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

Friday and Saturday 8\textsuperscript{th} and 9\textsuperscript{th} November 2013

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Programme Veterinary Cardiovascular Society meeting

BURLEIGH COURT, LOUGHBOROUGH
8th NOVEMBER 2013

8:30 - 9:00  REGISTRATION

9:00 - 9:40  Report of the KC/BVA project on heart screening
             Eva Pavelkova, University of Liverpool, Cheshire

9:50 - 10:30 Outcomes of the UK and Danish Cavalier heart screening schemes
               Simon Swift, North West Surgeons, Cheshire

10:30 - 11:00 COFFEE BREAK

11:00 - 11:40 Evaluation of mitral valve regurgitation using cardiac MRI
               Julia Sargent, Royal Veterinary College, London

11:50 - 12:30 ACVIM update
               Valentina Palermo, R(D)SVS, Edinburgh
               Susan Roberts, SLR Cardiology Referrals, West Yorkshire

12:30 - 13:30 LUNCH BREAK

13:00 - 14:20 Transoesophageal echocardiography in dogs
               Pedro Oliveira, Davies Veterinary Specialists Ltd, Herts

14:30 - 15:20 Transoesophageal echocardiography in humans
               Navroz Masani, Cardiff and Vale University Health Board, Cardiff

15:20 - 15:50 COFFEE BREAK

15:50 - 16:20 Validation/accreditation of echo skills
               Navroz Masani, Cardiff and Vale University Health Board, Cardiff

16:30 - 16:50 Case report: Complex congenital defect in a Bichon Frise
               Chris Fellows, Lakes Cardiology Services, Cumbria

17:00 - 18:30 AGM (and Breed-related conditions subcommittee)

17:00 - 18:00 Parallel session for Residents: Assessing a scientific paper
               Jon Wray, Dick White Referrals, Newmarket

From 19:00  SOCIAL DINNER
### Programme Veterinary Cardiovascular Society meeting

**BURLEIGH COURT, LOUGHBOROUGH**  
**9TH NOVEMBER 2013**

#### BREAKFAST

<table>
<thead>
<tr>
<th>Time</th>
<th>Topic</th>
<th>Speaker</th>
<th>Page</th>
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<tbody>
<tr>
<td>9:00-9:50</td>
<td>Pre-hypertrophic HCM: Review</td>
<td><strong>David Connolly, Royal Veterinary College, London</strong></td>
<td>8</td>
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<tr>
<td>10:00-10:50</td>
<td>CatScan: an epidemiological study of HCM in apparently healthy cats</td>
<td><strong>Rosie Payne, Royal Veterinary College, London</strong></td>
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#### COFFEE BREAK

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<th>Time</th>
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<tr>
<td>11:00-11:30</td>
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<th>Time</th>
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<th>Speaker</th>
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<tbody>
<tr>
<td>11:30-12:10</td>
<td>Spironolactone in cats</td>
<td><strong>Malcolm Cobb, Nottingham University, UK</strong></td>
<td>16-18</td>
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<tr>
<td>12:15-12:35</td>
<td>Case report: Different manifestations of congestive heart failure in three cats</td>
<td><strong>Jorge Prieto, University of Glasgow, Glasgow</strong></td>
<td>19</td>
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<td>12:40-13:00</td>
<td>Case report: PPDH and surgical complications in a young Maine Coon</td>
<td><strong>Hannah Geere, Royal (Dick) School of Veterinary Studies, Edinburgh</strong></td>
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<tr>
<td>13:00-14:00</td>
<td>ECVIM update</td>
<td><strong>Liva Vatne, Groruddalen Dyrelinikk, Oslo, Norway</strong></td>
<td>21-22</td>
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<td>14:00-14:40</td>
<td>ECVIM update</td>
<td><strong>Emily Dutton, North West Surgeons, Cheshire</strong></td>
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<td>14:45-15:25</td>
<td>Discussion of HCM screening</td>
<td>Open-floor discussion opened by <strong>Virginia Luis Fuentes, Royal Veterinary College, London</strong></td>
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#### COFFEE BREAK

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<th>Time</th>
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<td>16:00-17:00</td>
<td>Panel: Challenging ECGs</td>
<td><strong>Jo Dukes-McEwan, Mike Martin and Nuala Summerfield. Chair: Mark Patteson.</strong></td>
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17:00 **CLOSE**
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Speakers:

Eva Pavelkova MVDr MRCVS

Eva graduated from the Veterinary University of Brno, Czech Republic, in 2006. She then spent 3 years working in small animal practice in the UK. She completed general internship at a referral hospital in Prague, Czech Republic from 2009-2010 before moving back to the UK. She enrolled with the RCVS Certificate of Advanced Veterinary Practice (Cardiology) in 2011 and is currently working as a Cardiology Intern at the University of Liverpool. She got appointed by the Veterinary Cardiovascular Society to undertake a project on heart screening in dogs on behalf of the British Veterinary Association and the Kennel Club in May 2013. Apart from cardiology, her major veterinary interest is charity work for the Tibetan Volunteers for Animals (TVA) in Bylakuppe Tibetan Settlement in southern India.

Simon Swift MA VetMB CertSAC DipECVIM-CA (Cardiology) MRCVS

Simon Swift passed the certificate in small animal cardiology in 1990 shortly after graduating from Cambridge. He then worked in small animal practice in the Northwest of England before the call of cardiology became too strong. He returned to Liverpool in 2005 to complete his European Diploma joining Northwest Surgeons in 2009 where he remains. For the last 20 years he has advised the UK cavalier King Charles spaniel society on heart disease. He is currently an associate editor for JVIM.

Julia Sargent BVSc MRCVS

Julia graduated from Bristol Veterinary School in 2006. Following 3 years in small animal first opinion practice she worked as a cardiology intern at Southern Counties Veterinary Specialists, before completing the general internship at the Royal Veterinary College. In 2011 Julia took the position of Junior Clinical Training Scholar in Cardiology and began research regarding the application of cardiac magnetic resonance imaging to veterinary patients. She has continued this research in the role of cardiology resident and has enjoyed the opportunity to further develop interests in advanced imaging techniques, in addition to a particular passion for arrhythmia diagnosis and treatment.

Valentina Palermo DVM PhD MRCVS

Valentina graduated from the University of Milan in 2006. She then worked in private practice and in the Cardiology Department of the University of Milan, where she completed a PhD in Veterinary Clinical Sciences (curriculum Small Animal Cardiology) in 2009 discussing a thesis on Boxer Cardiomyopathy. In 2008 she worked as a clinical intern at the Veterinary Cardiorespiratory Centre in Kenilworth, UK. She joined the Hospital for Small Animals of the Royal (Dick) School of Veterinary Studies in November 2010 as a Boehringer-Ingelheim Senior Clinical Training Scholar in Cardiopulmonary Medicine.
Susan Roberts BVMS CertVC MRCVS

Sue is a Glasgow graduate with many years’ experience working in mixed and small animal first opinion practice in the North of England. Having achieved the RCVS CertVC in 2000 she is studying for the RCVS DVC on the alternative path under the supervision of Dr Jo Dukes McEwan whilst running a peripatetic diagnostic cardiology practice in West Yorkshire.

Pedro Oliveira DVM DipECVIM-CA (Cardiology) MRCVS

Pedro qualified from Porto University (Portugal) in 2005. From 2004 to 2006 he performed an internship and worked in the cardiology department of Clinica Veterinaria Gran Sasso in Milan (Italy). From 2006 to 2009 he worked in general practice and also performed referral cardiology services in Portugal. In 2009 he returned to Milan to enroll in a 3 year ECVIM-CA residency training program, specialty of cardiology, under the supervision of Claudio Bussadori, Dipl. ECVIM-Ca (Cardiology) and Oriol Domenech, Dipl. ECVIM-Ca (Cardiology) with the joint participation of the École Nationale Veterinaire de Lyon (Lyon, France) under the supervision of Prof. Jean-Luc Cadoré, Dipl. ECVIM-Ca (Internal Medicine). Since the beginning of 2011 he has performed interventional cardiology services in Porto (Portugal) and Barcelona (Spain). Pedro joined the Davies Veterinary Specialists Cardiology Service in October 2012.

Navroz Masani MBBS FRCP

Nav graduated from Guys Hospital Medical School in 1987. He trained in cardiology in London and Cardiff before undertaking an echocardiography fellowship in Boston, Massachusetts. He was appointed as consultant cardiologist and head of echocardiography at the University Hospital of Wales in 1999 and is currently Clinical Board Director of Specialist Services. He has been a council member, Chairman of Education and President of the British Society of Echocardiography. He is the director of the Cardiff Echocardiography course.

Chris Fellows BVMS CertVC MRCVS

Chris Fellows graduated from Glasgow University Veterinary School in 1990 and spent the next few years in private small animal practice near Leeds. He gained his Certificate in Veterinary Cardiology from practice in 1995, and shortly after spent 6 months teaching back at Glasgow whilst Chris Little was on sabbatical. After a further short period in Leeds he moved up to the Lake District where in 1998 he set up his own small animal practice in Ulverston. In 2010 he left small animal practice to set up Lakes Cardiology Services, an ambulatory cardio-respiratory service serving the North West from Rutland House Referrals in St.Helens up to Kendal in Cumbria. He is married with two children, one cat, one spaniel and one horse. His hobbies include fell running, cycling and flamenco guitar, but most definitely not horse riding.
Jon Wray  MRCVS

Jon is a Bristol graduate and after a period in mixed practice in Somerset returned to the University of Bristol to perform a residency in Small Animal Medicine. He then worked at Willows Referral Service to develop an internal medicine referral service before moving to the Animal Health Trust, Newmarket where he latterly became Head of the Internal Medicine Unit. After a return to Bristol as a Clinical Fellow in Cardiorespiratory Medicine, Jon took up a post as an Internal Medicine Specialist at Dick White Referrals, Newmarket where his time is divided between Internal Medicine, Cardiology and Interventional Radiology. He holds the RCVS Diploma and RCVS Specialist status in Small Animal Medicine (Internal Medicine), the RCVS Certificate in Cardiology and is enrolled to sit the RCVS Diploma in Veterinary Cardiology when his family lets him. He is previous Chief Examiner for the RCVS Certificate and Diploma in Small Animal Medicine and sits on the RCVS Medicine Board and is an Honorary Associate Professor of Small Animal Internal Medicine at the University of Nottingham. Jon’s clinical interests are all aspects of cardiology but especially pericardial disease, promoting the use of interventional radiology (particularly in airway and hepatic disease), and in interventional endoscopy. Outside work he mostly clears up after his children.

David Connolly  MRCVS

After qualifying from the Royal Veterinary College, David pursued a career in research with the Medical Research Council. Having obtained a PhD in molecular genetics, he continued research for three further years, investigating the molecular mechanism underlying early embryonic development. Following a period working for the PDSA in the Midlands, he returned to the RVC and completed a Residency in the Medicine Service. During this time, David obtained the RCVS Certificates in Small Animal Medicine and Cardiology, and subsequently the ECVIM Diploma in Cardiology in 2003. David is currently Head of the Clinical Cardiology Service at the Queen Mother Hospital for Animals.

Rosie Payne  BVetMed MRCVS

Rosie graduated from the Royal Veterinary College, London in 2008. After spending a year in small animal private practice she returned to the Royal Veterinary College to study for her PhD entitled “An epidemiological study of hypertrophic cardiomyopathy in cats” under the supervision of Professor Virginia Luis Fuentes and Doctor David Charles Brodbelt. She submitted her PhD thesis in July 2013 and immediately started her residency in cardiology at the Royal Veterinary College.
Jorge Prieto Ramos LdoVet MRCVS MRCVS

Jorge graduated from the Faculty of Veterinary Medicine of Zaragoza (Spain) in 2009. Following veterinary school he completed a rotating internship in a referral hospital in Barcelona and after that he worked in general practice. He has visited and worked in Universities in Nicaragua (2007) and Mexico (2009). Jorge spent 3 months doing an externship in the Veterinary Cardiorespiratory Centre, Kenilworth and 3 months as a Cardiology Intern at the Royal (Dick) School of Veterinary Studies in Edinburgh. In September 2012 he began a Residency in Cardiology under the European College of Veterinary Internal Medicine (ECVIM-CA) in the Small Animal Hospital of the University of Glasgow. Jorge is involved in studies regarding acquired heart disease in dogs and cats.

Hannah Geere  MRCVS

Hannah graduated from the R(D)SVS in 2006. During the undergraduate years, she undertook an intercalated Master of Science, by Research, in Virology. Upon graduation Hannah went into mixed practice in the Midlands, and began to develop a particular interest in Cardiology. In 2008 she moved into purely small animal practice, before returning back to the R(D)SVS to undertake a PhD in canine cardiac stem cells. In 2011 Hannah started the residency in cardiopulmonary medicine, again at the (RD)SVS, and is due to complete this in November 2014.

Liva Vatne  BVM&S CertVC

Liva Vatne graduated from the R(D)SVS, Edinburgh, in 2002. She worked in general practice in South East England for six years before returning to her home country, Norway. Liva obtained her RCVS certificate in veterinary cardiology in 2009. She currently works as a general practitioner and cardiologist in a first opinion and referral practice in Oslo.

Emily Dutton  BVM&S CertVC MRCVS

Emily graduated from Edinburgh University and worked in general practice for several years whilst obtaining the RCVS Certificate in Veterinary Cardiology in 2003. She has worked exclusively as a veterinary cardiologist in a multidisciplinary hospital since 2006 before joining the team at Northwest Surgeons in 2012. Emily is currently following a residency programme for the RCVS Diploma in Veterinary Cardiology with her research interest being brain-heart interactions. In her spare time, Emily is investigating dilated cardiomyopathy in Scottish deerhounds.
This project is focused on research into current breeding schemes for cardiac diseases in dogs in the UK and worldwide. This project was initiated in May 2013 on behalf of the British Veterinary Association and the Kennel Club, in an attempt to improve criteria for screening dogs for heart diseases prior to breeding in the UK.

The main goals of this project were to compile a record of current heart testing schemes in dogs worldwide and the criteria being used for cardiac assessment and to design new breeding schemes for congenital and acquired cardiac diseases in dogs of various breeds.

The canine breeds of interest were selected based on their popularity in the UK and their predisposition to congenital and acquired cardiac diseases. The following breeds and heart diseases were included in the research: Boxer (aortic stenosis, arrhythmogenic right ventricular cardiomyopathy), Bullterrier (aortic stenosis, mitral valve dysplasia), Newfoundland (aortic stenosis, pulmonic stenosis, dilated cardiomyopathy), French Bulldog (aortic stenosis), Rottweiler (aortic stenosis), English Bulldog (pulmonic stenosis), Labrador Retriever (tricuspid valve dysplasia), Golden Retriever (aortic stenosis), Great Dane (dilated cardiomyopathy), Irish Wolfhound (dilated cardiomyopathy), Scottish Deerhound (dilated cardiomyopathy), Doberman (dilated cardiomyopathy), Cavalier King Charles Spaniel (myxomatous degenerative valvular disease) and Dachshund (myxomatous degenerative valvular disease).

An online questionnaire was created and sent via VIN to veterinary surgeons involved in heart testing worldwide. The questions involved in the survey covered a wide range of data, including qualifications of veterinary surgeons performing heart screening, official and unofficial schemes in place for individual canine breeds, tests used for screening examinations in the canine breeds and cardiac diseases mentioned above and criteria used for their assessment.

A total of 100 respondents from 19 different countries took part in the survey. Most responses were received from the USA, UK, Spain, France and Italy. Most of the respondents had a further qualification in veterinary cardiology (ACVIM Cardiology Diplomate, ECVIM-CA (Cardiology) Diplomate, RCVS Cardiology Certificate Holder and RCVS Cardiology Diplomate). Additional information on heart testing schemes in Germany, Sweden and Italy were also obtained and analysed alongside the results from the online survey.

The results of the project will be presented in detail during the talk.
Degenerative mitral valve disease is the most common cardiac disease recognised in the dog accounting for 75-80% of all cardiac disease in dogs.\(^1\) It is most commonly seen in small breed dogs and is particularly prevalent in the cavalier King Charles spaniel (CKCS) with approximately 50% of CKCS having a murmur due to mitral regurgitation by 6 years of age.\(^2\)

An examination of the Swedish insurance database revealed that the claims incidence for heart disease in CKCS peaked at 6 years with a lag of 2-3 years before a peak due to claims for death due to heart disease.\(^3\) A survey by the Kennel Club in 2006 suggested that 43% of CKCS die from heart disease.\(^4\)

Several studies have suggested an inherited basis. Swenson et al\(^5\) graded CKCS parents in terms of loudness of murmur and age of onset and showed that the offspring from parents with a high parental score developed a louder murmur at a younger age than dogs bred from parents with a low parental score. In a more recent examination\(^6\) of the UK CKCS database, breeding from 4-5 year old CKCS showed that the presence of a murmur was highly inherited. The Lupa project identified 2 loci at CFA 13 and 14 as being associated with the disease.\(^7\) Unfortunately, an early analysis\(^8\) of the Swedish database did not identify a change in the incidence of murmurs in 6 year old CKCS.

The results of the Danish scheme were reported by Dr Olsen\(^9\) at AVCIM this year. A total of 997 CKCS were examined from 2002 to 2011 and each dog was evaluated 1-4 times with a total of 1380 examinations. Auscultation and echocardiography were performed to assess the severity of mitral regurgitation and the degree of mitral valve prolapse. The risk of having a murmur in 2010-2011 was compared to 2002-2003 using linear regression. In 2010-2011, dogs were estimated to have a 74% reduced risk of having mitral regurgitation than dogs in 2002-2003 if they were a product of the breeding program (\(p=0.0017\)). Dogs were defined as a product of the breeding program if both parents were approved by the breeding program. In conclusion, the Danish mandatory breeding program based on auscultation and echocardiography, significantly reduced the prevalence over the 8-10 year period.

In 1991, the UK CKCS Club with advice from Dr Darke set up a screening scheme for members with the advice to breed from dogs that developed murmurs later in life. This scheme was based on auscultation to document the presence or absence of a murmur and to make the scheme widely available both GP vets and “cardiologists” could certify. Following a seminar in 1996, this was refined to suggest that dogs should be tested annually and that an over 5 year list of dogs clear of a murmur should be published. Only dogs examined by a cardiologist would be included in this list. Dogs that developed a murmur after 5 years of age could be used for breeding. However, younger dogs could be bred if
both parents were free of a murmur at 5 years.

The database was derived from breed screening in cavalier King Charles spaniels in the UK from 1991 to 2010. A total of 16,887 examinations were performed on 8860 dogs. Multivariant models showed male dogs developed murmurs at a younger age than females (7.9 years v. 8.1 years) and cardiologists detected murmurs in younger dogs than GP vets (7.2 years v. 8.8 years). Comparing 4 quartile time periods, there was a significant increase in the median age at which 50% of cavaliers will be murmur free determined by GP vets (8.3 to 9.9 years, P 0.001) over 1991-1995 and 2006-2010. Although this trend was seen for cardiologists (7.0 to 7.4 years), it was not significant.

This study suggests the current UK breed program is having a significant impact increasing the age of onset of murmur consistent with DMVD in cavalier King Charles spaniels.

References

1. Das KM and Tashjian 1965 Chronic mitral valve disease in the dog Veterinary Medicine Small Animal Clinician 60 1209-1216
2. Darke PGG 1987 Valvular Incompetence in cavalier King Charles spaniels Veterinary Record 120 365-366
5. Swenson L et al 1996 Relationship between parental cardiac status in cavalier King Charles spaniels and prevalence and severity of chronic valvular disease in offspring Journal of American Veterinary Medical Association 2008 2009-2012
6. Lewis T et al 2011 Heritability of premature mitral valve disease in cavalier King Charles spaniels Veterinary Journal 188 73-76
7. Madsen MB et al 2011 Identification of 2 loci associated with development of myxomatous mitral valve disease in cavalier King Charles spaniels Journal of Heredity 102(S1) S62-S67
8. Lundin T and Kvart C 2010 Evaluation of the Swedish breeding program for cavalier King Charles spaniels Acta Veterinaria Scandinavica 52 54
9. Birkegaard AC et al 2013 Evaluation of the breeding program aiming at reducing the prevalence of myxomatous mitral valve disease in cavalier King Charles spaniels in Denmark Proceedings of ACVIM Forum
Mitral valve disease in humans is a relatively common acquired condition that chronically can lead to left ventricular (LV) dysfunction and left ventricular dilation. A thorough and prompt assessment of severity of the mitral valve (MV) lesion is imperative in order to evaluate the functional severity of the disease and assess the suitability of a patient for MV surgery. Conservative treatment is generally implemented in patients where degree of regurgitation is only mild, whereas surgery is reserved for cases where regurgitation is severe, there is evidence of left ventricular dilation or systolic dysfunction or the patient is symptomatic. In common with our veterinary patients, the recognised technique for assessment of patients with mitral regurgitation is with transthoracic echocardiography (TTE), which may be augmented with transoesophageal echocardiography and/or real-time 3 dimensional echocardiography where it has not been possible to obtain the optimal images through TTE alone.

Quantification of mitral regurgitation, hemodynamic effects and detailed imaging of MV morphology with the use of cardiac magnetic resonance imaging (CMRI) is well described in the human literature and has proven useful where values cannot be obtained by other non-invasive techniques, e.g. due to limited acoustic access or the presence of highly eccentric regurgitant jets. Cardiac catheterisation has previously been used where non-invasive methods have failed to provide the desired information, however CMRI has been shown to compare favourably to cardiac catheterisation for assessment of the magnitude of regurgitation and its influence on left ventricular volumes and systolic function.

MV regurgitation can be quantified using CMRI via application of velocity-encoded, phase contrast sequences. To provide the patient regurgitant volume, the acquired aortic outflow volume is subtracted from the left ventricular stroke volume. Left ventricular stroke volume is calculated through summation of multiple short-axis frames, using Simpsons rule. Regurgitant volume is divided by the left ventricular stroke volume to obtain the regurgitant fraction. In isolated valve regurgitation regurgitant volume and fraction may be derived from subtraction of the right ventricular stroke volume from the left ventricular stroke volume. Calculation of right ventricular stroke volume is impeded however, by extensive trabeculation of the right ventricle, which can lead to inaccuracies during measurement. Alternative methods have compared forward aortic flow to the left ventricular inflow volume, measured at the MV annulus.

CMRI has also been shown to be useful in the assessment of valve morphology and motion. Detailed imaging of the mitral valve leaflet and assessment for the presence of torn/ruptured chordae tendineae has significant implications when planning surgical repair.
TTE provides adequate information regarding the presence of mitral valve prolapsed however this is can be hampered, as previously discussed. TEE can provide further information, however this is often avoided due to its semi-invasive nature. CMRI is less invasive and has been demonstrated to show comparable ability to transoesophageal echocardiography to identify jet number, location, direction and presence of leaflet prolapse. It has been demonstrated, however, to have been less sensitive in the detection of torn chordae tendineae.

**Veterinary use of CMRI:**

Although CMRI is a technique that has been widely used in human patients in a variety of clinical settings from evaluation of ischemic heart disease to functional and structural cardiac abnormalities in human patients, experience using CMRI in the veterinary clinical field remains limited. Some additional information has been gleaned, however, from experimental cardiology research using animals as models for human disease. A limitation in veterinary patients is the requirement for general anaesthesia to provide necessary restraint for imaging in addition to lack of experience and skill with this technique. The former may be undesirable due to the potential to increase risk to patients in more advanced stages of MV disease. Additionally anaesthesia is known to induce changes in systolic function which may alter valve haemodynamics, leading to inaccurate measurement and quantification of MR severity. Three major sources of artefact in CMRI are respiratory motion, cardiac motion and extremes of blood flow, however these problems have now been largely overcome by the use of ECG gating, faster imaging sequences with more advanced machines and the use of breath-hold sequencing.

There is only very limited information regarding the clinical use of CMRI in dogs. There are currently no reports in the veterinary literature that describe the haemodynamic assessment of valvular heart disease using CMRI. One study describes the remodelling of right and left ventricular geometry in response to chronic MV regurgitation, however no further qualification or quantification of the MR was attempted. There has been recent interest in the application of CMRI in the differentiation of neoplastic and non-neoplastic causes of pericardial effusion, in addition to the use of CMRI to acquire further morphological understanding in complex congenital disease. Hockings et al validated the use of multislice cine gradient echo CMRI to measure cardiac output through comparison with a widely accepted thermodilution method. Basso et al performed post-mortem CMRI of formalin fixed hearts from Boxer dogs with arrhythmogenic right ventricular cardiomyopathy. It was found that replacement of right ventricular myocytes with fat or fibrofatty tissue, characteristic of the disease in both human patients and Boxers, was accurately identified with CMRI when confirmed on histopathological examinations.

CMRI has been shown to be a useful tool in further evaluating the severity of MR and reaching decisions regarding case management in human patients. Although currently mitral valve repair and replacement is rarely performed canine patients, accurate quantification of mitral valve regurgitation severity is likely to yield important information
regarding prognosis and staging of the disease process in addition to its use as a powerful research tool. CMRI has been shown to be applicable to animals in clinical and experimental veterinary research and is a promising modality and this may provide new method in which to evaluate an extremely common condition in small animal veterinary practice.

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References:


ACVIM update

Valentina Palermo,
Royal (Dick) School of Veterinary Studies, Edinburgh, UK
Susan Roberts,
SLR Cardiology Referrals, West Yorkshire, UK

Intense programme with Cardiology presentations in Scientific, Workshop and Interactive sessions: 8 in the pre-Congress Cardiology Specialty Symposium, 21 Oral Abstracts, 46 Poster Abstracts, 11 Cardiology Comprehensive Reviews, 10 Cardiology Research Focus, 8 in the Cardiology Research & Review, 4 in the Equine Cardiology Clinical Workshop plus Cardiology presentations in the pre-Congress American Board of Veterinary Practitioners and in the American Association of Equine Practitioners. Scientific sessions comprised: Research Focus (SOTA) presentations - experts present information from last 2 years, Comprehensive Review lectures (Fundamental principles or Applied medicine), Clinical workshops interactive, and problem based approach. Abstracts are published in the Journal of Veterinary Internal Medicine (JVIM May-June issue, vol. 27, pp604-756)) accessible online. Other proceedings are available via The Veterinary Information Network (VIN).

1) Pre Congress Symposium

OPEN HEART SURGERY:
Dr Massami Uechi (Nihon University) reported numerous successful open heart procedures under cardiopulmonary by-pass in small-breed dogs and cats.

Atrial or ventricular septal defects in cats can be repaired using e-PTFE (expanded polytetrafluoroethylene) grafts. Complete endocardial cushion defect was repaired with e-PTFE patches in 2 dogs which were alive and asymptomatic 5 and 8 years after the surgery.

11 dogs with valvular PS and 1 with subvalvular and valvular PS were treated surgically between 2006 and 2011. One patient died post-operatively and 1 developed suicide right ventricle. Open valvotomy, pulmonary valve commissurotomy, with or without biomembrane patch grafting under cardiopulmonary bypass were effecting in reducing the obstruction and reduce the pressure gradient to <50 mmHg in small dogs and cats with severe pulmonic stenosis. Open valvotomy may induce pulmonic regurgitation, so valve replacement may be required in such cases. Autologous prosthetic implants (valve conduit with the sinus of Valsalva) were developed and used to replace the pulmonary valves in a Beagle model. The molds were placed in the dorsal subcutaneous space of the dogs for 4 weeks until they were covered with autologous connective tissue and were then implanted to the main pulmonary arteries. Good results at 14 weeks post-implant.

Dr Leigh Griffiths (UC Davis) presented on Tricuspid Valve Dysplasia (TVD) raising more questions than answers. He gave a comprehensive review of canine tricuspid valve (TV) anatomy and pathology. Anterior and parietal leaflets move, septal leaflet has 3 chordae 7-
10mm long and hardly moves at all. **Types of TVD pathology:** commissural fusion, septal leaflet tethering, absent or vestigial chordae, tricuspid regurgitation (TR). Better to use the term “Ebstein-like anomaly” in dogs rather than “Ebstein Anomaly” with fenestration of leaflets and papillary muscle attached directly to parietal leaflet. **Natural history** of TVD cases at UCD in last 10 years, 8 year follow up, 77% yet to develop CHF. Factors predictive of progression are the degree of volume overload, vena contracta area, and myocardial dysfunction. Why do some dogs go into CHF post successful TV surgery (maybe 2 years post op) and others not? (cf post MV surgery). Same situation occurs in humans where there is good tolerance of TR. **Surgical timing:** R-sided CHF poor prognosis so operate before CHF develops. Decision regarding surgery based on prediction of progression based on radiographs. The right heart is more poorly protected than the left heart following cardioplegia (same in humans). **Surgical options:** annuloplasty, papillary splitting, chordal reconstruction/splitting, valve replacement preferred – easier! Out of 12 dogs, 83% survival to discharge, 58% long term, 17% thrombus incidence, 20% pannus incidence. **Future directions:** combined valve correction with cavopulmonary anastomosis, better valves, better assessment of PHT (over estimation of PA pressures with massive vena contracta therefore must use catheter assessment of PHT).

**PEARLS AND PITFALLS OF PULMONIC STENOSIS (A.Estrada, R.Pariaut, S.Moise, S.Jesty)**

Selection of dogs: Moderate (especially symptomatic and severe RV hypertrophy) and severe pulmonic stenosis. They suggest in all dogs coronary angiography to discover coronary anomalies and contrast study for patent foramen ovale (increased sensitivity if performed from the saphenous vein). There are pro and cons of having PFO, thus worth knowing its presence also to warn the anaesthetist. In the dogs with subvalvular pulmonic stenosis they recommend balloon selection with ratio 1:1.

Dog position: in dorsal position is easier to pass the introducers in both femoral and jugular, the dog can be subsequently rotated to obtain a more standard position for angiography. Femoral or jugular approach might be easier according to the hypertrophy so is worth preparing both sites and be flexible.

Balloon selection after measuring annulus in multiple planes, both systole and diastole, ratio used 1.2-1.4. It is very important also to consider the length of the balloon measuring the distance between the hinge point and the post-stenotic dilation because if the balloon slips during inflation and is not deflated fast enough can be trapped between valve and PA causing PA rupture. If annulus is more than 20 mm double balloon technique has to be preferred.

Beta-blockers dose up to 1.5-2 mg/kg BID if tolerated. They administer it also the morning of the procedure.

Problems during procedure: suicide RV, diagnosis with measuring RV pressures after ballooning, tachycardia, drop in blood pressure. Treatment is fluid loading with Hetastarch and IV esmolol. To prevent it fluid loading, no use of anticholinergic during GA, keep low HR.
during procedure (60-80 bpm). **Arrhythmias**: someone uses lidocaine CRI or give bolus just before ballooning.

If necessary to repeat the procedure, they might use high pressure balloons.

**Dr David Sisson** talked about **left ventricle outflow tract obstruction (LVOTO) in dogs**. Pyle and colleagues in 1976 provided the first pathological description of SAS in dogs defining 3 grades of lesions in affected Newfoundlands. They also described remodelling of the intramural coronary arteries and subendocardial myocardial fibrosis with severe outflow tract obstruction. Such changes are consistent with the perfusion deficits and are the probable substrate for the formation of malignant ventricular arrhythmia.

Given the diversity of pathologic changes in different breeds and individuals there is the possibility that SAS in dogs and humans may not be a single disorder but several different genetic disorders with different spectra of anatomic lesions leading to obstruction of the LVOT. The pathologic lesions observed in Bull Terriers (stiff and thickened aortic leaflets composed of dense fibrous tissue with islands of cartilaginous metaplasia) are unique and distinct from other forms of LVOTO in dogs. This breed often manifests congenital mitral valve stenosis and/or insufficiency in associations with aortic stenosis. Dogs with SAM and dynamic LVOTO without any trace of fixed obstruction in the LVOT may constitute a different disease. Dogs with dynamic obstruction are often regarded as having HOCM but there is rarely documented evidence of myocardial fiber disarray at necropsy.

The development of the LVOT is a complex and incompletely understood process involving the synchronized participation of the developing muscular interventricular septum, the proximal portion of the conotruncal cushion and the anterior margin of the AV cushions.

The discrete form of SAS in humans is regarded by many investigators to be a consequence of disturbed blood flow in an abnormally formed LVOT. The hypothesis is that turbulent flow via local induction of growth promoter stimulates the development of an encircling ridge of fibroelastic tissue. Persistence of endocardial cushion tissue in the LVOT has also been cited as a contributing element for the development of this membrane. The inciting regional disturbances of blood flow have been attributed to a variety of morphological alterations (aorta displacement, reduced diameter LVOT, reduced aortic distance, reduction angle between aorta and septum, abnormal muscular trabeculae, abnormal papillary muscles), but the precise nature has not been identified.

Discussion on a threshold velocity for the detection of mild obstructions during screening is pointless in the author’s opinion (SAS cannot be conclusively diagnosed if velocity is less than 2.5m/s and no lesions are evidenced) as these consideration will be altered when a genetic test is available.

Treatment: exercise restriction, beta-blockers (despite there aren’t published reports indicating that beta-blockers alter the course of SAS: do beta-blockers decrease risk of sudden death and congestive failure?). Balloon valvuloplasty decreased the pressure gradients but didn’t influence the long term outcome (are there any cases where
morphology could be more amenable to BV?). Surgery decreased the pressure gradient but no difference in survival has been shown compared to atenolol-treated dogs. (Which surgical techniques are better?)

The inference that dogs develop an anatomical substrate that leads unavoidably to sudden death even after relieving the obstruction is not proven. It is also interesting to speculate about the possibility of targeting the development of the developing membrane in young dogs prior to the development of severe obstruction.

Dr Joseph K Perloff MD (world expert in this field) heroically presented via Skype from his sick bed on human Cyanotic Congenital Heart Disease describing the pathophysiology of maladaptive changes. Erythrocytosis in response to tissue hypoxia enhances oxygen carrying capacity. Improved tissue oxygenation may result in a state of equilibrium at higher haematocrit but adaptive failure occurs if increased blood viscosity impairs oxygen delivery.

Points of veterinary interest: hydroxyurea blunts erythropoietin rebound, intrapulmonary haemorrhage as a result of pulmonary neovascularization is a common cause of death in Eisenmengers Syndrome (do not bronchoscope) and haematocrit level (measured electronically not analogue) per se is not a criteria for phlebotomy.

Dr Mark Oyama reviewed Haemodynamic Calculations. Data gathered during cardiac catheterisation and /or echocardiography can be used to calculate haemodynamic parameters which give a deeper assessment of cardiac anatomy and performance than simple parameters such as pressure gradient. Volume formulas based on LV diameter: Cube, Teicholtz, Diameter-Length, Bullet, Simpsons used to calculate end-systolic and end-diastolic volume. EF superior to FS (EF 50% is validated for humans not dogs). Calculations of LV volumes are all based on assumptions about LV geometry. He classified them as Level 1 Cube, Level 2 Diameter-Length, Level 3 Bullet, Level 4 Simpsons (only Bullet formula validated for dogs). Valve orifice area is the gold standard for assessment of stenotic or regurgitant lesions in humans because it is independent of flow unlike pressure gradient which is affected by flow. He was enthusiastic about the Gorlin Equation (the area calculations were verified by necropsy measurements) and outlined the tedious complexities of performing Thermodilution and its limitations.

Found in Translation: Pearls of Veterinary Cardiology from the Overseas Literature in Japan, France, Germany and Italy

Dr Masami Uechi, DVM, PhD reviewed Torasemide a loop diuretic which combines the effects of furosemide and spironolactone acting mainly in ascending loop of Henle. It is more potent and longer lasting than furosemide in dogs and cats. Short term administration of both increased urine volume, long term use decreased urine volume. In dogs and cats, furosemide caused dose dependent increase in urine volume which peaked 2-3 hrs post administration and persisted for 6 hours. Torasemide peaked 2-4 hrs and persisted for 12 hours. In dogs, torasemide significantly decreased urine K+ excretion for 7 days. Plasma aldosterone increased significantly after both drugs but was markedly higher after
torasemide compared with furosemide. Torasemide is 10x more potent in dogs and cats and has a longer diuretic effect. Both diuretics increase blood urea nitrogen and plasma creatinine concentrations. Torasemide may be more potent in that effect.

Roberto Santilli, DVM, PhD, DECVIM-CA (Cardiology) presented 3 studies:

**Utility of Holter monitoring in dogs with arrhythmic syncope** in which he referred to the European Society of Cardiology (ESC) Guidelines for the diagnosis and management of transient loss of consciousness (T-LOC or syncope) and gave an overview of the different causes. Out of the 112 cases in his study, 22/112 had arrhythmias (11/22 brady, 2/22 VT, 9/22 NSR). Holter monitoring had a diagnostic yield of 44.6% in dogs with arrhythmic or neurally mediated T-LOC and showed that Holter recording is an important tool in dogs with suspected arrhythmic or neurally mediated T-LOC with cardio-inhibition particularly when episodes are frequent. It is important to differentiate T-LOC aetiology from arrhythmia due to intrinsic AV nodal disease (eg SSS).

**Vagally mediated paroxysmal atrial fibrillation in dogs** was investigated in 7 dogs using 24hr Holter monitoring. All dogs had T-LOC during the recording followed by paroxysmal AF. Enhanced parasympathetic tone due to appropriate vagal reflex results in heterogeneity of atrial refractoriness, atrial action potential abbreviation and increased pulmonary vein firing all effects that trigger and maintain AF.

**Comparison between conventional and computerised electrocardiography in cats:** Conventional, computerised, computerised printed methods were used to record Lead II ECGs (50mm/sec, 1cm/mV) in 30 cats in right lateral recumbency. All had sinus rhythm with no wandering pacemaker. Rhythm constancy in cats due to the innate higher sympathetic tone compared with higher parasympathetic tone in dogs. The study demonstrated differences between the 3 methods with the computerised method giving duration of P and QRS above reference values for the conventional method.

Valerie Chetboul took the session brief literally and chose 2 French language papers out of her 107 publications. **Echocardiography in Ophidians** from the Swiss Archive for Veterinary Medicine (the oldest veterinary journal in the world, since 1850s) described standardised views in snakes, and bacterial endocarditis as the commonest disease. She gave an overview of **Pulmonic Stenosis in French Bulldogs (Frenchies-the only true Parisian breed!)** which has had a 10-fold increase in popularity (and in incidence of PS) in the last 10 years. Significant predisposition in the breed for PS, followed closely by Bulldogs and Boxers. Complex obstructive lesions common- valvular, supravalvular lesions and hypoplastic PA. Although there was no angiocardiographic data, aberrant coronary arteries were suspected in 14% of dogs.

Matthias Schneider presented 2 retrospective analyses. **7000 ECGs:** Dogs compared with Tilley 1983 had more AVB, less VT and VPCs. Cats compared with Cote 2009 had more AF. **Endocardial diseases** (2003-2008), 400 dogs, 280 cats with the focusing on cats. Diffuse fibrosis in the LV (fibrosis, elastofibrosis), positive correlation with LA size, so maybe a role
in diastolic dysfunction. Mitral endocardiosis in cats correlates with endocardial fibrosis and LV hypertrophy (not with age as in dogs). He drew attention to standardised echocardiographic examination for specific breeds published by the Collegium Cardiologicum. They measure from the apical view except in Irish Wolfhounds which are done from the R parasternal because of published references.

2) Scientific Sessions

There was a huge emphasis in the scientific sessions on methods of diagnosing and staging acquired canine heart disease with particular focus on echocardiography and biomarkers.

Dr Gerhard Weiss presented Clinical Appraisal of the Role of Echocardiography in Acquired Canine Heart Disease. MVD: there are considerable difficulties in assigning guidelines in dogs for MVD. DCM: he summarised and discussed the Guidelines based on the ESVC Task Force DCM Diagnostic Criteria. 3D echo is the future!

Dr Jens Haggstrom and Ingrid Dr Ljungvall presented Can I Trust My Echocardiographic Assessments of Cardiac Dimension and Volumes? They began by describing echocardiography as geometrical and optical illusions and went on to critically evaluate accepted clinical practice and raised many questions. Multicentre collaborations are needed to establish normal values particularly with regard to breed screening.

Dr Jens Haggstrom and Dr Katya Hoglund presented A Critical Review of Neuroendocrine Hormones as Cardiac Biomarkers in Dogs. They briefly covered the existing publications on measurements and values of the hormones in the RAAS system and natriuretic peptides (NPs) in CHF in DCM and MVD. They recommended use of NPs only as an adjunct to other diagnostic tests and advised against screening animals using BNP.

Mrs Katherine Scollan presented Left Ventricular Volume and Function in Dogs Assessed by 3D Echocardiography. 3D echo offers some solutions to problems highlighted so far although not available in clinical practice. The advantages are that there are no LV geometric shape assumptions, no apical foreshortening and fast analysis. The disadvantages are limited availability, expensive and require several heart beats.

Dr David Sisson and Katherine Scollan continued the theme with Left Atrial Size and Function in Dogs Using Echocardiography and MDCT. ECG gated Multidetector cardiac CT offers high spatial and temporal resolution depending on gantry speed, HR and acquisition mode. MDCT is a research tool and is the gold standard for assessing other modalities.

Dr David Sisson gave a fantastic presentation on LA size and Function which included superb CT images demonstrating the complexities of LA anatomy. The American Society of Echocardiography (ASE) recommends CT, 3D echo, 2D (Simpsons) for estimating LA volume all of which are well validated in humans and appear accurate in dogs. A single linear measurement is a complete ‘no no’.

Mr William Brown presented Arrhythmogenic Cardiomyopathy in 163 Boxer Dogs a study
over 10 years based on clinical not echo signs.

Dr Philip Fox’s presentation Myxomatous Mitral Valve Disease: Relevant Pathology described gross dissection techniques and detailed degenerative pathologic structural features. He found a massive variety of lesions compared with only 4 types in the Whitney classification.

Dr Etienne Coté presented an ongoing single centre study on the Effect of Vagal Manoeuvres on the Heart Rate of Healthy Dogs a sparsely documented technique used to terminate AV nodal dependent tachycardia.

Dr Philip Fox: International Collaborative Study to Assess Cardiovascular Risk and Evaluate Long Term Health in Feline Hypertrophic Cardiomyopathy (Reveal). 60 collaborators globally revisited the epidemiology of HCM related death in a large non referral population. In summary results revealed no difference between HCM and HOCM in development of CHF, ATE, syncope, time to cardiac morbidity, time to cardiac death, time to non cardiac death, cardiac death with or without syncope, overall survival.

Dr Lisa Freeman discussed the Effect of Diet on the Progression of Hypertrophic Cardiomyopathy (HCM) in Cats (nutrigenomics).

Dr Suzanne Cunningham explored the Yin and Yang of cardiac inflammatory signalling in her presentation The Fire Within: The Role of Inflammation in Cardiovascular Health and Disease focusing on human data.

Dr Lisa Freeman and Dr John Rush gave Top Ten Treatment Tips for Feline Heart Disease: Feeding and Pharmacology -attention to detail can make a difference.

Dr Denise Schwartz presented a review Obesity is Linked to Heart Abnormalities in Humans. What About Dogs? In summary, studies so far demonstrate same changes in obese dogs as in obese humans.

3) CARDIOLOGY RESEARCH papers

ASSOCIATION OF DILATED CARDIOMYOPATHY WITH THE STRIATIN MUTATION GENOTYPE IN BOXER DOGS. K Meurs. DCM in the boxer is strongly associated with the striatin deletion. 3/33 (10 %) of boxers developed DCM without having the striatin mutation, thus here is at least one other cause of DCM in the dog. The term ARVC with or without myocardial dysfunction should be used to describe the disease in her opinion.

MICRODOSE CT CARDIAC ANGIOGRAPHY IN NORMAL CATS. O'Brien. The purpose of this study was to determine if very low doses intravenous iodinated contrast CT angiography evaluation resulted in significant enhancement for characterisation of cardiac chambers in lightly sedated normal cats using a novel motion restriction device (VetMouseTrap VMT). Conclusion: Survey CT is safe fast can evidence pulmonary oedema very well and can provide information about LA/Ao. Micro dose contrast is safe and fast.
SLEEPING AND RESTING RESPIRATORY RATES IN DOGS AND CATS WITH STABLE CONTROLLED CHF. M Rishniw. Conclusion: Clinicians should try to keep SRR less than 30 where possible, if increasing further investigations or increase diuretic is recommended. Next phase is to investigate how quickly the SRR normalise with appropriate therapy (longitudinal monitoring in dogs and cats with severe subclinical disease).

ATRIAL DEPOLARISATION WAVES LOCALISATION ON SURFACE ELECTROCARDIOGRAM IN DOGS WITH SUPRAVENTRICULAR ARRHYTHMIAS. R Santilli. The aim of this study was to determine atrial depolarisation (AD) waves localisation in a large number of dogs to assess its utility to differentiate supraventricular arrhythmias.

ANALYSIS OF THE FELINE ARTERIAL TROMBOEMBOLISM: CLOPIDOGREL VS ASPIRIN CAT TRIAL (FAT CAT). D. Hogan. This is the final analysis of the study with the aim of determine if there was a difference in secondary recurrence of ATE between aspirin and clopidogrel over 1 year study period. The median survival time for the primary endpoint was 192 days in the Aspirin group vs over 365 days in the Clopidogrel group (Difference more than 173 days).

EFFECT OF ATENOLOL ON QUALITY OF LIFE, ACTIVITY AND CARDIAC BIOMARKERS IN CATS WITH ASYMPTOMATIC HCM. T DeFrancesco. The hypothesis was that atenolol could have an effect on quality of life and or activity level. Overall quality of life score was slightly higher in the atenolol group at 6 months vs baseline. No difference was identified in overall and peak activity between normal and HCM cats pre-treatment (both 80% time day inactive!! Lazy cats!!).

PROSPECTIVE EVALUATION OF NT-proBNP, HIGH SENSITIVITY TRO PonIN I AND PDK4 FOR THE DETECTION OF OCCULT DCM IN 255 DOBERMAN PINSCHERS. S. Gordon. The objective was to screen a large population of asymptomatic/apparently healthy dogs to evaluate the clinical utility of blood-based screening using NTproBNP (tested on a commercially available NT-proBNP ELISA Cardiopet® and with an experimental 2nd generation NT-proBNP ELISA, will soon be available), high sensitivity Troponin I and PDK4 screening. When troponin and BNP were combined (with cut-off of 0.139 ng/mL for cTnI and 535 pmol/L for NT-proBNP) there were zero false negative and limited number of false positive. So in combination they rule out echocardiographic evidence of disease with high accuracy. In this way it is possible to lower the number of dogs that need to have an echo, 81%.

4)  EQUINE SESSIONS

Signature event: Draft Consensus Statement for Equine Athletes with Cardiovascular Abnormalities Chaired by Virginia Reef with Panel members John Bonagura, Rikke Buhl, Gunther van Loon, Colin Schwartzwald and Lesley Young. This was a really interesting session on a consensus statement in the making attempting to establish the basis of the statement “safe to ride”. Confined to horses used in performance based activity sport or athletic horses.
Dr Gunther van Loon presented Treatment of Common Arrhythmias: How and When to Treat with many similarities to SA cases (apart from the risk to ride!) He went into great detail about medical and interventional treatment.

Rikke Buhl’s presentation on Exercise Induced Changes in the Equine Athlete's Heart described in detail the myocardial adaptations in endurance and strength events of the athlete’s heart in humans and horses. TBs and SBs have a combination of strength and endurance remodelling.

Emmanuelle van Erck gave 2 presentations from a clinical practice perspective on Valvular Disorders of Equine Athletes and Assessing Functional Impact of Cardiovascular Disorders in the Field. She described field exercise tests in “real life” conditions rather than on a treadmill.
Transoesophageal echocardiography (TOE) allows imaging of the heart through the oesophagus with a small phased-array transducer mounted on the tip of a modified flexible endoscope. The close proximity of the oesophagus to the heart and minimal intervening structures enable acquisition of high resolution images and optimal study of the heart base anatomy and related structures (Loyer and Thomas, 1995). TOE is useful not only for diagnostic purposes but also for guidance and monitoring of surgical or minimally invasive cardiac procedures as well for some non-cardiac procedures and anaesthesia.

**Technique**

**Transducers**

Several transducers are available from each manufacturer varying in size, length and transducer frequency. Different sized probes are available for use in adult and paediatric patients. In humans, use of the paediatric probe is recommended in all patients weighing less than 20 kg (Hilberath et al., 2010). In dogs, the adult probe could be used safely in dogs as small as 3-4 kg but the paediatric probe should be considered in smaller dogs or cats (Domenech and Oliveira, 2013).

The transducer can be steered cranially or caudally within the oesophagus and rotated in a clockwise or anticlockwise direction. The guidance control allows the tip of the TEE probe (transducer) to be flexed ventrally (forward bending or anteflexion) or dorsally (backward bending or retroflexion) by at least 90°. A locking mechanism allows the tip to be kept in the desired position. Rotation of the phased array transducer from 0 to 180° within the transducer tip is also possible in multiplane probes. This allows production of numerous views of the heart from the same position within the oesophagus. Increasing the angle from 0° to 180° is described as forward rotation and the opposite as backward rotation.

**Examination**

TOE is usually performed under general anaesthesia with the patient in right lateral recumbency (Domenech and Oliveira, 2013). Different positioning may be used if preferred as it does not seem to limit image acquisition and quality (Loyer and Thomas, 1995). Use of a mouth gag is strongly recommended to avoid damage from the teeth.

The probe is inserted in the mouth with the flat side of the transducer tip facing down and is advanced into the oesophagus in the unlocked position. Manipulation of the probe should be gentle at all times to avoid damage to any structure in its path. Resistance is often encountered at the level of the cranial mediastinum with the probe pressing against the aortic arch. Retroflexion of the tip will allow further advancement and imaging of the heart will start at this point.
TOE views are obtained from three positions in the oesophagus (cranial, middle and caudal) and a transgastric position (Loyer and Thomas, 1995). The display of TOE images usually conforms to the standards of the American Society of Echocardiography (Shanewise et al., 1999). Cranial structures are displayed to the left of the screen and caudal structures to the right of the screen (Bussadori and Domenech, 2012; Domenech and Oliveira, 2013).

Cranial views
From a cranial position in the thorax several views may be obtained. The array angle should be set to 0° (or slightly higher) for transverse views and close to 90° for longitudinal views.

Transverse views include (1) a view of the aorta in cross-section with the right ventricular (RV) outflow tract to the left and the RV inflow region to the right (Fig. 1A), (2) a view of both ascending and descending sections of the aorta with the pulmonic valve on the left, the main pulmonary trunk and right pulmonary artery on the near field and the RV outflow tract on the far-field (Fig. 1B) and (3) a view with both pulmonary artery branches and main pulmonary trunk (Fig. 1C). In some dogs, anticlockwise rotation of the transducer may allow visualization of the left atrial appendage.

Figure 1. Cranial transverse views: A. Right ventricular inflow and outflow tracts. B. Pulmonary artery. C. Pulmonary artery bifurcation. Legend: Ao. Aorta; PA. Pulmonary artery; RV. Right ventricle.

Longitudinal views include (1) a view of the left ventricular (LV) outflow tract and aortic arch (Fig. 2A), (2) a view of the aortic arch including the brachiocephalic trunk and left subclavian artery (Fig. 2B), (3) a view of the RV outflow tract, pulmonary trunk and left pulmonary artery in long-axis (Fig. 2C). A patent ductus arteriosus may be visualized using this view (Fig. 2D).

Figure 2. Cranial longitudinal views: A. Aorta; B. Aortic arch; C. Pulmonary artery; D. Patent ductus arteriosus. Legend: Ao. Aorta; BT. Brachiocephalic trunk; LSA. Left subclavian artery; PA. Pulmonary artery; PDA. Patent ductus arteriosus.
**Middle views**

Middle views may be produced by further advancing the probe in a neutral position up to the point in which interference by the trachea is no longer present. Anteflexion is necessary to optimize contact with the oesophageal wall.

Transverse views include (1) a 4-chamber long-axis view with the atria seen on the near-field and the atroventricular valves and ventricles on the far-field of the image (fig. 3A); (2) a 2-chamber view of the left ventricle (Fig. 3B). A longitudinal view including part of the left atrium, mitral valve, left ventricular inflow and outflow tracts, aortic valve and ascending aorta in the same plane (Fig. 3D) may be produced from this position.

![Figure 3. Middle views: A. 4-Chamber transverse view. B. 2-Chamber transverse. C. Longitudinal view of left ventricular inflow and outflow tracts. Legend: Ao. Aorta; LA. Left atrium; LV. Left ventricle; RA. Right atrium; RV. Right ventricle.](image)

**Caudal views**

From the middle position, further advancement and anteflexion of the probe will produce caudal views.

Transverse views include (1) a view of the aortic valve in transverse cut in the centre of the image surrounded by the left atrium on the near-field, the right atrium and right ventricular inflow tract on the right and the right ventricular outflow tract and pulmonic valve on the left (Fig. 4A); (2) a view of the left auricle (Fig. 4B); and (3) a short-axis view of the left ventricle at the level of the mitral valve cusps (Fig. 4C). Short-axis images of the left ventricle below this area are difficult to obtain due to lung interference (Loyer and Thomas, 1995).

![Figure 4. Caudal transverse views: A. Right ventricular inflow and outflow tracts. B. Left auricle. C. Short-axis of the left ventricle at the level of mitral valve. Legend: Ao. Aorta; LA. Left atrium; LAu. Left auricle; LV. Left ventricle; MV. Mitral valve; RA. Right atrium; RV. Right ventricle.](image)
Caudal longitudinal views include (1) a view very similar to the middle longitudinal views with the left ventricular outflow tract in a more horizontal position relative to the ultrasound beam, and (2) a view of the right atrium and RV inflow tract.

**Transgastric view**
A transverse short-axis view of the left ventricle at the level of the papillary muscles may be obtained in some dogs from within the stomach with the probe completely anteflexed.

**Complications**
Complications with TOE may include risk of damage to any structure in the probe's path from the mouth to the stomach, compression of mediastinal structures, airway compromise, transmission of infectious disease and dysrhythmias among others. In veterinary medicine there is little information regarding complication rates with TOE. A few studies have provided evidence of minimal risk for oesophageal damage in dogs (Urbanowicz et al., 1990; Loyer and Thomas, 1995). Partial obstruction to pulmonary venous flow has been described from the caudal oesophageal position (Kienle et al., 1997).

**Indications**
In veterinary medicine TOE is increasingly used in referral centres mainly as a monitoring tool in the interventional cardiology setting and as a diagnostic tool to a lesser extent. It’s use in non-cardiac surgery (i.e. minimally invasive occlusion of portosystemic shunts) has also been described. Use of TOE for guidance and monitoring of cardiac surgical procedures, monitoring of patients under anaesthesia and in the intensive care setting will likely increase in the future, similarly to what happens in human medicine.

**TOE in the interventional cardiology setting**
TOE is an invaluable tool in the intervention cardiology setting as it provides high quality real-time imaging free of ionizing radiation. It contributes to a reduction in exposure to ionizing radiation and also the amount of radiographic contrast used.

TOE is very useful in PDA occlusion procedures in dogs (Pariaut et al., 2004; Saunders et al., 2010) as it provides appropriate visualization of the PDA and fairly accurate measurements of ductal dimensions (Domenech et al., 2009; Saunders et al., 2010; Saunders et al., 2007). A recent study involving 40 dogs that underwent percutaneous PDA occlusion with Amplatz canine duct occluder (ACDO) reported that TEE provided anatomic information with regard to PDA morphology which closely approximated that provided by angiography while aiding in device deployment, release and confirmation of closure (Saunders et al., 2010).

TOE may also be useful in balloon valvuloplasty procedures for pulmonic stenosis. It allows further study of the anatomy and accurate measurements of the pulmonic annulus, particularly in dogs with limited acoustic windows (Locatelli et al., 2011). As a monitoring tool TOE may assist on assessing the position of catheters within the heart and mainly of the balloon catheter relative to the pulmonic annulus, however it does not allow visualization of the waist produced during inflation (Domenech and Oliveira, 2013). The success of the procedure may be ascertained indirectly by assessing an increase in valve
leaflet movement and regurgitation although accurate estimates of the drop in pressure
gradient across the obstruction is often not possible since appropriate and consistent
alignment for Doppler interrogation may be difficult to obtain (Domenech and Oliveira,
2013).

Other applications of TOE in the interventional cardiology setting described in veterinary
medicine, include guidance of atrial and ventricular septal defect occlusion (Bussadori et al.,
2007; Gordon et al., 2009) and removal of heartworm with flexible alligator forceps (Arita et
al., 2003)

**TOE as a diagnostic tool**

TOE can be valuable in the diagnosis and study of congenital heart defects. In some cases
with suspicion of coronary artery anomalies, TOE may allow confirmation without use of
selective angiography (Saunders, 2011). Application of TOE for the diagnosis of other
conditions may also be found in the veterinary literature such as aortic aneurysm, cranial
vena cava syndrome and pulmonary thromboembolism (Chetboul et al., 2003; Mulz et al.,
2010; Venco et al., 1998). TOE may also be valuable for the diagnosis and accurate study of
heart base tumours and cardiac neoplasia (Domenech and Oliveira, 2013).

**Conclusions**

TOE is a valuable imaging technique that is becoming increasingly available in veterinary
medicine. It is increasingly used as monitoring tool in the interventional cardiology setting
and as diagnostic tool for cardiac disease in many cardiology referral centres and surely
with time other applications will be reported.
References:


The main indications for TOE in humans are reviewed. A systematic approach to performing and reporting TOE is presented with reference to the British Society of Echocardiography guidelines and using a number of illustrative case studies.
Validation/accreditation of echo skills

Navroz Masani
University Hospital of Wales and Spire Cardiff Hospital

British Society of Echocardiography developed voluntary processes for Individual Accreditation in Adult and Paediatric Transthoracic Echocardiography in 1993. The program has been extended to include Accreditation in Community, Critical Care and Transoesophageal Echocardiography. Accreditation involves an examination (written and cased analysis), supervisor report, logbook and sample case submissions. The process has become a professional standard for cardiologists and physiologists performing and reporting echocardiograms independently and has been adopted by the European Association of Echocardiography. A system of reaccreditation every 5 years has been instituted, that involves a combination of CPD “points”, logbooks and supervisor reports.
Case Report: Complex congenital defect in a Bichon Frise

Chris Fellows  
Lakes Cardiology Services, UK

A three month old Bichon Frise (male) was presented with a history of a grade II/IV systolic murmur detected at routine vaccination. He was bright and lively, with no exercise intolerance or breathing difficulties. He was normal size for his age and compared to his siblings.

On examination his heart rate was regular at 160 beats per minute. His pulses were normal, his mucous membranes were pink with a normal c.r.t and the only abnormality detected was the presence of a soft grade II/VI systolic murmur audible on both sides of the chest but difficult to localise. A low grade diastolic murmur was also present. This report presents the findings of the diagnostic tests performed in this unusual case.
Pre-hypertrophic HCM: review

David Connolly
Royal Veterinary College, Herts, UK

Hypertrophic cardiomyopathy (HCM) is identified in about 1 in 500 people. It is the leading cause of sudden death in young adults and results in significant disability in survivors. In people mutations in at least 11 genes encoding proteins of the cardiac sarcomere (with over 1,400 variants) are associated with HCM. In most cases HCM is inherited as an autosomal dominant trait with variable penetrance. Alterations in two genes, $\beta$-myosin heavy chain and myosin-binding protein C (MYBPC) account for approximately 75% of cases where an underlying mutation has been identified. HCM also affects up to 1 in 7 cats with an increased prevalence in pedigree cats such as Maine coons and Ragdolls where the specific mutations in the myosin-binding protein C gene have been identified.

In both species HCM is frequently progressive and is characterised by left ventricular hypertrophy without dilation, impaired diastolic function, heart failure, tachyarrhythmia and sudden cardiac death. The phenotypic diversity of feline HCM parallels that seen in humans and includes heterogeneous ventricular hypertrophy and inter/intra-familial clinical variability. Equally, principal aspects of the pathophysiology including myocyte disarray, interstitial fibrosis and intramural coronary artery disease, mitral valve abnormalities are comparable between the two species. Despite progress in elucidating the genetic basis of HCM there remains a remarkable deficit in understanding the molecular events that lead from sarcomeric mutation to the diverse disease phenotypes described above.

Myocardial fibrosis is a distinguishing feature of HCM and recent studies in genetically predisposed people (genotype positive, phenotype negative) indicate that far from being a late manifestation of HCM, augmentation of pro-fibrotic signalling pathways occur during the subclinical disease, before the onset of overt myocardial hypertrophy – that is during the pre-hypertrophic stage of the disease resulting in myofibre disarray, mitral valve abnormalities and arteriosclerosis. Furthermore evidence from rodent models engineered to express sarcomeric mutations suggests that the activation of pro-fibrotic pathways in cardiac fibroblast occurs early in the natural history of the disease and may play an essential role in driving subsequent myocardial hypertrophy through paracrine signalling including TGF-$\beta$ amongst others.

The lecture will describe some of these concepts and outline potential ways of exploring them in greater detail as well as outlining the close similarities between HCM in cats and humans.
References:

Maron BJ, Maron MS, Semsarian C. Genetics of hypertrophic cardiomyopathy after 20 years. J Am Coll of Cardiol 2012;60:705-715


Hypertrophic cardiomyopathy (HCM) is the most common inherited cardiac disease in people\textsuperscript{1,2} and cats.\textsuperscript{3,4} It is characterised by excessive thickening of the left ventricular myocardial walls and may result in sudden death, heart failure or thromboembolic events.\textsuperscript{5}

Echocardiography is the main clinical tool for diagnosis of human HCM\textsuperscript{6,7} and feline HCM,\textsuperscript{8} although in people cardiac magnetic resonance imaging (CMR) is becoming more commonly used for diagnosis.\textsuperscript{5,9} While the severe cases of hypertrophic cardiomyopathy (HCM) with obvious left ventricular hypertrophy (LVH) are simple to diagnose, the mild end of the phenotypic spectrum creates more of a diagnostic challenge. There is a wide phenotypic spectrum seen in both people\textsuperscript{6,10} and cats\textsuperscript{11} with some overlap in the characteristics of HCM, restrictive cardiomyopathy (RCM) and unclassified cardiomyopathy (UCM) phenotypes recognised in people\textsuperscript{12} as well as indistinct boundaries between HCM, RCM and UCM phenotypes in cats.\textsuperscript{13-15} Additionally, there is incomplete penetrance of known genetic mutations and varying genotypic-phenotypic correlations seen in both people\textsuperscript{16-20} and cats.\textsuperscript{21-23} The current consensus is that while any left ventricular wall thickness may be seen in individuals with human HCM,\textsuperscript{5,18} the use of an end-diastolic left ventricular wall thickness (LVWd) cut-off to define HCM is most practical in a clinical setting and as such cut-offs are used in both human and veterinary medicine.

Feline HCM reports have so far been mainly based on referred cases, although there have been some small studies of non-referral cats. The prevalence of murmurs in apparently healthy cats ranges between 15.5\% and 33.7\%, with physiological murmurs being common.\textsuperscript{3,4,24} The prevalence of feline HCM in apparently healthy cats has been reported as 14-16\%.\textsuperscript{3,4} The aim of this study was therefore to screen a cohort of apparently healthy cats in two feline rehoming centres, Battersea Dogs & Cats Home and Cats Protection’s National Cat Adoption Centre, to obtain cross-sectional prevalence data for HCM in a non-referral population (‘CatScan’).

Apparently healthy cats aged greater than 6 months who were waiting for rehoming were eligible for enrolment in the study. Cats with known or controlled hyperthyroidism or hypertension were excluded, as were cats with diabetes mellitus, cats with renal disease sufficient to cause the clinical signs of polyuria and polydipsia and cats with other systemic diseases causing clinical signs. Pregnant or nursing cats were not screened.

Basic data were collected about each cat including unique identification number, name, pen location, age (often as an estimated age range), date of birth (if known), sex, neutering status, breed, weight, body condition score assessed on a scale of 1-9 based on the Purina
scale (Laflamme, 1997) and whether the cat was a stray or had been relinquished to the home by a previous owner. If known, historical and concurrent medical problems were recorded. Cats were assessed as to their temperament, with cats rejected if they were deemed aggressive or too nervous to be handled.

Auscultation was undertaken on three occasions, initially in the cats’ pens, then prior to echocardiography in the echocardiography room and post echocardiography. If cats were purring they were not forced to stop unless they had purred during all three auscultation periods. Cats were dissuaded from purring using various distraction techniques including a running water tap, knocking on doors or under tables or the smell of surgical spirit. Systolic blood pressure was measured in all cats using the Doppler method. Echocardiography was performed with cats in right lateral recumbency on a purpose built table top using a MyLab echocardiography machine (ESAOTE, Cambridge, UK).

Classification of the presence of myocardial disease was based on echocardiographic findings of maximal end-diastolic left ventricular wall thickness (LVWd) and left atrial size measured using either short axis left atrial to aortic ratio (LA:Ao) or long axis left atrial length (LAD):

<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>LVWd &lt;5.5 mm</td>
</tr>
<tr>
<td>Equivocal</td>
<td>LVWd 5.5-5.9 mm</td>
</tr>
<tr>
<td>‘Low risk’ HCM</td>
<td>LVWd 6.0-8.9 mm with one of:</td>
</tr>
<tr>
<td></td>
<td>- LA:Ao &lt;1.50 and LAD &lt;16.0 mm, or</td>
</tr>
<tr>
<td></td>
<td>- LA:Ao 1.50-2.00 and LAD &lt;16.0 mm, or</td>
</tr>
<tr>
<td></td>
<td>- LA:Ao &lt;1.50 and LAD 16.0-20.0 mm</td>
</tr>
<tr>
<td>‘High risk’ HCM</td>
<td>LVWd ≥9.0 mm</td>
</tr>
<tr>
<td></td>
<td>or</td>
</tr>
<tr>
<td></td>
<td>LA:Ao ≥1.50 and LAD ≥16.0 mm</td>
</tr>
<tr>
<td>Other cardiac disease</td>
<td>The presence of other myocardial disease or congenital disease</td>
</tr>
</tbody>
</table>

In a subset of cats, residual blood was used to measure N-terminal pro-brain natriuretic peptide (NT-proBNP) and high sensitivity cardiac troponin I.

A summary of findings was provided to the rehoming centres and to the new owners of the cats in an owner pack. Information was also provided to pass on to the cats’ new veterinarian. All cats were invited to sign up to the longitudinal arm of the study, either via a sign-up sheet in their owner pack or via www.rvc.ac.uk/CatScan, being offered repeat echocardiographic examinations for free.

Murmurs in cats were found to be common, increasingly so with repeated auscultation. The positive predictive value of a murmur for detecting HCM was found to be low overall,
although increased with age, whereas the negative predictive value was high. Different LVWd were explored for the diagnosis of HCM. The prevalence of HCM was found to be similar to that previously reported, although few cats were considered to be high risk for future cardiac events. Models predicting the presence of HCM were generated, including models using the subset of cats in which NT-proBNP was measured, correctly classifying more than 85% of cats.

References:


**Safety of oral administration of spironolactone in cats with heart failure: interim results of the Seisicat study**

Rachel James, Malcolm Cobb and Jacqueline Gilmour  
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Spironolactone, an aldosterone antagonist, has been shown to decrease the risk of cardiac-related death, euthanasia, or worsening of cardiac failure in dogs with moderate to severe mitral regurgitation caused by myxomatous mitral valve disease, when added to conventional cardiac therapy. In cats, cardiomyopathy (CM) is the predominant cause of heart failure. Activation of the renin-angiotensin-aldosterone system (RAAS) occurs in cats with cardiomyopathy and signs of congestive heart failure. Spironolactone inhibits aldosterone-induced sodium retention in the kidney, leading to decrease cardiac preload, prevents aldosterone induced fibrosis and improves endothelial function. In Maine Coon cats with familial HCM, spironolactone at 2mg/kg POq12h for 4 months was not shown to improve the mitral annular velocity or reduce the left ventricular mass and 4 of the 13 treated cats developed severe ulcerative facial dermatitis.

To evaluate further the safety and efficacy of spironolactone in cats with CM, a double blind, randomized placebo-controlled study is being conducted with cats receiving spironolactone at 1.7 to 3.3 mg/kg PO sid or placebo for up to 15 months. This dosage was extrapolated from the current approved dosage in dogs (i.e. 2 mg/kg sid). As the study is still ongoing, the authors remain blinded as to which cats are receiving spironolactone so the following results relate to the whole population, receiving spironolactone or placebo, which are being administered in addition to conventional cardiac therapy (including at least furosemide and benazepril).

Twenty cats (17 DSH, 1 ragdoll, 1 Siamese and 1 Burmese) with CM of various types (15 hypertrophic, 2 dilated, 2 unclassified and 1 Arrhythmogenic Right Ventricular) have been enrolled so far.

To date, 5 cats have died at 2 to 228 days post inclusion, 8 cats have been euthanized at 34 to 215 days post inclusion, 2 cats were withdrawn from the study (at Day 26 and Day 67), 2 cats remain in the study and 3 have completed the 15-month follow-up.

Any adverse event (of cardiac origin or not) was systematically recorded. Severe cardiac failure leading to death or euthanasia was recorded for 8 cats which presented either with worsening cardiac failure (4), aortic thromboembolism (2) or acute respiratory distress (2). One cat died suddenly. Vomiting was observed in 2 cats and was followed in one of them by anorexia and loss of weight leading to euthanasia. The mean value of all hematology and serum or urine biochemistry parameters remains within the laboratory reference range. Hypokalaemia (< 3.5 mEq/l) was recorded in 6 cats at inclusion and in 4 cats at least one study visit after inclusion. Hyperkalaemia has not been recorded. No dermatitis has been recorded to date.
In conclusion, no severe adverse events except worsening of heart failure, aortic thromboembolism and one case of anorexia have been recorded in 20 cats with CM treated with spironolactone at 1.7 to 3.3 mg/kg PO sid or placebo for a mean duration of 147 days (2 to 427 days) in addition to conventional cardiac therapy including furosemide and benazepril.
Case Report: Different manifestations of congestive heart failure in three cats

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Small Animal Hospital, University of Glasgow, UK

Congestive heart failure is a clinical syndrome that can be seen in different forms and it is triggered by a variety of primary conditions. Presentation differs substantially between species and individuals and important factors such as the anatomy and physiology play an important role in this diversity. Cats in left sided congestive heart failure typically show pleural effusion and/or pulmonary oedema, whereas cats in right sided CHF may have pleural effusion and/or ascites with hepatic congestion. Many other presentations and clinical signs have been described in the literature and this series of three cases show some of the less common presentations.

CASE 1:
A nine year old male neutered Domestic Short Haired presented with a three week history of dyspnoea, tachypnea, mild lethargy and inappetence. On physical examination a grade III/VI systolic left sided murmur, arrhythmia and a heart rate of 160 bpm were present. Routine blood tests including T4 were unremarkable except a mild increase in urea. On echocardiography there was mild pleural effusion, marked pericardial effusion, left atrial enlargement with the presence of a thrombus, subjective right atrial enlargement and moderate reduction in systolic function. ECG showed right bundle branch block with frequent ventricular premature complexes. A diagnosis of Unclassified Cardiomyopathy with congestive heart failure was made. He was managed with oxygen, rest, Frusemide, Benazepril, Spironolactone, Pimobendan and Aspirin. No pericardiocentesis was performed.
He progressed well showing a clear improvement in clinical signs. On follow-up echocardiography three weeks after presentation there was a minimal amount of pericardial effusion present.

CASE 2:
A five year old male neutered Domestic Short Haired was referred with dyspnoea and radiographic changes compatible with pulmonary oedema and ascites. On physical examination he showed marked jugular distension, bilateral pulmonary crackles and an arrhythmia. There was mild azotaemia and increase of ALT on the routine blood profile. T4 was within normal limits. On echocardiography there was marked enlargement of all cardiac chambers with poor systolic function. On ECG ventricular premature complexes were present. A diagnosis of Dilated Cardiomyopathy with biventricular congestive heart failure was made. He was treated with Frusemide, Pimobendan and Aspirin. On revisit he
improved clinically but the ascites persisted. Three weeks after initial presentation he became anorexic for two days and as the owners were unable to medicate him he was euthanized.

CASE 3:
A twelve year old male neutered Domestic Short Haired presented with a one week history of non-painful hind limb swelling and mild lethargy. On physical examination he was tachycardic at 240 bpm with weak femoral pulses, subcutaneous oedema was palpated in all limbs but more marked on the posterior ones and he had a plantigrade gait. Routine blood profile showed adequate protein levels, azotaemia and a normal T4 value. Abdominal ultrasound revealed ascites with hepatic congestion. On echocardiography there was mild pericardial and pleural effusion, biatrial enlargement, mild thickening of the left ventricular free wall, poor systolic function and a thrombus in the left atrium. A diagnosis of Unclassified Cardiomyopathy with biventricular congestive heart failure including peripheral oedema was made. He was treated with Frusemide, Pimobendan, Aspirin and Clopidogrel. On follow-up there was a marked improvement in the clinical signs and the peripheral oedema and ascites had resolved. Subsequently he had an episode of thromboembolism affecting his right forelimb that completely resolved. About a week after this he had another thromboembolic episode, this time to the back limbs, and consequently he was euthanized.

The pathophysiology of congestive heart failure is complex and poorly understood in cats. Much of the information has been extrapolated from human or canine literature. There are some differences in the presentation, prevalence, consequences and severity of the clinical signs in cats compared with other species. Studies are needed for a better understanding of this clinical syndrome and its management.
References:

Cote E, MacDonald KA, Meurs KM, Sleeper MM. Chapters 19 and 22. In: Feline Cardiology. WB. 2011.


A 2 year old male neutered Maine Coon presented for echocardiography for pre-screening of HCM. The cat was reported to be general healthy and owners reported that the cat had grown to expected size, but poor body condition was seen, and body weight was 6.7kg, with a BCS of 4/9 at examination. Physical examination was unremarkable, with no evidence of increased respiratory rate or effort, and lung auscultation was unremarkable. Of note was a shifting of audible cardiac sounds from the ‘typical’ sternal location to lateral thoracic auscultation.

Cardiac echocardiography revealed the heart to be displaced dorsally within the thorax, and peristaltic intestines were seen surrounding the heart within the pericardial space. Thoracic radiography was performed which revealed a marked enlargement of the cardiac silhouette, dorsal displacement of the trachea, a hypoplastic liver and large bowel loops filled with faeces, which could be tracked to the level of the cardiac silhouette, consistent with peritoneopericardial diaphragmatic hernia (PPDH).

Surgical repair of the defect was performed and recovery was uneventful, with post-operative thoracic radiography confirming resolution of the defect. Re-assessment of the cat approximately 4 months later revealed weight gain to 8.1kg, and an increase in body condition to 5/9. Thoracic auscultation revealed audible heart sounds over the parasternal area, with no murmurs audible and a heart rate of 180bpm with fair strength matching pulses, and a detectable arrhythmia. ECG assessment revealed a bigeminal rhythm with rare right sided (left bundle branch block morphology) ventricular premature complexes. Echocardiography was performed and revealed a large anechoic structure associated with the pericardium. Thoracic radiography was performed which confirmed an enlarged cardiac silhouette and elevation of the heart from the sternum. Close monitoring was performed over the next 4 months, at which point a thoracic CT revealed a large fluid filled, non-contrast enhancing, cyst like lesion in the right ventrolateral aspect of the thorax approximately 9 x 4 cm. Exploratory thoracotomy and removal of the structure was performed 24 hours after CT and the patient recovered uneventfully. Fluid from the cyst was analysed and cultured and consistent with a pericardial cyst. No neoplastic, inflammatory or infectious processes were seen.

6 month follow up reveals no further cystic lesions, and a resolution of arrhythmia. The patient has lost weight and is now 6.7kg, although is an active outdoor cat. Heart rate was 170bpm, and echocardiography, ECG and thoracic radiography revealed no further abnormalities.

This report described the first analysis of a pericardial cyst developing post-operatively from repair of PPDH.
References:


This year the ECVIM-CA congress was held at the Arena and Convention Centre, Liverpool. The main subject for the European Society of Veterinary Cardiology lectures was pulmonary hypertension in small animals, as well as in adults and children.

Twenty-one cardiology abstracts were presented covering a wide variety of subjects in veterinary cardiology. There were also a number of poster abstracts on display, presenting current studies from different parts of Europe. We would like to thank VCS for the bursaries received to fund our attendance at the congress.

1) **LECTURES:**

**Dr J Häggström et al. (Sweden): Feline cardiomyopathy in Europe: New concepts and results of screening.**

Professor Häggström gave us an overview on the HCM screening program Pawpeds set up together with a Maine Coon breeder in Sweden in 1999. Pawpeds has expanded to include other inheritable diseases, more breeds and more examiners, and is now well established in several countries across Europe. Through the years 106 examiners have been involved, of which six see a large number of cats. Yearly, between 3500-4000 cats are screened; some undergo screening on a regular basis. The breeds involved include Maine Coon, Norwegian Forest Cat, BSH, Birma, Siberian, Ragdoll, Sphynx, Rex, Bengal, Persian and Exotic cats. The case details and test results are forwarded to administrators of the program for website publishing; the website has a list of recommended examiners as well as a list of results for each individual cat. Results and forms are regularly checked to ensure the quality of tests performed. A set of formal requirements has been stipulated for the selection of examiners; these include theoretical and practical training as well as having access to a high quality ultrasound system. Prof. Häggström found, after detailed data analysis, the normal reference value for diastolic left ventricular- and septal thickness to be <5 mm in cats under 6 kg. The analysis revealed that wall thicknesses vary in different breeds; certain breeds such as Sphynx and Persians have a greater LV/IVS wall thickness than others even if the values are corrected for body size. Certain diseases were more common in specific breeds e.g. Birmans had a higher prevalence of RCM and ARVC compared to other breeds.

Prof. Häggström emphasised the importance of recognizing unusual non-pathological phenotypes such as false tendons and ectopic papillary muscles, as well as other cardiac diseases. The cats are classified normal, equivocal, HCM positive (mild, moderate, severe) or other disease. Cats with values 5.0-5.9 mm should be considered equivocal unless the examiner have an explanation for the high value, such as unusual phenotype or poor image
acquisition; or finds clear objective and subjective evidence of HCM, such as local or uniform hypertrophy >5 mm, systolic anterior motion of the mitral valve and papillary muscle hypertrophy. Re-testing is always recommended in borderline cases. The data analysis revealed a median age of 1.8 years, age range 3mths-16 years; 9% of examined cats had to be sedated for the exam; 1.6% of the normal cats had a heart murmur, 19% of equivocal- and 57% of positive cats had heart murmurs. The results demonstrated a HCM prevalence of 3.6%, with 3.9% of cats testing equivocal. This prevalence may not reflect the prevalence in the general population as the group has a low median age, most cats are pedigree and cats tested HCM positive are unlikely to undergo re-examination. The presentation included a discussion of the genetics of HCM and the validity of the commercially available genetic tests. Prof. Häggström concluded that despite genetic screening, echocardiography is still required; he also suggested that histopathology remains the gold standard and encourages breeders to submit cats for post mortem evaluation.

Dr LH Olsen and Dr T Falk (Denmark). Vascular and endothelial function in mitral valve disease.

Olsen and Falk talked about the different stages of myxomatous mitral valve disease and associated vascular changes. These changes include hyperplasia in systemic and pulmonary arteries, intramural arteriosclerosis with resulting myocardial ischemia and extracellular myocardial fibrosis. Circulating biomarkers have been used to assess the severity of disease and to establish endothelial function and vascular changes. Olsen talked about how a study by Pedersen et al found decreased plasma nitric oxide metabolite levels in dogs with asymptomatic MVD compared to healthy dogs, whereas the levels were increased in some dogs with CHF. Von Willebrand factor is decreased in dogs with MVD, likely due to shear-stress induced destruction. A recent study by Falk et al showed that Cardiac Troponin I is raised with arteriosclerosis and fibrosis in dogs with MVD, and that cTNI is a potential biomarker for these vascular and myocardial changes. Moesgaard et al recently demonstrated how flow mediated vasodilation is decreased in dogs with MVD, using a non-invasive functional study with 2D ultrasound of the brachial artery. Olsen concluded that further studies are necessary, but that new knowledge regarding pathophysiological mechanisms may lead to improved prognostic, diagnostic and therapeutic strategies in the future.

Dr D Chan (RVC-UK). Pulmonary thromboembolism in dogs.

A wide variety of systemic diseases can predispose the dog to formation of PTE; the ultimate consequence of hypoxaemia results from ventilation:perfusion mismatch. Clinical signs associated with PTE are variable, but dogs often present with acute respiratory compromise, exercise intolerance and syncope. In some dogs a predisposing condition is recognised, however many cases go undiagnosed. Diagnostic tests can aid in the diagnosis, but findings may be nonspecific. Haematology/serum biochemistry is of limited value. Blood gas analysis will show hypoxaemia, hypocapnoea and increased alveolar-arterial oxygen tension gradient. Routine coagulation profiles are typically normal and of limited value. Testing D-dimers is common in human patients, but results have been inconsistent in dogs. A recent pilot study suggested that cTNI levels showed a stronger relationship than D
dimers in dogs. Thoracic radiographs may be normal, non-specific, or may show typical
signs: regional hypo-vascular lung areas distal to the thrombus, regional areas of pulmonary
infiltrates, pulmonary artery enlargement, pleural effusion and right-sided cardiac
enlargement. Echocardiography is considered safe, non-invasive and useful in the risk
assessment of PTE. Typical findings are RV dilatation and hypo-kinesis, septal
flattening/paradoxical septal motion, LV diastolic impairment and PA hypertension. Nuclear
medicine ventilation: perfusion scanning is theoretically a valuable diagnostic tool, however
practical difficulties associated with anaesthesia and radiation limit its clinical utility,
especially in compromised patients. CT angiography is considered the gold standard in
people, performed whilst the patient breath-holds. Although technological advances have
made the test more valuable in veterinary medicine, further studies are warranted to
evaluate the usefulness of this test, especially in the conscious patient.

2) ABSTRACTS:

MJM Dirven, Netherlands: Echocardiographic findings in 246 adult cats with heart disease
in general practice.
This abstract presented the echocardiographic findings in a group of 436 adult cats
generated from general practice and rescue centres. Eight of these had congenital disease
and 238 had acquired disease; 200/238 cats had left ventricular hypertrophy. One hundred
and five of these had dynamic left ventricular tract outflow tract obstruction, whilst isolated
DLVOTO was found in 12 cats. Seven cats were classified as having UCM, four as RCM, one
as DCM and one with ARVCM. Left atrial enlargement, described as LA:Ao ratio of >1.5, was
found in 40/200 cats with HCM, 6/7 cats with UCM, 4/4 RCM, 1/1 DCM and 1/1 ARVCM.

AS Hanås et al, Sweden: 24-hour Holter monitoring of cats with asymptomatic HCM
before and after treatment with atenolol.
Twelve cats underwent an echocardiographic exam, blood tests and blood pressure
measurement prior to being fitted with a seven electrode Holter-ECG. The 24-hour Holter
exam and blood pressure measurements were repeated after 30 days of treatment with
atenolol or placebo. Before treatment the mean HR was 158, mean systolic BP was 140
mmHg, the cats had normal sinus rhythm with tall R waves or left anterior fascicular block
and mild sinus arrhythmia. The median number of VPCs was 3 (IQR 1-29), the median
number of SVPCs was 0. No significant differences were noted within or between groups
with regards to heart rate, blood pressure and arrhythmia before and after treatment.
Hanås concluded that the study did not support the treatment of asymptomatic cats with
atenolol, but that a larger number of cats may be needed to provide more conclusive
results.

B Pedro et al, Liverpool, UK: Indices of myocardial strain and strain rate by
two-dimensional speckle tracking echocardiography in Great Danes.
Pedro presented a study of 127 GD assessed echocardiographically in the period 2008-2012.
Images obtained underwent offline analysis of radial and circumferential strain (RSst, CST)
and strain rate (RSR, CSR) by speckle tracking echocardiography (STE). 42 dogs were
classified normal, 38 dogs affected. Equivocal dogs and dogs diagnosed with other disease were excluded. The affected group had lower radial and circumferential strain and strain rate values than the normal group. CST and CSR seemed to be most useful in the differentiation of normal from affected dogs and may allow early identification of systolic dysfunction in pre-clinical DCM.

**J Lee et al, Japan. Pathological manifestations and clinical correlates in canine degenerative mitral valve disease at surgical biopsy**
Intra-operative biopsies from 117 dogs undergoing mitral valve repair were taken from the left atrial appendage, left ventricle and lung to assess pathological changes in different stages of DMVD. Pathological changes such as LV fatty replacement, immune cell infiltration and interstitial fibrosis were found in all ISACHC groups. Significant changes in heart failure cells in lung biopsies were only found in group III. Lee concluded that severe pathological changes take place from the earliest stages of DMVD and that the echocardiographic parameters LVEDd and EF may be effective in predicting myocardial pathologic state. This abstract won the prize for the best cardiology abstract presented at the congress.

**G Santarelli et al, Spain. Effects of combination of acepromazine/butorphanol on conventional echocardiographic measurements and global strain in healthy dogs.**
Six dogs of different breeds were given 0,02 mg/kg ACP and 0,2 mg/kg Butorphanol im. Echocardiographic exam was performed before and 30mins after administration. Conventional measurements of the LV and LA were performed, as well as TDI and radial/longitudinal global strain by STE. Following sedation the heart rate decreased, the LV diastolic volume increased and the LVWd increased, however other parameters did not change significantly.

**ECVIM update: Part 2**

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ECVIM Congress was held over three days in Liverpool this year. Lectures included clinical research abstracts as well as talks by veterinary and also human cardiologists with the main theme being pulmonary hypertension (PH). The three days ended with a panel discussion.

**1) ABSTRACTS**

**Roberto Santilli** spoke about 24-hour ambulatory ECG findings in English bulldogs (EBD) with myocardial disorders. The aim was to evaluate the prevalence of arrhythmias in the EBD with arrhythmogenic right ventricular cardiomyopathy (ARVC) compared to normal dogs of the same breed. 136 medical records were examined and those dogs with congenital heart disease, advanced AV block or accessory pathways were excluded. The inclusion criteria were that the dogs were not receiving antiarrhythmic medication and 22
hours of ECG trace was required. 35 dogs with myocardial disorders and 10 normal dogs were included. The Holter findings were similar to those reported in boxers with ARVC and included VPCs, runs of ventricular tachycardia and supraventricular tachycardia (SVT). He concluded that the dogs with cardiomyopathy had a higher number of ectopic beats, a higher ventricular rate and longer runs of SVT compared to normals. One of the limitations of his study was that the controls were significantly younger than the diseased dogs.

**M.P. Perego** looked at the response of SVT to manual cardioversion (MC). A video was shown as to how and where to strike a blow with the fist to a specific place on the thorax. The group retrospectively examined twelve-lead ECGs during MC in 13 cases with orthodromic atrioventricular reciprocating tachycardia (OAVRT) and in 6 cases during focal atrial tachycardia (FAT). MC interrupted SVT in 84% of cases (92% of OAVRT and 66% FAT). In 100% of cases, the last deflection before the interruption of the SVT was a P’ wave followed by a pause. In all cases sinus rhythm was present after the pause, however, the SVT then re-occurred. No complications occurred.

**K. Creamer** presented the results of 3 commercially available types of smartphone application software (Apps) to assess heart rate (HR). The aim of the study was to determine the degree of correlation between App-derived HR and ECG-derived HR. 37 dogs and 7 cats were included in the study. The Apps included the “Instant Heart Rate Monitor” (IHRM), the “Heart Beat Monitor” (HBM) and the “Heart Monitor” (HM). There was no significant correlation between the HR on ECG and the HR detected with either HM or HBM. The results suggested that IHRM was detecting half of the heart beats.

**K.F. Scollan** discussed administering mexiletine (in combination with sotalol) twice daily instead of three times daily per os. The results indicated that therapeutic serum mexiletine levels were maintained with twice daily dosing (8 – 10 mg/kg) when used in combination with sotalol (2.5 mg/kg) in dogs greater than 10 kg.

**A. Vollmar** looked at the prevalence of whole blood taurine in Irish wolfhound dogs with and without echocardiographic evidence of dilated cardiomyopathy. This study has been published in the most recent issue of the Journal of Veterinary Cardiology.

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**2) TALKS ON PULMONARY HYPERTENSION**

**Dr Michele Borgarelli** spoke about pulmonary hypertension (PH) as a secondary complication of left heart disease in dogs and included some findings from a VIN questionnaire answered by veterinary cardiologists on-line. Left sided heart diseases are a common cause of PH in dogs, with the most common being myxomatous mitral valve disease (MMVD). The pathophysiology of PH with left sided heart disease was discussed. The findings of a study was presented looking at prevalence and effects on survival of PH in dogs with stage B2 and stage C MMVD as per the ACVIM consensus statement on chronic valvular heart disease. 212 dogs were recruited between 2010 and 2011: 100 dogs were classified as stage B2 and 112 stage C. Survival times were then established with the end of the study period being March 2013. In this study, the prevalence of PH was found to be
found to be 39% (24 dogs stage B2 and 59 dogs stage C). 105 dogs died and those dogs with PH had worse mean survival than those without PH. LA:Ao ratio >1.7 and TR pressure gradient > 55 mmHg were associated with worse survival. Finally, treatment of PH was discussed with the primary focus being to decrease left atrial pressure. The question was asked whether we should trust our Doppler estimation of pulmonary hypertension and also whether we should perform treatment tests.

Dr Robert Naeije from the University of Brussels discussed the human classification of PH. In 2003, a mutation was discovered in association with “idiopathic PH” therefore separating out a heritable cause: bone morphogenetic receptor type 2 (BMPR2) gene mutation. There has been considerable progress in the diagnosis of PH over the last 2 decades with improved classification and targeted therapy. He then provided us with a second talk on “Imaging modalities for assessment of PH”. Some really nice cardiac MRI images were projected and the talk was based mainly on the right heart. Pulmonary hypertension was described as involving progressive remodelling of the pulmonary vasculature with progressive remodelling of the right heart and eventually, increased right ventricular dimensions. Two modalities predominantly used for evaluation of right ventricular (RV) function include echocardiography and cardiac MRI. Cardiac MRI is superior to echocardiography for detecting ischaemia, measuring RV mass and also detecting congenital cardiac abnormalities (as possible causes of PH). The advantages of MRI over echocardiography were discussed and summarised. In humans, right atrial pressure is assessed from the inspiratory collapsibility of the inferior vena cava using ultrasound. This is used in addition to the trans-tricuspid pressure gradient to estimate systolic pulmonary artery pressure. RV function was discussed with tricuspid annular plane excursion (TAPSE) being described as the most reproducible measurement (it is determined in M mode). Measurements with the strongest prognostic content in severe PH include RV fractional area change, RA area, the eccentricity index (parasternal short axis ratio of perpendicular and parallel to septum LV dimensions), TAPSE, strain and presence of pericardial effusion. He concluded that echocardiography was essential for diagnosis of PH and RV failure and that the procedure must include a complete set of measurements of the pulmonary circulation and the RV.

Dr Graham Derrick from Great Ormond Street Hospital presented an excellent talk on the assessment and management of human paediatric patients with PH. Survival time in children with untreated PH (< 1 year) is less than in adults (2 years). In people, short acting vasodilators (specific to the pulmonary circulation) are administered to assess “responder status”. A “responder” is a person who, after treatment, obtains a reduction of more than 10 mmHg pulmonary artery pressure to < 40mmHg (mean pulmonary artery pressure). The Fick method for assessing cardiac output is used for those patients with PH and congenital cardiac shunts (thermal dilution methods cannot be used in these cases). Invasive therapy used for paediatric patients with PH include atrial septostomy, ductal stent, surgical Potts procedure and transcatheter stent (Potts shunt). Mortality with atrial septostomy is up to 25% with the other disadvantage of the procedure being that they heal and close.