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Programme Veterinary Cardiovascular Society meeting

Hall 9, International Convention Centre, Birmingham
Wednesday 5th April 2017, pre-BSAVA meeting

8:30 -9:00	**REGISTRATION**	
9:00-09:40	Hypertrophic cardiomyopathy: cats vs. people ***Virginia Luis Fuentes**, Royal Veterinary College, London, UK*	7
09:45-10:25	Cardiac disease in the Dogue de Bordeaux ***Gavin McAulay**, New Priory Vets, Brighton, UK*	
10:30-11:00	**COFFEE BREAK & SPONSORS' EXHIBITION**	
11:00-11:30	The clinical approach to heart failure – Florida style! ***Simon Swift**, University of Florida, Gainsville, Florida, USA*	
11:35-12:05	Are ACE-inhibitors *always* effective? *Ever* effective? ***Marisa Ames,** Colorado State Univ., Fort Collins, Colorado, USA*	
12:10-12:40	Ineffective ACE-inhibition; what can be done about it? ***Marisa Ames,** Colorado State Univ., Fort Collins, Colorado, USA*	
12:45-13:45	**LUNCH BREAK & SPONSORS' EXHIBITION**	
13:45-14:15	Fun in the cath lab: novel interventional techniques ***Simon Swift**, University of Florida, Gainsville, Florida, USA*	
14:20-15:00	Progression of hypertrophic cardiomyopathy ***Virginia Luis Fuentes**, Royal Veterinary College, London, UK*	
15:05-15:25	Three-dimensional studies of congenital heart disease in a pony **John Keen,** Royal (Dick) School of Veterinary Studies, Edinburgh, UK	
15:30-16:00	**COFFEE BREAK & SPONSORS' EXHIBITION**	
16:00-17:00	Interactive session: What type of cardiomyopathy is it? **Virginia Luis Fuentes,** *Royal Veterinary College, London, UK* Chair: Malcolm Cobb	
17:00-18:00	Breed-related conditions subcommittee meeting (members only)	

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Prof Virginia Luis Fuentes MA VetMB PhD CertVR DVC MRCVS DipACVIM DipECVIM-CA

Virginia is Professor of Veterinary Cardiology, and Head of the Cardiology Service at the RVC. Her primary interests are cardiomyopathies in cats and mitral valve disease in dogs. She has a particular passion for progressing our understanding of the prevalence and progression of feline hypertrophic cardiomyopathy, and defining the key pathological changes in this disease. She is also interested in aortic thromboembolism, and in particular its pathogenesis and risk factors. Virginia also has interests in cross-sectional imaging techniques for the heart, such as cardiac MRI for mitral valve disease and CT angiography for congenital disease. Virginia hopes to improve the management of feline myocardial disease by participating in several prospective longitudinal clinical trials.

Gavin McAulay BVetMed CertEM (IntMed) CertVC MRCVS

Gavin runs the cardio-respiratory referrals service at New Priory Vets Brighton. Gavin graduated from the Royal Veterinary College in 1996. He spent five years in equine sports practice developing an interest in internal medicine and cardio-respiratory disease, gaining the RCVS Certificate in equine internal medicine. Gavin subsequently made the transition to small animal medicine, gaining the RCVS certificate in veterinary cardiology. He is pursuing the RCVS cardiology diploma from private practice and has worked as a clinician at the Cardio-respiratory service at the University of Glasgow. His current interests include pulmonary hypertension, interactions of cardiac and respiratory disease and feline myocardial disease.

Simon Swift MA VetMB CertSAC MRCVS DipECVIM-CA

Simon is Clinical Associate Professor in Cardiology at the University of Florida, in Gainsville, USA. He worked extensively as a Cardiologist in the UK, and has worked in both teaching hospitals and private referral practice. His particular interests include degenerative mitral valve disease, cardiac interventional procedures, and the management of arrhythmias.

Marisa K. Ames DVM DipACVIM

Marisa Ames, DVM, Diplomate ACVIM (cardiology) and assistant professor at Colorado State University College of Veterinary Medicine, is a 2007 graduate of the Ohio State University. She recently completed her cardiology residency and the Jane Lewis-Seaks postdoctoral fellowship at North Carolina State University. Her research interests include neurohormonal activation in heart failure (specifically the pharmacologic blockade of the renin-angiotensin-aldosterone system [RAAS], the effects various drugs on RAAS, and aldosterone breakthrough) and heartworm disease.

John A. Keen BVetMed PhD CertEM (IntMed) MRCVS DipECEIM

Following graduation from the RVC in 1996, John spent 4 years in mixed and then equine practice before being appointed the RCVS Clarke and Sparrow Resident in Equine Studies at the 'Dick' Vet in 2000, where he has remained ever since. John is currently a senior medicine clinician in the equine hospital and Director of the Equine Hospital and Practice. He gained an MSc and PhD investigating the pharmacology and physiology of digital laminar microvasculature; became a Diplomate of ECEIM in 2007; and is an RCVS and European specialist in equine internal medicine. He has a strong clinical interest in cardiology and research interests focus on equine cardiovascular disease, metabolic/endocrine disease, laminitis and the potential links between these disorders.

Hypertrophic cardiomyopathy: cats vs. people

Virginia Luis Fuentes
Royal Veterinary College, London, UK

Pending.

Cardiac disease in the Dogue de Bordeaux

Gavin McAulay
New Priory Vets, Brighton, UK

The Dogue de Bordeaux (DdB) is a large brachycephalic working dog classified as a Molosser breed. After a catastrophic decline in popularity in the mid-20th century, the DdB has recently grown in popularity, especially in the decade after 1998, when the breed saw a 1204% increase in numbers (Asher et al., 2009). The population and cardiac pathology of DdB in the UK is not well described however anecdotal reports suggest DdB are frequently presented for cardiac evaluation and diagnosed with atrial fibrillation (AF).

The DdB has been reported to be affected by various cardiac conditions including subaortic stenosis (SAS) (Höllmer et al., 2008; Ohad et al., 2013; Oliveira et al., 2011), tricuspid dysplasia (TD) (Ohad et al., 2013) and dilated cardiomyopathy (DCM) (Borgarelli et al., 2006; Martin et al., 2009). A predisposition to developing supraventricular tachyarrythmias has previously been suggested and in one study, all DdB diagnosed with TD had AF (Locatelli et al., 2011; Ohad et al., 2013).

This presentation reviews the results of a retrospective study describing the clinical phenotype of 64 DdB within a cardiac referral population within the UK. Congenital heart disease particularly sub-aortic stenosis was prevalent, as was echocardiographic evidence of cardiac neoplasia. Supraventricular arrhythmias, particularly atrial fibrillation, were frequently diagnosed. Potential aetiologies of atrial fibrillation are discussed.

The results of an online health survey of Dogue de Bordeaux with special emphasis on cardiac disease will also be discussed. This survey collected information regarding the prevalence of sudden / unexpected death within the breed, owner / breeder perceptions of heart disease in DdB and their attitude to a possible breed heart testing scheme.

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The clinical approach to heart failure – Florida style!

Simon Swift
University of Florida, Gainsville, FL. USA

Heart failure in the United States is perhaps unsurprisingly, similar to heart failure in the UK. However, we have some differences in the drugs that are available and the underlying disease processes.

Heartworm is prevalent in Florida and the climate requires year round prevention unlike in some parts of the Unites States. Unfortunately not all clients use prevention and so infection can occur from the mosquito. A small worm burden may not cause acute signs. However, the dog will develop pulmonary hypertension with dilated pulmonary arteries and right ventricular hypertrophy. Tricuspid regurgitation with pulmonary hypertension can lead to the development of right side congestive heart failure. Management can difficult, as the changes to the pulmonary vasculature are likely to be irreversible. Typically, sildenafil is used to try to reduce pulmonary artery pressures although the effect on tricuspid regurgitation velocity is usually disappointing. In addition, the standard treatment of furosemide, and ACE inhibitor with pimobendan is given once heart failure develops. Ideally, adulticidal treatment is used but doxycycline is given first to treat the commensal organism, Wolbachia. If the radiographic changes are marked, there is always a concern that killing the worms may result in acute death so steroids may be given although this is not ideal in the setting of acute heart failure.

If the worm burden is large, acute caval syndrome can develop. This is an emergency and requires surgical extraction of the worms. A large number worms fall back into the right ventricle and right atrium. They can often be visualized on echocardiography prolapsing through the tricuspid valve. The dogs are usually in right sided failure and have haemaglobinurea due to shredding of red cells in the heart. The dog is placed in left lateral recumbency and under sedation/light general anaesthesia, the right jugular vein dissected. A variety of extraction forceps have been used and my favourites are Ishihara forceps which are relatively atraumatic and have a steerable tip. It is important to avoid breaking the worms as this can trigger an anaphylactic reaction. A combination of fluoroscopy and echocardiography are used to help guide the forceps into the right ventricle and right ventricular outflow tract and grasp the worms. Extraction continues until no further worms can be removed and the echocardiography is used to confirm the result. It is not uncommon to leave one or two worms in the pulmonary arteries which will then be treated using an adulticide.

Treatment of left sided congestive heart failure has changed in the last few years in the states as sodium nitroprusside has become very expensive. Initial treatment depends on how critical the patient is. For the more dyspnoeic patients, further investigations may need to be

delayed while the patient is stabilized. Ideally, chest radiographs are obtained to confirm the presence of left sided congestive heart failure with pulmonary oedema visible in the peri-hilar region.

Treatment then includes:

1. Oxygen supplementation in a cage: This helps improve blood oxygenation and reduces the activity of the patient reducing energy use. Smaller dogs and cats can be placed in an oxygen cage but larger dogs may overheat if the cage is not large enough
2. A potent loop diuretic such as furosemide is given intravenously so that it acts rapidly and has some vasodilatory action as well decreasing preload. Doses of 2-4mg/kg are commonly used
3. Pimobendan is given at 0.2-0.3mg/kg for its calcium inodilator properties. An intravenous form is available in Europe but not in the USA
4. Anti-anxiolytic agents: If the patient is stressed, a low dose of an opioid such as buprenorphine or butorphanol may help

At this point, the patient should be observed over the next 1-2 hours for response. The production of a large volume of dilute urine is a good sign the diuretics are having an effect. The respiratory rate and effort should be counted every 30 minutes and a declining trend seen. If this is not apparent, another dose of furosemide should be administered or the patient switched to a furosemide CRI at 1mg/kg/hr. It is important to monitor response carefully and decrease the dose rate once the respiratory rate declines or the patient could become severely dehydrated with electrolyte abnormalities. Alternatively, the more potent loop diuretic, torasemide can be used. Blood pressure should be measured and treatment tailored to the value obtained.

5. Vasodilators: Patients in acute heart failure may be normo- or hypertensive. With degenerative valve disease, decreasing the afterload with an arteriolar dilator can be very beneficial. Sodium nitroprusside could be used to set blood pressure exactly and 90 – 100 mmHg systolic is the range we aim for. We have used hydralazine more recently and it seems to be effective although prolonged use often results in a rebound tachycardia. We have also started using intravenous nitroglycerine and are currently looking to whether it is effective – it is difficult to identify an effect on blood pressure.
6. Other positive inotropes: If the blood pressure is low in patients with systolic dysfunction, a sympathomimetic agent such as dobutamine may be indicated. The dose of dobutamine is 5-20µg/kg/min as a short term intravenous agent.

7. Antiarrhythmic agents: Fast ventricular tachycardia should be controlled due to the associated risk of sudden death and the inefficiency of ventricular contractions. Atrial fibrillation if fast should be slowed with care as the dog may be relying on the increased heart rate to maintain output. However, often controlling heart failure reduces the adrenergic stimulus and the rate will fall anyway. Too aggressive rate control can be detrimental.

Over the next 24-48 hours, the patient is switched to oral medications including furosemide and pimobendan. Generally, an ACE inhibitor is started once the patient is out of heart failure and eating well. Spironolactone can also be started at this time for its anti-aldosterone effect.

Are ACE-inhibitors *always* effective? *Ever* effective?

and

Ineffective ACE-inhibition: what can be done about it?

Marisa K. Ames
Colorado State University, Fort Collins, CO. USA

The interruption of RAAS is a key strategy in the therapy of congestive heart failure (CHF) and chronic kidney disease with proteinuria (CKD_P), and is typically achieved via the administration angiotensin-converting enzyme inhibitors (ACEI), angiotensin II (ATII) receptor blockers (ARB), and mineralocorticoid receptor antagonists (MRA), alone or in combination. ACEI have been shown in several placebo-controlled, double-blinded studies to improve survival and reduce symptoms in dogs with CHF, due to both myxomatous mitral valve disease (MMVD) and dilated cardiomyopathy (DCM).[1-4] As a result, there is consensus among veterinary cardiologists that ACEI should be part of the 'standard therapy' for CHF in dogs.[5] Similarly, ACEI have been shown to improved outcomes in dogs (and cats) with proteinuria.[6-8] However, even though ACEI are very effective in reducing ACE activity in dogs[9,10], aldosterone levels may not always be adequately suppressed.[11-14]

Incomplete pharmacologic blockade of the RAAS system by adequately dosed ACEI and/or ARB is referred to as aldosterone breakthrough (ABT). The incidence of ABT in people being treated for CHF, CKD_P, or systemic hypertension is somewhere between 10% and 53%.[15] Although the existence and importance of ABT are uniformly accepted, there is no consensus regarding the definition, mechanism, and clinical implications of ABT in people with either CHF or CKD_P. Of note, however, is that ABT formed the rationale behind the RALES, then EPHESUS, and EMPHASIS studies that proved the benefit of MRA in patients with heart failure and reduced ejection fraction (HFrEF).[16-18] ABT does exist in veterinary medicine and its incidence has been studied in the authors' clinics. Our laboratory studies[8-10] and early clinical data suggest that RAAS activation occurs in clinical and preclinical heart disease and in proteinuric renal disease.

A validated aldosterone radioimmunoassay has been used in the determination of the urinary aldosterone to creatinine ratio (UAldo:C). The UAldo:C, using 'spot' urine samples has been correlated to both serum aldosterone levels[13] and 24 hour urinary aldosterone excretion.[19] We have used the UAldo:C to demonstrate and study ABT in an experimental model of RAAS activation.[11-13] A specific ABT definition is needed for clinical cases and two strategies have been applied to this end. The first, used when a UAldo:C is available (ideally prior to initiation of therapy), defines ABT as a UAldo:C that exceeds the baseline value for that patient. The second definition of ABT utilizes the UAldo:C of 55 normal dogs (mean UAldo:C of 0.44 µg/g, ± 0.23). We found that the UAldo:C rarely exceeds 1.0 µg/g and does not change significantly with age (range 1-16 years) in normal dogs. The mean +2SD of the maximum UAldo:C of dogs most reflective of our patients (>5yrs, n=21)[20] was 1.0 µg/g. This value was used to create the ABT cut-off definition. Therefore, in patients receiving an ACEI, a UAldo:C >1.0µg/g indicates ABT, while in those not yet receiving an ACEI, a UAldo:C >1.0µg/g simply indicates RAAS activation. The UAldo:C is less useful as a determinant of ABT in patients receiving MRA, as blockade of the mineralocorticoid receptor leads to an increase in circulating aldosterone levels.[21]

We have evaluated the UAldo:C in 78 dogs with cardiac disease. Half of these dogs had MMVD and the remainder had DCM, congenital heart disease, or pulmonary hypertension. Forty-five dogs were sampled while receiving an ACEI, and most were on long-term ACEI therapy. Of these, 34 were receiving furosemide for CHF.

- The percentage of ABT in the 34 dogs receiving furosemide and ACEI was 37%
- The percentage of ABT in the 11 not receiving furosemide was 36%
- For 24 dogs, sampled prior to an ACEI, RAAS activation was observed in 45%
- For 9 dogs with CHF receiving spironolactone, the UAldo:C was significantly elevated
- In the 39 dogs with MMVD, receiving an ACEI without spironolactone, ABT was present in
    - 32% of dogs with CHF and
    - 30% in dogs without CHF

We have evaluated the UAldo:C in 47 dogs with proteinuria.

- Serial UAldo:C and urinary protein:creatinine (UP:C) measurements were available in 15 dogs receiving an ACEI:
    - UAldo:C increased or was unchanged from baseline in 6/15 (40% ABT)
    - UP:C increased (worsened) in 3/6 dogs with ABT and 4/9 dogs without ABT
- Only a single UAldo:C was available for an additional 32 proteinuric dogs
    - Of 19 receiving an ACEI, the incidence of ABT was 7/19 (37% ABT)
    - For 13 proteinuric dogs not receiving an ACEI, 4/13 (31%) had RAAS activation

Despite significant advances in the treatment of CHF and CKD_P in dogs, morbidity and mortality remain high. The importance and impact on outcome/survival of ABT in veterinary patients remains to be determined and mechanisms of ABT remain under investigation. In the authors' opinion, improved pharmacotherapy for these diseases, with MRA and other strategies, will likely require a better understanding of ABT. As an example, it is not known whether ARBs or dual RAAS blockade (ACEI and ARB) would decrease the incidence of ABT in dogs, though in people this does not appear to be the case.[22-24] Theoretical benefits of ARBs include blockade of the actions of ATII at the AT_1 receptor regardless of its formation (via ACE or chymase). Also increased circulating AT II, resulting from AT_1 receptor blockade, may stimulate the AT_2 receptor, thought to be counter-regulatory to the maladaptive actions mediated by the AT_1 receptor. Regarding the MRA spironolactone, evidence supporting its use (in addition to an ACEI) in dogs with CHF due to MMVD already exists.[25] The use of eplerenone, or the non-steroidal MRA finerenone, has not been studied in veterinary patients. It is also worth noting that, on average, human subjects in the RALES trial had low-normal circulating aldosterone levels, yet a population benefit was still seen with MRA. This finding is possibly explained by the release of natriuretic peptides which counteract RAAS (reduce circulating aldosterone concentrations), ongoing cardiac damage being due to local tissue production of aldosterone, and possibly the activation of the MR by glucocorticoids in damaged cardiac and vascular tissue.[26] RAAS peptide profiling (i.e. screening for ABT) may still be helpful in individualization of pharmacotherapy, yet, as with the humans in the RALES trial, patients without ABT may still benefit from MRA. Our data support the use MRA in *at least* one quarter to one third of dogs with either heart or proteinuric kidney disease. The proportion of dogs that benefit from MRA, however, may be underestimated by only considering dogs

with increased urinary aldosterone levels, as evident in the RALES data.[16] As ABT occurs in some patients receiving ACEI, prior to the onset of CHF, initiation of early supplemental MRA therapy may be indicated. Furthermore, ABT occurs early (<1 week) in our experimental model of RAAS activation[8-10] It may then be that, in lieu of testing for ABT, a MRA is simply chosen whenever an ACEI is initiated.

Exciting advances and research in the field of valve repair, valve replacement, stem cell therapy, and gene therapy for dogs are on-going, yet medical therapy for heart failure will remain a cornerstone of management for most patients. Advances in our understanding of existing molecules such as the ACEI, ARB, and MRA will likely continue to improve the medical therapy of CHF and CKD_P. Additionally, further study of the counter-regulatory axis of RAAS (ACE2, Ang(1-7), and Mas) may open new medical therapeutic options. Combination products should also improve the cost and convenience of treatment and clinical research now underway may further streamline pharmacotherapy. Finally, future studies of the chronobiology of RAAS system activity indicates that timing of administration of RAAS blocking drugs such as ACEI may influence their efficacy.

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Fun in the cath lab: novel interventional techniques

Simon Swift
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The University of Florida College of Veterinary Medicine is blessed by having a state of the art catheter lab with 30 fps fluoroscopy and a haemodynamics system as well are being situated a 15 minute walk from Shands paediatric cardiac hospital. We enjoy great relations with the cardiologists there and attend catheter and surgery conferences regularly. As a result of this, we have jointly developed novel techniques to address some of the problems we face, learning from techniques used in children.

Sub-aortic stenosis: We developed the combined high pressure and cutting balloon technique for this procedure. There is no doubt that we can decrease the gradient in the short term but in some the stenosis redevelops. Currently there is no evidence that dogs that undergo the procedure have a longer survival than those who do not, although clinically the owners report improvement in exercise tolerance and decreased syncope. Furthermore, it is difficult to understand how this procedure would produce superior results to surgical resection and infundibular myomectomy as performed by Chris Orton. We tend to offer it to owners of dogs with gradients over 130 mmHg as they have a particularly bad long term prognosis with a median survival of 2.8 years with the caveat that the effects on survival have not been demonstrated. While the high pressure balloons (rated burst pressure of 18 atm) are available in a variety of sizes, the cutting balloons are only available up to 8 mm so they may be too small for the typical breeds of dogs susceptible to SAS.

Pulmonic stenosis: The outcome for these patients undergoing balloon valvuloplasty is never certain. While most type A dogs have a significant reduction in their pressure gradients, some do not respond well or show a rapid return to previous gradients. The outcome is more uncertain in dogs with type B lesions which include valve dysplasia and an element of pulmonary artery hypoplasia. It may be that the patients with a poor outcome had stretching of their valve rather than tearing of the leaflets. We have developed a stent technique using a balloon expandable bare metal stent to open the outflow tract in dogs that respond poorly to balloon valvuloplasty. We have performed this in 5 dogs and all dogs continue to do well despite significant pulmonic regurgitation. There is no evidence of right ventricular volume overload in dogs we have followed for over 3 years.

Small dogs with severe stenosis can present a particular problem if the concentric hypertrophy reduces the right ventricular lumen or there is significant dynamic collapse of the RV lumen as the catheter will not turn into the outflow tract. This can be made worse if there is severe tricuspid regurgitation and a large right atrium. In some dogs, it is easier to reach the pulmonary artery via the jugular vein and in some via the femoral vein. Steerable catheters and weasel wires can also help. However, if the right ventricular outflow tract cannot be accessed, we have developed a hybrid technique that involves direct catheterization of the right ventricle in a mini-thoracotomy. The 12 F introducer can be used to guide the stent into position.

Atrial pacing: For patients that will benefit from atrial pacing – and that population is not well defined – the stability of the atrial lead can be a problem. Atrial leads have a J bend in them that allows them to lie in the right auricle and engage with the trabecula muscles. We have been looking at a technique using a catheter with a J bend at the end. The active lead is a fed down this and used to maintain interatrial septal apposition while the screw thread is deployed. We now have reasonable success with this technique.

Interaction with our paediatric colleagues continues to generate ideas and their assistance has been invaluable in developing the practical techniques we use in Florida. And we have gators and manatees!

Progression of hypertrophic cardiomyopathy

Virginia Luis Fuentes
Royal Veterinary College, London, UK

Pending.

Three-dimensional studies of congenital heart disease in a pony

John Keen
Royal (Dick) School of Veterinary Studies, University of Edinburgh, UK

Three-dimensional (3D) imaging has the potential to improve our assessment, knowledge and teaching of congenital heart disease in animals. This case report describes the evaluation of congenital heart disease in a pony and the subsequent development of three-dimensional models of the heart following euthanasia.

A 6-year-old Welsh Section A pony presented for evaluation of a cardiac murmur detected at routine health check for vaccination. The pony was unridden and had no outward clinical signs of cardiac dysfunction, such as failure to thrive or reluctance to exercise at pasture. Clinical examination revealed a regular heart rate of 40 beats/minute. Mucous membranes were pink and moist with a normal capillary refill time. Palpable pulses and jugular vein filling were within normal limits and there was no evidence of subcutaneous oedema. Non-invasive blood pressure (coccygeal artery) was 83/54 (mean 61) mmHg. Cardiac auscultation revealed a left sided, grade 5/6 band-shaped pansystolic murmur, with its point of maximal intensity (PMI) at the pulmonary valve, and a musical component at end systole. A grade 3/6, decrescendo early to mid-diastolic murmur was also audible at both the pulmonic and aortic valves. On the right side of the chest, a grade 5/6 harsh, band-shaped pansystolic murmur was present with the PMI over the tricuspid valve, radiating cranioventrally.

Two-dimensional (2D), Doppler and real-time 3D echocardiography was used to confirm the clinical suspicion of a ventricular septal defect (VSD) with left to right shunting from the peri-membranous portion of the left ventricular outflow tract. An overriding aorta and four-leaflet pulmonary valve were also noted but there were no signs of pulmonary stenosis or right ventricular hypertrophy. A subjectively moderate degree of aortic insufficiency was present with regurgitant flow into the left and right ventricles. There was also mild pulmonary regurgitation and tricuspid regurgitation, but no mitral regurgitation. The maximum diameter of the VSD was 2.1cm using 2D echocardiography but 3D echocardiography suggested this was an underestimate since the maximum diameter following manipulation of a 3D zoom acquisition of the left ventricular ouflow tract was 3.5cm. The speed of flow through the VSD was 3.9m/s, giving a pressure gradient of 60.8 mHg and a calculated RV pressure of 22mmHg. Right ventricular stroke volume assessed by PW Doppler was 900ml while that of the left ventricle was 1.2l. The left ventricular inflow E waves were higher than expected for a Welsh pony, but within the range of normal fit Thoroughbreds. The left ventricular internal diameter was enlarged for a pony of this size, as were left atrial measurements.

These findings suggested volume overloading of the left heart, likely a combination of a non-restrictive VSD and significant aortic regurgitation. Given these findings, use as a child's riding pony was not recommended. The owner opted for euthanasia. Immediately following euthanasia, the heart was retrieved and suspended upright in a sealed tub of Jore's solution to facilitate acquisition of a CT and MRI dataset. Subsequent post mortem examination confirmed the findings from echocardiography examinations. CT and MRI datasets were uploaded into specialist software (*Materialise Mimic inPrint*) to reconstruct the heart in 3D (Figure 1a and b) and from this, printed

models of the heart structure and blood volume were derived.

Although this was a relatively simple example of a congenital heart defect, 3D imaging could help in clinical evaluation and understanding of more complex defects. In particular, the use of 3D reconstructed models has a place in undergraduate and postgraduate teaching, negating the need for specimens stored in formalin.

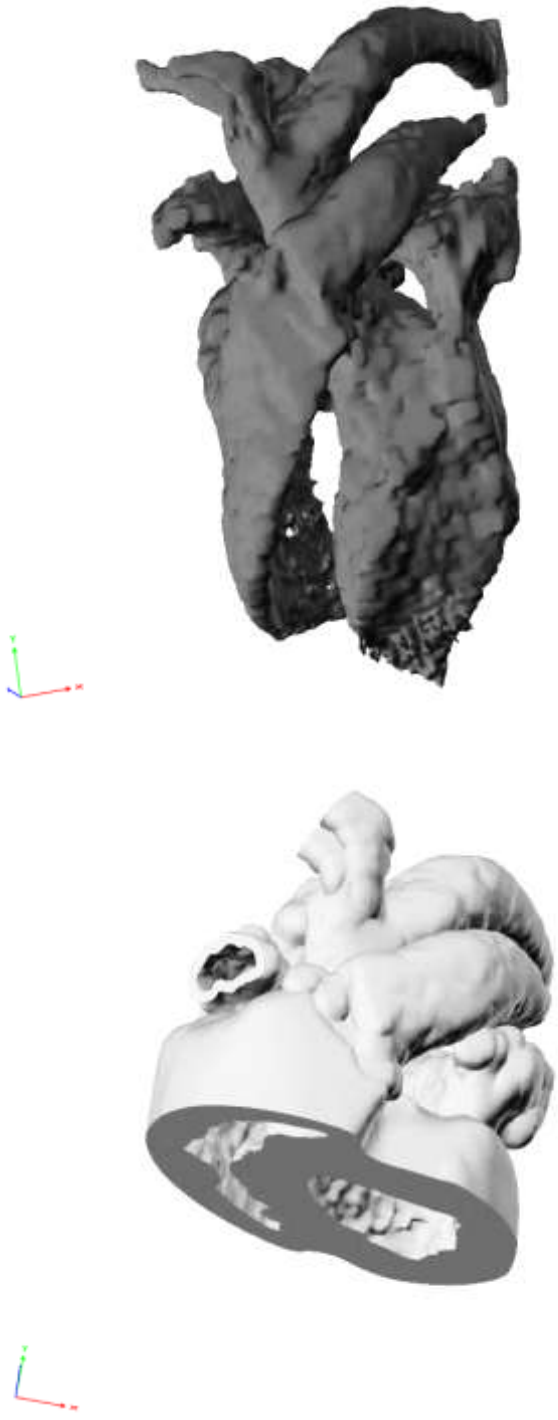

Fig 1: Reconstructed 3D images of blood volume

(a) and heart structure (with most of ventricles removed)

b) derived from 3D datasets in a Welsh Section A pony (*Materialise Mimic inPrint* software).

Interactive session: Which cardiomyopathy is this?

Virginia Luis Fuentes, RVC

Chair: Malcolm Cobb