduated from the University of Bristol and worked in general practice for 6 years before undertaking a residency and Masters degree at the Royal Veterinary College. He is an ACVIM Diplomate and ECVIM-CA Diplomate in Cardiology and an RCVS Recognised Specialist. He also has both Bronze and Silver Swimming Certificates (1994). He has worked in private referral practice and academia, and is now service lead in Cardiology at Langford Vets, University of Bristol. He is predominantly clinical, with a particular interest in interventional procedures and a burgeoning interest in equine arrhythmias. Kieran is a past member of the VCS Committee and past-Chair of the ACVIM Cardiology Research Committee, and currently sits on the ECVIM-CA Cardiology Credentials Committee. He has four children, an angry cat, and an incredibly patient wife who allows him to occasionally cycle rather long distances. @kborgeat (twitter) & @cardio_vetbristol (Instagram)

Dr. Christopher J.L Little BVMS PhD DVC FRCVS

Chris qualified from the University of Glasgow way back in 1981. He spent part of his early career in academia where he developed interests in internal medicine, ear disease and especially in cardiology. He has published widely and is now a Fellow of the RCVS (Royal College of Veterinary Surgeons). Chris gained the RCVS Diploma in Veterinary Cardiology in 2001. Chris has owned many pets: cats, dogs and small furries such as guinea-pigs. He currently has two dogs; a Lurcher called Tallulah and a scruffy cross-bred Terrier called Jemima.

Geoff Culshaw BVMS DVC MRCVS

Geoff graduated from Glasgow Vet School in 1994. After 11 years in general practice, he joined the Royal (Dick) School of Veterinary Studies in 2005, obtaining the RCVS Diploma in Veterinary Cardiology (2008) and specialist status (2011). He is currently Senior Lecturer in Small Animal Cardiopulmonary Medicine, and a Clinical Research Associate of The Roslin Institute

Geoff's interests include cardiovascular-renal interactions in health and disease, accessory pathways in cats and dogs, and the molecular basis to canine MMVD. In 2018, he completed a PhD at the Queen's Medical Research Institute, investigating endothelin-1 in renal salt handling in early Type 1 diabetes mellitus. This has led to post-doctoral research on localising and targeting inappropriate renal sodium transport to restore circadian regulation of blood pressure in Type 1 diabetes.

Dineke Rybak – van der Veen MRCVS

I grew up in a small town in the Netherlands with my family of eight. In January 2016 I graduated from the University of Utrecht and started working as a small animal vet in a mixed practice in the Netherlands. After having worked there for about 14 months I decided it was time for the next step and switched to a busy small animal practice in the UK. When I got the opportunity to start a small animal rotating internship at Dick White Referrals, I grabbed it with both hands. During the internship my interest in cardiology only grew, and I got the opportunity to visit several universities and congresses, such as ECVIM and the VCS autumn meeting, and set up a research project, which I will be presenting here. After this very educational and interesting year, I am looking forward to start a future internship or residency in cardiology while in the meantime working as a locum vet across the UK.

Dr. Sonya Gordon BSc DVM DVSc DACVIM (Cardiology)

Dr. Sonya Gordon is board certified in cardiology by the American College of Veterinary Internal Medicine and is a Professor of Cardiology at Texas A&M University College of Veterinary Medicine and Biomedical Science where she had been on faculty since 1998. She teaches in all years of the DVM program and is routinely an invited speaker at local, national and international veterinary meetings. Dr. Gordon practices medicine 30%-50% of the time, which facilitates her research interests that are realized in large part through involvement in multicenter collaborative clinical trials and collaborative translational research. These opportunities, coupled with her involvement in multicenter international studies, have provided her with a global perspective with respect to veterinary cardiology. She has published numerous manuscripts and book chapters and co-authored one practical small animal clinical cardiology book entitled *The ABCDs of Small Animal Cardiology*. Dr. Gordon considers her home College Station, Texas where she shares her life with her husband, 4 dogs and 2 cats.

Marina Domingues MRCVS

Marina graduated from Lusófona University in Lisbon, Portugal. After graduating, she moved to the UK to undergo an internal medicine scholarship at the Liverpool Small Animal Teaching Hospital. Posteriorly, she completed a rotating internship at Dick White Referrals. Since then, Marina has been working in first opinion small animal hospitals across the UK. Marina enjoys all aspects of internal medicine and cardiology, aspiring to one day specialise in internal medicine.

Sudden Cardiac Death in Atrial Fibrillation

Kieran Borgeat
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Introduction

The Framingham Heart Study has evaluated cardiovascular disease in the residents of Framingham, Massachusetts since 1948. In the original cohort, 5,209 adults between age 30 and 62 at the time were enrolled and followed longitudinally (the first of five groups of study participants to date). Atrial fibrillation (AF) developed in 621 individuals over a 40-year follow-up period. When other confounding variables were controlled for, AF was associated with an increased risk of death; the first time that this had been proven.¹ This was recently verified in a meta-analysis that featured almost 600,000 patients with AF in a population of over 9 million patients.²

In this systematic review,² 7 studies evaluating the effect of AF on the risk of sudden cardiac death (SCD) were eligible for meta-analysis; the relative risk of SCD was 1.88 compared to people without AF. From the reviewed studies, prevalence of SCD in humans with AF was reported at 0.6-28.9%.

In veterinary patients, SCD is anecdotally reported in dogs with AF, but no studies specifically reporting prevalence or investigating risk factors have been published. In one recent publication reporting the effect of heart rate on survival in dogs with AF,³ SCD was reported in 4/21 (19%) dogs that had died, but further analysis was prohibited by a low event rate. We sought to identify a prevalence of SCD in a (relatively) large population of dogs from multiple centres, and to try to identify measurable risk factors based on signalment, ECG findings, echocardiographic measurements or Holter variables.

The Study

Ethical approval was sought and gained from the University of Bristol and the Royal Veterinary College (VIN/18/054 and SR2019-0016 respectively). Retrospective analysis of computerised patient records was undertaken at seven referral centres in the UK: Langford Vets (University of Bristol), The Royal Veterinary College, Lumbry Park Veterinary Specialists, HeartVets, Pride Veterinary Centre (University of Nottingham), Highcroft Veterinary Referrals and Southern Counties Veterinary Specialists.

Data was collected on dogs diagnosed with atrial fibrillation. To be included in the study the following data had to be available: patient signalment and a basic history, standard 2D echocardiographic measurements, 24h Holter ECG analysis and some outcome data (date of

last contact and alive/dead status). Where relevant, circumstances and cause of death were recorded.

Death was classified as SCD if the dog had died spontaneously (not euthanised) without evidence of new or worsening clinical signs over the preceding 24-hours and no other outward cause evident (for example, road traffic accident or suspected rodenticide toxicity would not be classified as SCD).

Data was stored in Microsoft Excel and then transferred to IBM SPSS 24 for Mac for analysis. Descriptive statistics were calculated, specifically to report the prevalence of SCD. Survival was analysed using a Cox proportional hazards method, specifically to look for factors associated with SCD.

Results

Data from 142 dogs with atrial fibrillation that were eligible for inclusion in the study was recorded. Results of data analysis will be discussed and risk factors considered and compared with the literature in humans.

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3. Pedro B *et al* (2018). Retrospective evaluation of the effect of heart rate on survival in dogs with atrial fibrillation. *Journal of Veterinary Internal Medicine* **32** 86-92

Cock-up! The Perils of Too Much Viagra

Dr. Christopher J.L Little
Barton Veterinary Hospital & Surgery, Canterbury, UK

Maureen was a ten year old Jack Russell Terrier entire bitch, She had an unremarkable previous history. She was referred to me with a history of dullness, lethargy, anorexia, laboured breathing , exercise intolerance and rapid weight loss which had been present for more than two weeks. The primary vets had identified heart murmurs, mild cardiomegaly, mild cardiomegaly and abdominal enlargement, Treatment with furosemide and benazepril had given disappointing results; the owner reported severe polydipsia but no clinical improvement.

Clinical examination of this dog revealed dullness and a very quiet demeanour. Rectal temperature was 36.5°C and the dog's skin and ears felt cold to the touch. Respiratory rate was normal, 28 brpm. Breathing was slightly laboured and there seemed to be some adventitious sounds during expiration. Coughing was absent and there was no hyperpnoea. Percussion resonance was normal. Heart rate was fast,168bpm and regular. Pulses were weak, virtually impalpable. A heart murmur was auscultated: grade III / VI left hand side, grade IV / VI on the right side of the chest. The abdomen was tense but a fluid thrill was not detected and no other abnormalities were recorded. Rectal examination revealed scant but very dark faeces.

Haematology from Maureen was unremarkable with no anaemia and a normal differential white cell count.

Clinical Biochemistry from the dog showed slightly elevated liver enzymes, obvious (but mild) hypoproteinaemia, and mild azotaemia. Electrolyte abnormalities were not identified.

A urine sample from Maureen was rather dilute (USG 1.016) but otherwise normal.

Echocardiography was interesting: The right heart was enlarged with flattening of the interventricular septum. Paradoxical septal motion was present. The right atrium was dilated. Tricuspid regurgitation was present with rapid flow rate from the ventricle into the atrium. No pathology of the pulmonic valve or right ventricular outflow tract was found. The chambers of the left heart were small. The liver was enlarged. The caudal vena cava was dilated and as it's was noted.

A working diagnosis of right-sided (congestive) heart failure due to tricuspid valve incompetence and pulmonary hypertension was made. High dose sildenafil therapy with additional pimobendan was initiated. That evening Maureen ate for the first time in over two weeks. She seemed brighter.

Overnight Maureen's respiratory rate increased progressively. By the early morning she was tachypnoeic, more than 120brpm, She was coughing frequently and markedly dyspnoeic. The chest sounded very crackly.

Treatment with vigorous diuresis and oxygen therapy was given. Sildenafil doses were reduced. Intravenous pimobendan was given. In spite of these measures Maureen died in extremis. Terminally a large volume of pink clear fluid poured from the dog's airway as she died, attesting to the presence of severe pulmonary oedema.

In this case aggressive treatment of pulmonary hypertension caused left-sided heart failure by unloading the right heart and causing overwhelming consequence for the lungs and left heart. This has taught me to carefully consider the contraindications to sildenafil therapy and to proceed cautiously in the use of this drug.

Pilot Study Investigating Optimal Positioning of a Novel Ambulatory ECG Device

Dineke Rybak - van der Veen MRCVS
Ruth Willis BVM&S DVC MRCVS
Dick White Referrals, Newmarket, UK

Introduction

Bardy Diagnostics (BardyDX) has developed a novel device for ambulatory electrocardiogram (ECG) monitoring in humans, called the Carnation Ambulatory Monitor (CAM) device. The CAM device is a patch monitor that in adult human patients is attached over the sternum and, in pediatric patients, placement on the dorsum may also yield a diagnostic recording.

The device continuously records an ECG trace and there is an event button that can be pressed if symptoms occur. It records two simultaneous ECG leads and comes in 2 versions - one that records for 24 hours and one that records for up to 7 days. This device has been marketed since 2017 and as the device is small, lightweight, comfortable and can be worn whilst showering, it has generally been well accepted by human patients. The CAM device and analysis software was specifically designed to improve P wave detection, as previous patch devices sometimes failed to show the P wave clearly making arrhythmia characterization challenging.

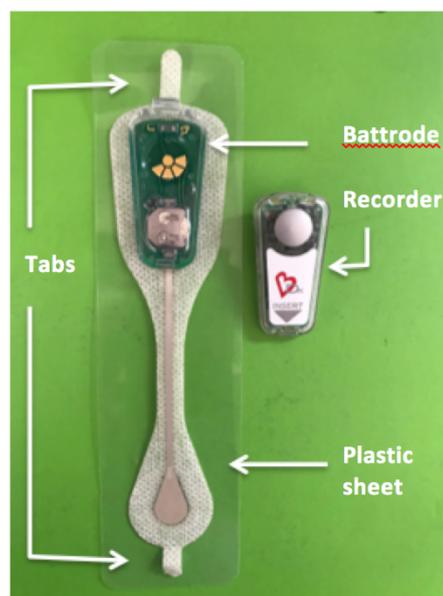


Figure 1. CAM device prior to assembly and activation. The adhesive battrode section is 14cm long x 3.5cm wide and the assembled device weighs 15g

Because the CAM is a relatively small device with a novel shape, it is potentially suitable for use in dogs and cats. The aim of this pilot study was to evaluate whether the CAM device could be used to obtain a diagnostic resting and ambulatory ECG trace in dogs and also to find the optimal positioning of the device on the dog's thorax.

Materials and Methods

Ten apparently healthy dogs of varying chest conformations were recruited. A separate CAM device was assigned to each dog. Each dog was carefully clipped in 4 positions; the left side of the chest (position 1), the right side on the chest (position 2), over the sternum (position 3) and dorsally between the shoulder blades (position 4). The skin was cleaned with alcohol drenched swabs and then dried thoroughly prior to application of the device. The CAM was attached and activated in accordance with the manufacturers recommendations. In some small dogs the device was shortened by creating a small fold in the long section of the adhesive battrode part.

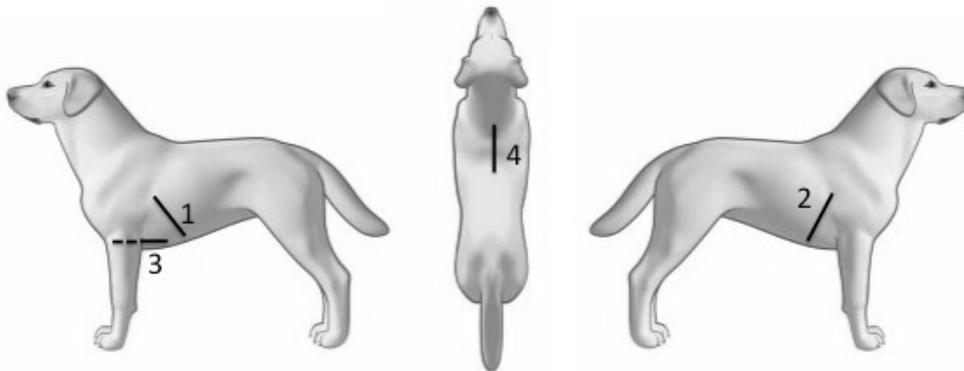


Figure 2. The four positions used to obtain recordings. 1 – left lateral thorax; 2 – right lateral thorax; 3 – over sternum; 4 – over dorsum.



Figure 3. Device attached in position 1 (left lateral thorax)

Whilst the device was in position 1, a standard six lead ECG was recorded with dog in right lateral recumbency for 5 minutes to allow comparison of the CAM trace with a standard ECG recording. The CAM device was then secured in place using cohesive bandage and the dog taken outside for 5 minutes of lead exercise. After this the device was gently removed and replaced in position 2 followed by 5 minutes of lead exercise. This was repeated for positions 3 and 4.

During analysis a section of the trace with clear P-QRS-T complexes and minimal baseline artifact was selected and the amplitude of 5 consecutive P and R waves was measured. The failure rate was defined as the number of P or R waves that were undetectable and therefore unable to be read in this section of the ECG trace. The best quality trace was defined as the position with the highest mean amplitude of both P and R waves. This trace was then compared to the trace of lead II of the standard resting 6 lead ECG recording.

Results

For the left lateral position the failure rate was 3%. For the right lateral position the failure rate was 5%. For the sternal position it was 30%. The dorsal position had a failure rate of 42%. The mean amplitude of the P waves with the CAM in position 1 over all dogs was 0.40 ± 0.35 mV, in position 2 the overall mean was 0.54 ± 0.30 mV, in position 3 the overall mean was 0.41 ± 0.21 mV and finally the mean for position 4 was 0.32 ± 0.23 mV. The mean of the R waves in position 1 over all dogs was 4.10 ± 2.22 , in position 2 it was 3.29 ± 1.82 mV, in position 3 2.95 ± 1.87 mV and in position 4 the overall mean for all R waves was 1.80 ± 0.54 mV. As the mean amplitude of the R wave was greatest with the CAM device in position 1, this was selected as the best position to compare the variance to lead II of a standard 6 lead ECG. For three dogs Levene's test showed that the variances in the P waves for the ECG and CAM device in position 1 were not equal. For all other dogs the variances were equal. For the R waves, the variances for the ECG and CAM in position 1 were not equal for 4 dogs.

Discussion and conclusions

This study shows that the use of the CAM device is feasible in dogs and can yield a diagnostic resting and ambulatory ECG trace. Moreover, the software that comes with the device is capable of detecting canine P-QRS complexes. The best position for placement of the device on the dogs chest is diagonally on the left side, but the same positioning on the right side of the chest will also yield a diagnostic trace. The trace quality is dependent on the preparation of the skin, so patients need to be carefully clipped and their skin prepared in accordance with the manufacturer's instructions. In our study the device was easy to apply and well tolerated by the dogs, although the duration of recording was short.

Future studies could involve the comparison of the CAM to other types of ambulatory ECG (Holter) monitor in dogs to assess arrhythmia detection. It would also be interesting to test the device on cats, as the size and possibility to decrease the length of the device would potentially make it more comfortable to wear than a Holter monitor.

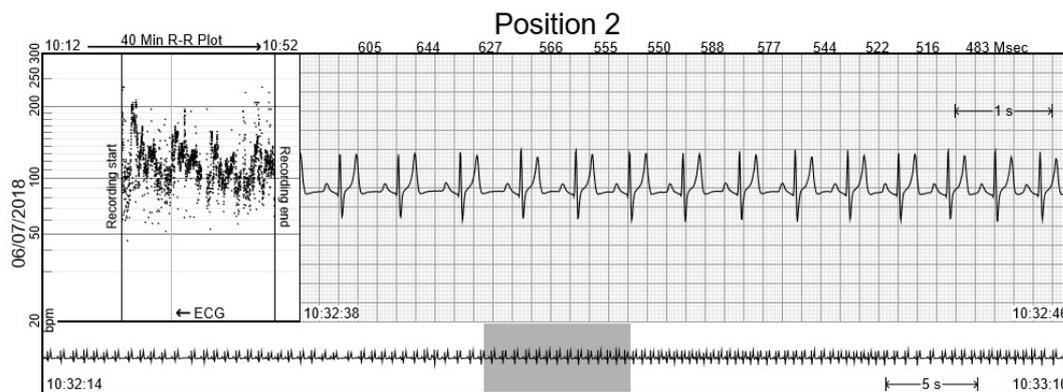
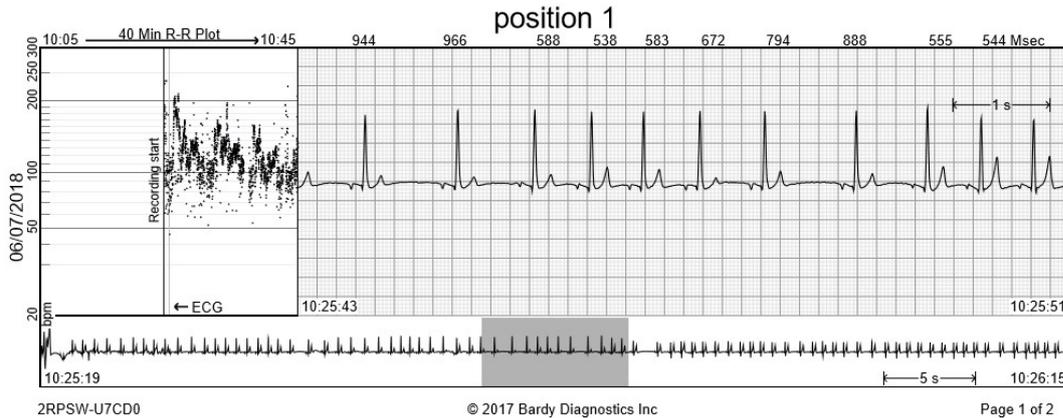


Figure 4. Example of trace of ECG from the CAM device in position 1 (left lateral thorax) and position 2 (right lateral thorax)

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Conflict of interest

Whilst this study was supported by Bardy diagnostics the authors have no financial interest in the company and have not received any payment.

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Gene transfer therapy in veterinary cardiology: overview & early results of an ongoing pilot study

Dr Sonya Gordon

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Established dilated cardiomyopathy (DCM) is a lethal disease in humans. When congestive heart failure (CHF) develops, the annual mortality is approximately 50%. Other than supportive therapy, there is no specific treatment for established DCM in humans. Recently glucagon like peptide – 1 (GLP-1) has been found to have cardioprotective effects independent of those attributable to tight glycemic control. In addition, ultrasound targeted microbubble destruction (UTMD) is a minimally invasive method to direct gene or protein therapy to the heart or pancreas in vivo. Preliminary data in rats with CHF secondary to Adriamycin induced cardiomyopathy has demonstrated that a single treatment of ultrasound targeted microbubble destruction (UTMD) delivery of GLP-1 gene plus a nucleus localizing signal (NLS) using a plasmid vector leads to overexpression of transgenic GLP-1NLS in the nuclei of rat cardiomyocytes and evidence that transfected cells underwent robust proliferation leading to myocardial regeneration that lead to reversal of established adriamycin cardiomyopathy.

Our pilot study will investigate if GLP-1NLS gene myocardial nuclear delivery via UTMB can reverse idiopathic DCM in Doberman pinschers. Inclusion criteria include Doberman pinschers with CHF secondary to DCM that are well controlled on optimal medical management (furosemide, pimobendan, +/-ACEi, +/-spironolactone, +/- sotalol, +/- mexilitine) and are in predominantly sinus rhythm. A second aim is to investigate the molecular signaling pathway responsible for proliferation of adult cardiac muscle cells induced by GLP-1NLS.

Myocarditis in the UK: A Case Series From a Non-Believer...

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Introduction

Although myocarditis is a differential diagnosis commonly brought into play for challenging cases of myocardial disease in dogs and cats, there are few convincing case reports of myocarditis in patients originating in the United Kingdom, and a lack of histopathological review for many presumed cases identified in clinics. *Trypanosoma cruzi* associated myocarditis is well-recognised in dogs in the Southern United States, and other protozoal agents such as *Toxoplasma*, *Neospora* and *Leishmania* have been implicated in European cases. Bacterial agents have also been implicated, such as *Borrelia burgdorferi* or *Bartonella spp.* Viral agents (*Parvovirus* or *Distemper*) and toxins (especially adder envenomation) are also possible causes.

Three years ago, I was sceptical that myocarditis occurred in dogs in the UK, outside of the occasional case with an adder bite or perhaps a “post-viral” dog with dilated cardiomyopathy (DCM) phenotype. Since then, our team has seen a number of dogs with severe inflammatory myocardial disease, confirmed on histopathology. In this lecture, we shall briefly review these cases and pathological findings, and consider the relevant background literature in addition to reviews from human medicine.

Summary and interesting case points

A table on the following page summarises the nine cases of myocarditis described; one cat and eight dogs. Aside from one dog with intracellular *Leptospira* bacteria and another with a recent history of an adder bite (witnessed by the owner), the cases do not have a definitive diagnosis (although some tests are pending at the time of writing). However, 3/8 dogs were diagnosed with granulomatous myocarditis (cases 6, 8 and 9), similar to cardiac sarcoidosis in humans, where immunosuppressive or anti-metabolite treatment may be helpful if a diagnosis is made antemortem. Perhaps we are missing a syndrome of disease in veterinary patients because of a lack of endomyocardial biopsy or cardiac MRI? Notably, histopathology of these cases suggested chronicity, but their clinical signs were apparently acute or subacute in nature, presumably owing to the onset of a haemodynamically significant arrhythmia. Should we consider endomyocardial biopsy in dogs with acute signs and arrhythmias? Obviously, this decision would be accompanied by risks of anaesthesia and the procedure itself in a patient that could well be clinically unstable.

ID	Species Age	Clinical signs	ECG findings	Echo findings	cTnl (ng/mL)	Histopathology comments
1	Dog; ME 16 wks Dalmatian	Collapse Pulmonary oedema	VT, ST elevation, junctional tachycardia	Systolic dysfunction, regional hypokinesis	> 50	Lymphocytic infiltrates, replacement fibrosis, spirochaetes within cardiomyocytes (confirmed Lepto)
2	Dog; FN 8 years French Bulldog	Collapse	Sinus arrest, atrial flutter, AV block, SVT	Unremarkable, possibly thickened tricuspid valve	0.49	Lymphoplasmacytic inflammation, myocardial necrosis including of both nodes, extensive fibrosis suggesting chronicity
3	Cat; FE 17 wks DSH	Pulmonary oedema	ST segment elevation, VPCs	Patchy appearing LV myocardium, LA dilation	120	Multifocal mineralising and necrotising myocarditis
4	Dog; ME 3 years Cocker spaniel	Adder bite, lethargy	Junctional tachycardia with occasional VPCs	Hyperechoic, thick myocardium, small PE	> 50	Acute, necrotising myocarditis
5	Dog; MN 7 years Lurcher	Dyspnoea, lethargy, cough 24h	VT, sinus with aberrant conduction	Systolic dysfunction, LA dilation	> 50	PENDING
6	Dog; FE 1 year Golden Retriever	Sudden death on arrival	Not available; presumed SVT	Not available	Not available	Granulomatous myocarditis, replacement fibrosis
7	Dog; FE 1 year Border collie	Weight loss, submandibular mass	First and second degree AV block	Mass in left atrium arising from the interatrial septum, systolic dysfunction	0.2	Severe, sub-acute, diffuse pyogranulomatous myocarditis and vasculitis with vacuolar degeneration and necrosis. Also LNs distantly involved. ZN neg.
8	Dog; FN 9 years Springer spaniel	Pulmonary oedema, lethargy	VT, first degree AV block, sinus with aberrant conduction	Systolic dysfunction, LA dilation	18.1	Granulomatous subendocardial LV myocarditis with necrosis and replacement fibrosis. Hepatic LN also granulomatous inflammation.
9	Dog; FN 9 years Samoyed	Pulmonary oedema, syncope	VPCs	Systolic dysfunction, LA dilation	24.6	Granulomatous myocarditis with replacement fibrosis

One dog (case 7) had systemic pyogranulomatous inflammation (ZN stain negative), involving multiple lymph nodes, a salivary gland and the spleen – here, the heart was considered to be “collateral damage” rather than a primary myocarditis. This may fit with *Borrelia* or *Bartonella* infection, or systemic sarcoidosis, in which case biopsy and testing of affected peripheral tissue may have been enough to provide a working diagnosis. On echocardiography, this case appeared to have one discrete mass, arising from the interatrial septum, but in reality the myocardium was more diffusely involved, as were the walls of the great vessels.

The feline case (case 3) was 12 weeks old and had mineralised foci throughout the myocardium, presumably a reflection of a previous insult that caused extensive necrosis; the most likely differential at this age is feline infectious enteritis, but the typical lymphocytic infiltrate and intracellular inclusion bodies of a viral infection were lacking.

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Heart rhythm during episodes of collapse in Boxers with frequent or complex ventricular ectopy: a cross-sectional study

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Intermittent collapse is a common presenting complaint in Boxer dogs. These collapse episodes are often attributed to ventricular tachycardia (VT), particularly in Boxers with frequent and complex ventricular ectopy however this has rarely been documented. Previous studies have shown that Boxers with frequent ventricular ectopy may be bradycardic during collapse events with a change in heart rate and rhythm suggestive of a neurally mediated event. The possibility of both brady- and tachyarrhythmias during collapse events highlights the challenge faced when selecting anti-arrhythmic treatment. An additional complicating factor is that collapse as defined by owners may not fulfil the medical definition - a sudden loss of postural tone that is not necessarily associated with loss of consciousness.

The main aim of the present study was to describe the heart rate and rhythm of Boxer dogs during episodes of collapse using ambulatory electrocardiography (AECG). Additionally, the predictive value of the presence of frequent or complex ventricular ectopy for collapse associated with VT or changes suggestive of a neurally mediated event was also investigated. Our hypothesis was that arrhythmias other than VT may be seen in association with episodes of collapse in Boxer dogs. We also hypothesized that the presence of frequent or complex ectopy on AECG may not be predictive of collapse associated with VT.

Materials and Methods:

From a database containing 3662 AECG recordings from dogs in the UK and Ireland obtained between 2005 - 2014, a total of 659 recordings and associated reports belonging to 429 Boxer dogs referred for investigation of suspected cardiac disease were reviewed. Information regarding signalment as well as the frequency and complexity of ventricular ectopy was obtained from these recordings. Frequent ectopy was defined as more than 50 ventricular beats (VPCs) during the recording or per 24 hour period, and complex ectopy defined as multiple consecutive VPCs. The recordings without frequent ventricular ectopy or complex ectopy, were included in Group 1. Those in which frequent ventricular ectopy or at least one example of complex ectopy was documented were included in Group 2. In those recordings in which collapse was reported (usually by the dog's owner), the minimum, mean, maximum heart rate as well as the number of collapse episodes during the recording period were also documented. Furthermore, positive predictive values were calculated with the aim of investigating whether the presence of frequent or complex ventricular ectopy could predict heart rhythm during episodes of collapse.

Results:

Of the 659 AECG recordings reviewed, 250 (38%) recordings from 171 dogs were included in Group 1 and 409 (62%) recordings from 286 dogs included in Group 2. For all 429 dogs, the

median age of the dogs at time of recording was 6 years (range 0.1-14 years), and the proportion of males was 58 % (n = 250; 95 % CI 53-63 %, P <0.001). A median ventricular beat count of 4 (range 0 – 46) was observed in Group 1 recordings and a median of 796 ventricular beats (range 2 – 337,250) was documented in Group 2 recordings. Dogs in Group 2 were significantly older than dogs in Group 1 (median age of 7 years and 5 years respectively; P = 0.005). Additionally, there were a higher proportion of male dogs in Group 2 (65% versus 47%; P = 0.02).

A total of 90 collapse events were documented in 72 AECGs from 68 dogs, comprising 33 events (from 30 dogs) in Group 1 and 58 events (from 38 dogs) in Group 2. In group 1, sinus rhythm was documented during 19 collapse events, changes suggestive of neurally mediated collapse during 13 and persistent atrial fibrillation during 1. In group 2, sinus rhythm was observed in association with 37 collapse events, changes suggestive of neurally mediated collapse with 14, VT with 6 and AF with 1. Furthermore, five dogs in group 2 in which the AECG documented changes suggestive of neurally mediated collapse also showed the concomitant presence of AF, either as a permanent arrhythmia (3 dogs) or paroxysmal arrhythmia (2 dogs).

The presence of frequent or complex ventricular ectopy was a poor predictor of VT associated collapse and was more likely to predict neurally mediated collapse in this population of Boxer dogs. However the importance of ventricular tachycardia cannot be discounted as one episode of VT collapse was a terminal rhythm.

Discussion and conclusions:

In our population of Boxer dogs, a high prevalence of collapse episodes and also frequent and/or complex ventricular ectopy on AECG was observed, similar to what has been reported previously in the veterinary literature. Our findings suggest that arrhythmias other than VT may be observed around the time of collapse episodes in Boxer dogs with frequent and complex ventricular ectopy. Sinus rhythm was the most common collapse rhythm observed in our population and, as this is generally considered to be a haemodynamically stable rhythm, non-cardiac illness was suspected to be the cause of the reported collapse episodes. The lack of further clinical information about the dogs constitutes an important limitation of this study but also reflects the population of dogs encountered in clinical practice where dogs with both cardiac and systemic disease present with collapse and ventricular ectopy. This study also highlights that collapse as defined by the owners does not always fulfil the medical definition and is likely to encompass a wide spectrum of presentations.

This study also found that changes suggestive of neurally mediated collapse can often be observed in Boxer dogs similar to what has been previously reported. In some of these dogs, the development of AF was observed after the suspected neurally mediated event. Furthermore, in some dogs with evidence of permanent AF on AECG, changes of heart rhythm suggestive of a neurally mediated response appeared to occur around the time of the collapse event.

The findings of the present study emphasize the challenge of empirical selection of anti-arrhythmic treatment, especially as beta adrenergic antagonist medication may increase the

frequency of neurally mediated events. Further studies are necessary to establish the best treatment approach in Boxer dogs presenting with collapse and frequent ventricular ectopy.

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Can VHS predict EPIC echo inclusion criteria?

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Myxomatous mitral valve disease (MMVD) is the most common cardiovascular disease in the dog and can lead to progressive cardiac chamber enlargement and resultant congestive heart failure (CHF) in approximately one third of this population. Cardiomegaly secondary to MMVD is a known risk factor for development of CHF and the EPIC Trial reported that pimobendan significantly delayed the time to onset of CHF in these dogs. In addition, the new ACVIM MMVD consensus statement, as presented, has made a strong recommendation for initiation of pimobendan in dogs with Stage B2 MMVD; where stage B2 is defined as the EPIC echocardiographic inclusion criteria. The EPIC Trial had three cardiac size inclusion criteria, two echocardiographic (LVIDDN ≥ 1.7 and LA:Ao ratio [2D Swedish] ≥ 1.6) and one radiographic (VHS > 10.5). However, echocardiography is not always readily available and therefore there is interest in how to identify dogs with Stage B2 MMVD in the absence of echocardiography. Factors that may be useful include; murmur grade, VHS, and breed.

Several reports and publications have suggested a VHS ≥ 11.5 may be predictive of EPIC echocardiographic inclusion criteria (now ACVIM Stage B2) in dogs with MMVD, however this cutoff is based on small sample sizes, or was made based on inferences from other publications. This session will review data that was presented in part as an abstract at ECVIM in 2017. The original study was retrospective and sought to identify a VHS cutoff, in a large cohort of dogs with MMVD (N $\cong 800$), with a high specificity and acceptable sensitivity that can be used to identify dogs that meet or exceed the EPIC echocardiographic inclusion criteria. Utility of the new VLAS method for the assessment left atrial enlargement has been recently published. This novel objective radiographic measurement and has the potential to improve overall accuracy of radiographic prediction of EPIC echocardiographic inclusion criteria and was therefore performed on radiographs from the original study and included in the reanalysis of the original study data set. This session will review some of key results.

Better In Than Out: Transvenous Over Transthoracic Electrical Cardioversion Of Atrial Fibrillation In The Dog

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Atrial fibrillation (AF) consists of unco-ordinated electrical activity across the atrial syncytium. Currently, the “mother rotor” and the “multiple wavelets” theories are competing as the inciting cause, but substrates consisting of atrial mass/volume, myocardial inflammation, myocardial fibrosis and disruption to ionic transport are necessary for maintenance of the fibrillatory state.

Once established, AF promotes further myocardial remodelling that consolidates the substrate, such that “AF begets AF”¹. Where underlying cardiac disease, and, particularly, atrial enlargement are not present (lone AF), individuals can maintain a degree of appropriate rate control through fluctuations in autonomic input into the AV node. Despite this, atrioventricular synchrony, which contributes to ~20% of cardiac output, is lost and alternative physiological processes, such as increasing preload, are required to maintain stroke volume.

Management of AF is controversial in both human and companion animal cardiology. Options include no intervention, medical management of rate control, and cardioversion (pharmacological or electrical). There are pros and cons for all approaches. In general, decision-making on whether to cardiovert lone AF in dogs is based on factors such as whether or not an individual is demonstrating clinical signs (eg exercise intolerance) and whether the clinician subscribes to the belief that AF contributes to the development of cardiomyopathy. These may depend on the performance/working status of the dog, and its breed (eg Irish wolfhound).

This case concerns a three year, six month old non-working entire male Labrador retriever, which was presented to the R(D)SVS for investigation following two episodes of syncope. On presentation, the dog was bright, alert and responsive with no signs of cardiovascular compromise, 37.2kg, BCS 6/9. Auscultation identified an irregularly irregular rhythm (heart rate 140 beats/min, pulse rate 114/min) that was confirmed on ECG to be a narrow complex atrial fibrillation. BP (indirect, oscillometric) was 150/107 mmHg. Echocardiography identified reduced systolic function but no other significant structural disease. Chest radiography, full body CT, routine biochemistry and haematology, including total T4 and troponin I, were all similarly unremarkable. Twenty-four hour-Holter ECG was performed because the cause of syncope was not apparent, and there were concerns about the possibility of OAVRT. This confirmed sustained atrial fibrillation (including during pre-syncope), with no restoration of sinus rhythm or aberrant conduction.

In the absence of any other underlying cause for syncope, cardioversion was recommended and treatment with amiodarone initiated. One month later, standard transthoracic electrical cardioversion (TTEC) was attempted² but four electrical shocks at 30-100J (Lifepak 20e; Medtronic) failed to convert AF to sinus rhythm. Amiodarone was maintained afterwards, and, following the publication by Jung *et al.*³, transvenous electrical cardioversion (TVEC) was performed three weeks later. This time, the AF was successfully converted to sinus rhythm on the first electrical shock (30J). Ten minutes later, on removing the TVEC electrode catheters, an iatrogenic VPC initiated AF. The catheters were replaced and cardioversion repeated, which, again, successfully converted on the first delivery of 30J. Ten minutes later, the TVEC catheters were again removed, this time without a VPC, and sinus rhythm was maintained. To date, six months post procedure, the dog remains in sinus rhythm and free from clinical signs. Follow-up 24-hour Holter ECG has failed to identify evidence of an accessory pathway, although there are occasional interpolated VPCs. We have also performed TVEC on one other dog, which again cardioverted on the first shock (30J).

Protocol for transvenous electrical cardioversion

In TVEC, a voltage is applied across two separate electrode catheters, one placed in the right atrium, the other in the left branch of the main pulmonary artery. The main advantage of TVEC over TTEC is that electrical energy is delivered directly to the atria, independently of dog size (transthoracic impedance), meaning that less energy is required. The main disadvantages are that TVEC is more invasive, requiring surgery and fluoroscopy. However, using our experience in radiofrequency ablation in Labradors, and human Amplatzer ductal occluder deployment in German shepherds, we have adapted the protocol of Jung *et al.*³ so that both electrode catheters can be accurately placed from the same jugular vein, thus removing the need for femoral venous catheterisation. Catheter placement is fluoroscopically-guided but we have also used TOE as an additional imaging tool.

Both TVEC electrode catheters are 180 cm long and 7F in diameter, but require a 0.018" 260cm guidewire. Placement of the pulmonary artery TVEC electrode catheter is performed first, aided by passing it through a 7F Torquevue or Cooks delivery sheath that has been advanced into the pulmonary artery using a 150cm 0.035" guidewire. The sheath is then retracted while advancing the catheter as far along the pulmonary arterial tree as possible. Any part of the electrode which is estimated to be below the pulmonic valve is deliberately sheathed to reduce inadvertent delivery of energy to the ventricle. The second TVEC electrode catheter is then inserted through a 7F introducer sheath and advanced into the caudal vena cava so that the electrode lies entirely within the right atrium. A further modification is that a 5F temporary pacing catheter is inserted transvenously (lateral saphenous) into the right ventricle for bradycardia support, in case of sinus arrest after the electrical shock. Pre-placed transthoracic pads are in case ventricular fibrillation occurs, although this can also be managed through intraventricular defibrillation. A shock of 30J has been 100% successful on the first attempt thus far (n=3).

The authors emphasise that, as with all interventional procedures, close communication between interventionalists and anaesthetists is maintained at all times. We have an established crash plan that is rehearsed immediately before delivering the first shock, including confirming that **the synchronisation mode has been activated**, and that everyone is aware of their individual roles. Delivery of anaesthetic gas is suspended immediately before delivering the shock, the TOE probe is removed, and everyone must acknowledge they are not in contact with the dog or table.

Recovery in both cases has been unremarkable. One dog developed a seroma at the incision site as a minor complication.

We believe that TVEC is both feasible and effective in dogs with AF, and that the adaptations that we have made to the technique make it less invasive. It also offers the option of bradycardia support, and is not more technically demanding than standard cardiac interventions. It may also be effective in dogs in which TTEC has not been successful, although we have no reason to believe that TVEC offers a reduced rate of AF recurrence than TTEC.

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In hospital management of canine CHF: strategies & challenges

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Management of canine CHF in hospitalized patients is predominantly based on expert opinion and experience and is also dependent in large part on the type of hospital (24 hr care possible or not), equipment (oxygen cages, IV pumps, syringe pumps, telemetry), available medications (IV pimobendan etc.) and of course the owner's financial resources.

This interactive session will discuss, practical considerations for in hospital monitoring of dogs with CHF. An audience response live-polling system will be used to facilitate audience participation in the discussion.