



Proceedings of the Autumn Meeting of the Veterinary Cardiovascular Society

Friday 8th – Saturday 9th November 2019

(Joint meeting with the British Equine Veterinary Association)

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Friday 8th November 2019
Chesford Grange Hotel, Kenilworth, Warwick, CV8 2LD

8:30 -9:00	REGISTRATION	
9:00-09:50	Introduction to congenital echocardiography <i>Dr Navroz Masani, Cardiff and Vale University Health Board</i>	12
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17.00-18.00	AGM (VCS members only)	
19.00-19.30	Pre-dinner drinks	
19.45	DINNER	

Saturday 9th November 2019
Chesford Grange Hotel, Kenilworth, Warwick, CV8 2LD

9:00-9:30	REGISTRATION	
9:30-10:20	Development and use of interventional clinical audit – a tool for everyone? <i>Chris Linney, Willows Veterinary Specialists</i> <i>Mike Martin, Veterinary Cardiology Consultancy</i>	59
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17.00	CLOSE	

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Dr. Navroz Masani, Consultant Cardiologist, Cardiff and Vale University Health Board

Dr. Masani trained in medicine at Guys Hospital Medical School. He subsequently undertook cardiology speciality training in London and Cardiff, and completed an Echocardiography Fellowship in Boston, USA. He was appointed as a consultant cardiologist and director of echocardiography at University Hospital of Wales in 1999. Dr. Masani is a British Society of Echocardiography Council member, and has also served as Chairman of Education and Past President. He is currently the director of the Cardiff Echocardiography Course.

Dr. John Bonagura DVM MS DACVIM (Cardiology, Internal Medicine)

John Bonagura is a faculty member in the Dept. of Clinical Sciences at NC State University CVM and Professor Emeritus of the Ohio State University. John is board-certified by ACVIM (Cardiology & SAIM) and past president of the Cardiology specialty. He has been Visiting Research Fellow at the University of Edinburgh and Gilbreath-McLorn Endowed Professor of Cardiology at the Univ. of Missouri. John has published over 225 scientific papers and book chapters and co-edits Kirk's Current Veterinary Therapy. He has received over 30 awards for teaching including the OSU campus Award for Distinguished Teaching. Dr. Bonagura is a recipient the BSAVA Bourgelat award, Doctor Honoris Causa from the Universitat Autònoma de Barcelona, the Kirk Lifetime Achievement Award from ACVIM, and Distinguished Alumnus Awards from OSU and the Animal Medical Center in New York.

Prof. Colin Schwarzwald DrMedVet PhD DACVIM DipECEIM

Colin graduated in 1997 from the University of Zurich School of Veterinary Medicine. Between 1998 and 2001 he was intern and clinical instructor at the Equine Hospital of the University of Zurich and worked on his doctoral thesis to attain the Dr. Med Vet degree. Between 2001 and 2004 he completed an ACVIM Large Animal Internal Medicine residency program at The Ohio State University. Concurrently, from 2001 to 2006, he was also enrolled in a PhD program and conducted several research projects in the field of equine cardiology, in cooperation with Dr. John Bonagura, Dr. Robert Hamlin, Dr. William Muir, and several members of the cardiology section. In 2006 he moved back to the Vetsuisse Faculty of the University of Zurich, where he was employed as a Senior Lecturer in the Equine Internal Medicine Section. In 2009 he completed his 'Habilitation' titled 'Advances in equine cardiology', before being appointed to a full professorship in Equine Internal Medicine in 2012. He is currently the director of the Clinic for Equine Internal Medicine and Chair of the Equine Department at the Vetsuisse Faculty of the University of Zurich. His academic and clinical interests include large animal and comparative cardiology, with emphasis on echocardiography, cardiac electrophysiology, cardiovascular pharmacology, and cardiac biomarkers.

Poppy Bristow BVetMed MVetMed DipECVS MRCVS

Poppy graduated from the Royal Veterinary College and completed a rotating internship followed by a Small Animal Surgical residency at the Queen Mother Hospital for Animals, RVC. Poppy became a lecturer in Small Animal Surgery at the University of Pennsylvania in 2014, gaining her European College of Veterinary Specialists diploma in 2015. Poppy returned to the UK to work in a private referral hospital before moving back to the RVC in 2017 to undertake a two year fellowship in cardiothoracic surgery. Poppy is now a Lecturer in Small Animal Surgery, sub-specialising in Cardiothoracics and works mainly on the open heart surgery programme.

Siddarth Sudunagunta BVetMed Cert AVP (VC) MRCVS

Sid graduated from the Royal Veterinary College in 2011 and worked for one and a half years in general practice before returning to the RVC for a rotating internship in small animal medicine and surgery. Following this, he spent two years in general practice before joining the cardiology department at the Small Animal Teaching Hospital, University of Liverpool as an intern in 2016. In September 2017 he began a cardiology residency, also at the SATH. During this time he also completed the CertAVP in Veterinary Cardiology.

Vi-Hue Tran MPhil

Vi-Hue Tran is a Research Associate based at the Institute of Cardiovascular Science in UCL, London. He has been involved in the morphology of the congenitally malformed heart since 1997, when he first started at the Heart and Lung Institute, Imperial College, London working under the expert supervision and guidance of Professor Robert Anderson as a research technician. In January 2008, he obtained his Master of Philosophy degree in cardiac morphology reviewing the inter-relationship between bronchial and systemic to pulmonary collateral arteries in hearts with tetralogy of Fallot and pulmonary atresia. His main role is providing education, teaching cardiac morphology to UCL MSc and medical students. He also gives cardiac morphology lectures and live video demonstrations within his unit and at various national and international venues such as the Echocardiography course in Cardiff and the European Association of Cardio-thoracic Surgery (EACTS) course in Windsor.

Tobi Wagner DrMedVet. Dip ECVIM-CA (Cardiology) MRCVS

Tobi graduated from the University of Munich in 2004, where he continued working in a veterinary echocardiography research project until 2005. After completing a Small Animal Rotating Internship in Georgia, USA, Tobi worked for one year with the busy cardiology department at the University of Giessen, where he gained a lot of interventional cardiology

experience. He then moved to the Royal Veterinary College, London, completing his cardiology residency and attaining his ECVIM-CA(Cardiology) Diploma in 2010. Following the residency, Tobi joined Southern Counties Veterinary Specialists where he has been working for the last 9 years. Tobi works full-time as a Specialist Veterinary Cardiologist, continuing to develop the very successful interventional service at SCVS and has recently started closing intrahepatic shunts using a novel technique. Tobi has recently become a Clinical Director at SCVS.

Chris Linney BVSc GPCertSAP CertAVP(VC) DipECVIM-CA (Cardiology) MRCVS

Chris qualified from the University of Liverpool in 2007. After 2 years in general practice, he returned to Liverpool University for further postgraduate training in cardiology. He worked at Liverpool Vet School for 5 years where he completed an Internship and residency in cardiology at Liverpool Vet School before promotion to Lecturer. After his time at Liverpool, Chris moved to the West Midlands to work alongside Mike Martin at the Veterinary Cardiorespiratory Centre. Chris now works at Willows Veterinary Centre and Referral Service where he is Head of Cardiology. Chris has a strong interest in management of congenital heart disease, cardiac interventions and congestive heart failure. He is currently studying for a Master's of Science in Interventional Cardiology.

Mike Martin MVB DVC MRCVS

Mike qualified from University College Dublin, Ireland in 1986. He gained the RCVS Diploma in Veterinary Cardiology and Specialist status in 1995 and has successfully been re-validated every five years ever since. Since 1992 to 2015, he ran his own private referral practice (The Veterinary Cardiorespiratory Centre, Kenilworth). Since October 2015 he moved his Cardiology Service into Willows Referral Centre, Solihull, UK. Since the beginning of 2018 he has again gone self-employed, providing consultancy work, training cardiac interventions at specialist centres around Europe, lecturing and education at CPD events around the world.

He has published over 40 scientific peer reviewed papers. He is author of two small textbooks: *Small Animal ECGs: An Introductory Guide* (3rd edition) and *Cardiorespiratory Diseases of the Dog and Cat* (2nd edition) published by Wiley-Blackwell. He is author of the chapter on 'Syncope' in the Textbook of Veterinary Internal Medicine (Ettinger, Feldman & Cote, 2016).

He is the proud recipient of some BSAVA awards: in 1993 the Dunkin Award, in 2000 the Melton Award, in 2006 the Petsavers Award, in 2010 the Dunkin & Blaine Awards and in 2017 the JSAP Achievement Award. He has been examiner for Diploma in Veterinary Cardiology for the Royal College of Veterinary Surgeons. He has been both Honorary Secretary and Chairman of the Veterinary Cardiovascular Society (UK).

Eoin Kilkenny DrMedVet MRCVS

Eoin graduated from Szent István University, Budapest in 2014 and initially worked in mixed practice in Ireland before moving to the UK to focus entirely on small animal practice. Eoin took up first opinion positions at hospitals in Bradford and Peterborough and in 2017 enrolled in a cardiology certificate. Following a rotating internship at Anderson Moores Veterinary Specialists, Eoin completed a cardiology internship at Lumbry Park Veterinary Specialists and has continued there as a cardiology resident since January 2019.

Maria Inmes Oliveira DVM MRCVS

Maria graduated from the University of Trás-os-Montes e Alto Douro (UTAD) in Portugal in 2014. It was there, and through externships undertaken at a variety of first opinion and referral centres, that she first recognised her passion for veterinary cardiology. After graduating she worked in a small animal hospital in the North of Portugal for a few months and then completed an academic small animal internship at the teaching Hospital of UTAD. She eventually moved to East Yorkshire where she worked in a first opinion small animal hospital for a year. Her interest for cardiology grew stronger and in order to pursue this, Maria undertook a rotating internship in Lumbry Park Veterinary Specialists, followed by a cardiology internship in Southern Counties Veterinary Specialists.

Maria has started a cardiology residency at the Royal (Dick) School of Veterinary studies at the University of Edinburgh in July 2019.

Gunter van Loon DVM PhD DipECEIM Assoc. Member ECVDI

Gunther van Loon graduated from Ghent University, Belgium, in 1992 and has worked at the Ghent University, Department of Large Animal Internal Medicine, ever since. In 2001 he finished his PhD on "Atrial pacing and experimental atrial fibrillation in equines". In 2004 he became Diplomate of the European College of Equine Internal Medicine and in 2011 Associate Diplomate of the European College of Veterinary Diagnostic Imaging. He is now Professor in Large Animal Internal Medicine at Ghent University and his major interests are cardiology (arrhythmias, electrophysiology, cardiac pacing, echocardiography, TDI, 2D ST), and thoracic and abdominal ultrasound. Areas of research include 2D Speckle Tracking, Tissue Doppler Imaging, cardiac biomarkers, assessment of arterial wall stiffness and great vessel pathology, atrial fibrillation. Gunther has published mainly in the field of equine internal medicine and cardiology and lectures regularly at national and international courses and conferences.

Omri Belachsen DVM CertAVP MRCVS

Omri graduated from the University of Košice, Slovakia in 2011. He spent two years in small animal practice in the UK before completing a rotational internship at Willows Referral Centre. He then joined Southern Counties Veterinary Specialists (SCVS) as a Cardiology intern. During this time, he completed an advanced veterinary practice certificate at Liverpool University. In March 2017, Omri began a cardiology residency at SCVS.

Hannah Stephenson BVMS (Hons) CertSAM Dip ECVIM-CA (Cardiology) MRCVS

Hannah graduated in 2005 and after a short career in mixed practice she undertook a rotating internship at the RVC and then a residency in cardiology at the University of Liverpool. During this time she developed an interest in heart disease in Great Danes, which developed in to a wider interest in breed-specific cardiac disease. During her recent stint on the VCS committee she became chair of the breed-related subcommittee of the VCS, fuelling her passion for promoting and developing heart testing in the UK. She currently runs a peripatetic cardiology consultancy and spends many fun-filled days as a small cog in the fascinating machine of dog and cat breeding.

Joanna Aitken BSc BVSc(Hons) GCLT FHEA MRCVS

Joanna graduated with 1st Class Honours from the University of Sydney, Australia, in 2013. Since graduation she has worked in both private practice and university settings across Australia and the United Kingdom. Her interest in cardiology, ECC and teaching has led to her current position as a Teaching Fellow at the University of Surrey and recognition as a Fellow of the Higher Education Academy. She is also currently a permanent member of the Vets Now Emergency team in Surrey. Joanna starts her residency under the American College of Veterinary Emergency and Critical Care at the University of Melbourne in January 2020.

Ela Barczak DVM CertAVP(VC) MRCVS

Ela graduated from Wrocław University Of Environmental And Life Sciences in 2013 with a strong interest in cardiology. In 2012 she undertook a three-month externship in Cardiology at the University of Liverpool Small Animal Teaching Hospital, via an Erasmus scholarship. After a short period of working in first opinion practice in Poland, she moved to the UK and was awarded a Leonardo da Vinci scholarship to undertake a six-month externship in cardiology at the University of Glasgow. Ela continued with a position in a very busy hospital in Wolverhampton and as a locum for Vets Now. During that time she completed the RCVS CertAVP in Cardiology. She is also actively involved in regional BSAVA as a volunteer. In

September 2019, Ela joined Small Animal Teaching Hospital, University of Liverpool, as the rotating intern and hopes to undertake a Residency program in the coming years

Dr. Luca Ferasin DVM PhD CertVC PGCert(HE) DipECVIM-CA(Cardiology) GPCert(B&PS) MRCVS

Luca Ferasin graduated with honours in 1992 from the University of Bologna. After 3 years research in endocrinology at the BBSRC Institute in Cambridge, he was awarded his PhD in 1996. Following 3 years as Assistant Professor at Padua University, Luca moved to Bristol University, where he taught cardio-respiratory medicine of the dog and cat for 7 years. In 2005-2007, he was Associate Professor in Cardiology at the University of Minnesota. Between 2008 and 2014 he ran his own cardiology consultancy company, comprising a mix of private clinical referral work, telemedicine and post-graduate teaching. He obtained the RCVS certificate in cardiology in 2001, the certificate in Teaching & Learning in Higher Education in 2002, the ECVIM diploma (cardiology) in 2004 and a Certificate in Business & Professional Studies in 2011. Luca has vastly contributed to the veterinary literature with articles, abstracts, and book chapters, including the chapter on coughing in the latest edition of Ettinger's textbook of Internal Medicine. He also acted as chairman of the ECVIM examination committee and was member of the RCVS examination board and BSAVA congress committee. Luca is a regular speaker worldwide. His main professional interests include feline cardiology, exercise physiology, as well as investigation and management of syncope and coughing.

Introduction to Congenital Echocardiography

Dr. Navroz Masani

Cardiff and Vale University Health Board, Cardiff, U.K.

Introduction to Congenital Echocardiography

Nav Masani
University Hospital of Wales

Echo in CHD

- Is the RA on the right?
- Does the RA connect to the RV?
- Is there an RV?
- Does the RV connect to the PA? or the Ao?
Or both? Or neither?

Standard "view by view" approach

vs

CHD "segment by segment" approach

Segmental approach to scanning

Atria

- venous connections, situs
- morphology, septum

Atrio-Ventricular connections

- con/discordant/double
- absent
- A-V valves

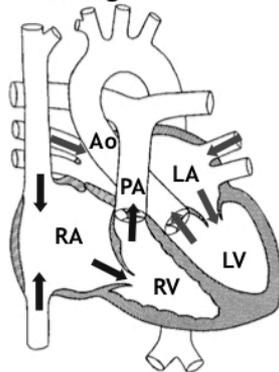
Ventricles

- morphology, function
- septum
- outflow tracts

Ventriculo-arterial connections

- con/discordant/double
- absent
- A-V valves

Great arteries



Segmental approach to scanning

Atria

- venous connections, situs
- morphology, septum

Atrio-Ventricular connections

- con/discordant/double
- absent
- A-V valves

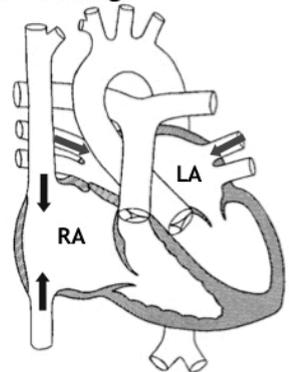
Ventricles

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Great arteries



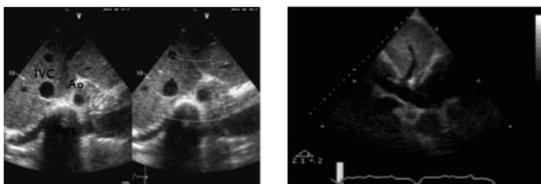
Atrial situs

Is the RA on the right?

Normal atrial situs (solitus)

- the IVC never joins the LA

Subcostal view



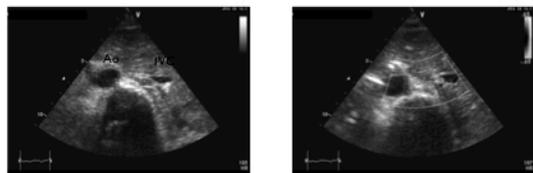
Situs Inversus

Is the RA on the right?

Normal atrial situs (solitus)

- the IVC never joins the LA

Subcostal view



Atrial isomerisms

Abnormal veno-atrial connections

Rotation abnormalities

- Midline liver
- Asplenia/polysplenia

Subcostal view

Pulmonary veins/atrial septum

Anomalous PV drainage

- sinus venosus ASD
- RH volume overload

Veno-atrial connections

Segmental approach to scanning

Atrio-Ventricular connections

- concordant (RA→RV)
- discordant (RA→LV; CCTGA)

- double inlet (eg "single ventricle")
- absent (eg Tricuspid Atresia)

- A-V valves (eg Ebstein's TV)

Ventricles

- morphology, function
- septum
- outflow tracts

Defining the ventricles

Right ventricle

- TV offset
- TV chordal attachment
- moderator band
- Trabeculations
- inflow/body/outflow

Defining the ventricles

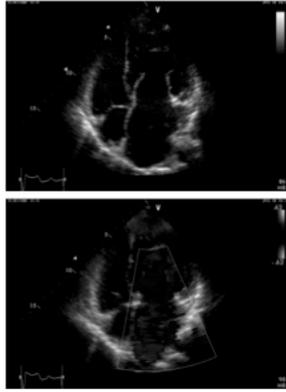
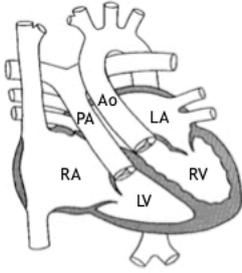
Left ventricle

- adjacent inflow/outflow tracts

Atrio-ventricular discordance

Congenitally corrected TGA

- A-V and V-A discordance
- Systemic RV
- "TR"



Atrioventricular Septal Defect

ASD

- partial AVSD
- primum ASD

VSD

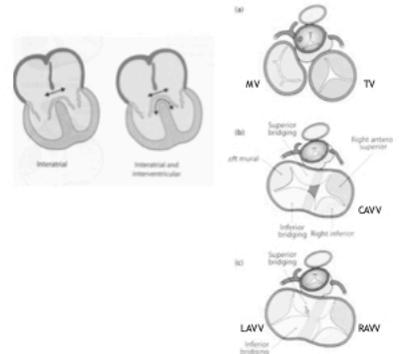
- intermediate AVSD
- complete AVSD

Common AV valve

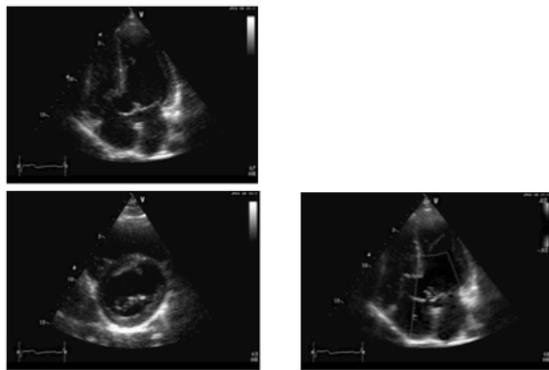
- AVV regurgitation

CHB

Downs syndrome



Complete AVSD

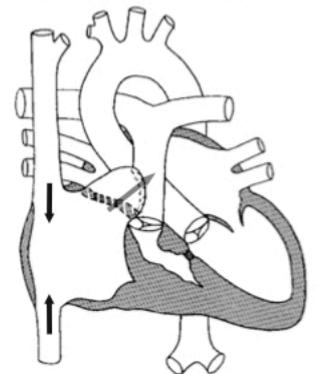


Absent RA-RV connection The Fontan circulation

Tricuspid atresia
Pulmonary atresia

Single ventricle

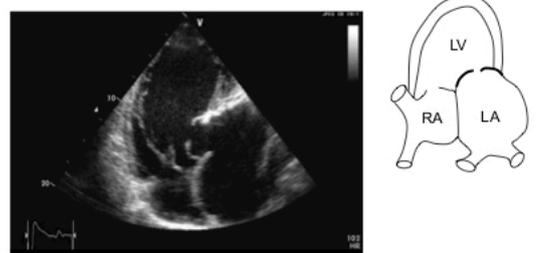
RA connection to PA
IVC & SVC connection to PA



Absent RA-RV connection Tricuspid atresia



Double inlet AV connection



Segmental approach to scanning

Atria

- venous connections, situs
- morphology, septum

Atrio-Ventricular connections

- con/discordant/double
- absent
- A-V valves

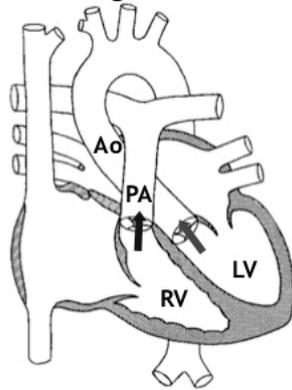
Ventricles

- morphology, function
- septum
- outflow tracts

Ventriculo-arterial connections

- con/discordant/double
- absent
- V-A valves

Great arteries



Ventriculo-arterial discordance

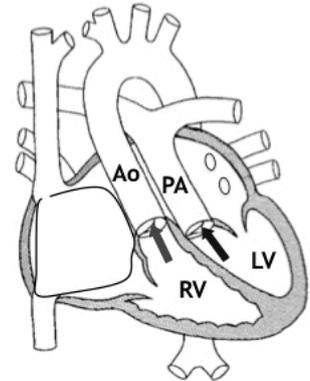
Transposition of the great arteries

TGA with "atrial switch"

- Senning/Mustard op

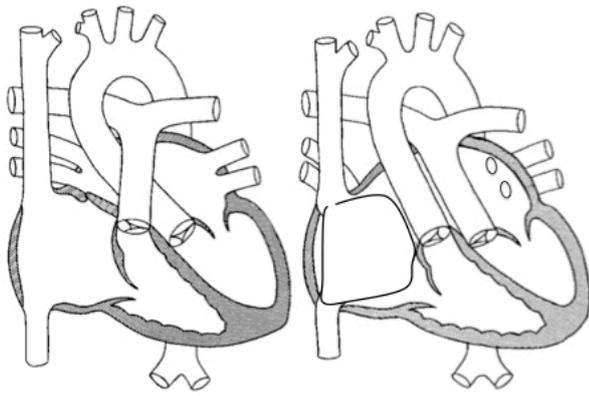
long term sequelae

- systemic RV dysfunction
- R AV valve regurgitation
- baffle obstruction
- baffle leak



Criss-cross concordant

Double barrel discordant



Defining the great arteries

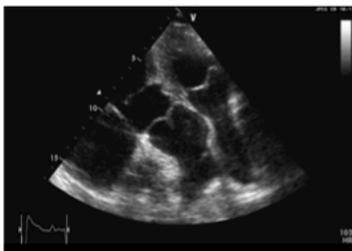
PA branches early cf. the aorta

Normal GA's cross

Malposed GA's run parallel



V-A discordance (transposition)



TTE: segmental approach

Structure by structure

Subcostal-apical-parasternal-suprasternal

Connections

- Normal (concordant)
- Discordant
- Absent
- Double

Introduction to Equine Congenital Echocardiography

Dr. John Bonagura & Dr. Colin Schwarzwald
NCSU, Raleigh, USA & University of Zurich, Switzerland

The equine heart develops from an embryonic tube to a four-chambered pump secured by four valves and partitioned to serve the high resistance systemic and low resistance pulmonary circulations. The embryogenesis of the heart is complicated, and occasionally the processes controlling normal cardiac development fail. Horses with mild to moderate congenital heart disease (CHD) often tolerate the disease well, though reproductive value of the animal surely suffers. Regrettably, the management options for moderate to severe CHD in horses are severely limited compared to the situation in children and in dogs and cats. Cardiac surgery is rarely performed in horses and even image-guided interventions are limited and challenging in this species, largely due to their rapid growth and large size.

The main issues of concern for the equine practitioner are rendering an accurate diagnosis, staging the severity of disease, and directing an accurate prognosis for life and use. Cardiologists who focus on small animals can often add value to an echocardiographic consultation by emphasizing the principles of echocardiographic diagnosis, while recalling that large animals sometimes survive to maturity with complex CHD. These notes provide a basic framework for understanding and diagnosing the most important cardiac malformations in horses. The nomenclature used in these notes is “traditional” and the authors accept there are alternative words and terms used for a number of defects and anatomic landmarks. The goal of our presentation is the demonstration of a common and some uncommon cardiac malformations we have observed in foals and mature horses.

CLINICAL PATHOPHYSIOLOGY OF CONGENITAL HEART DISEASE

Some Aspects of Normal Cardiac Development

It is helpful to understand the fundamental circulatory patterns of the fetus as this can be especially relevant to echocardiographic examination during the initial days after foaling. There are two fetal circulations: one serving the embryo and the other communicating with the placenta. It is helpful to consider the maternal placental circulation as a “bypass” for the fetal lung. Functionally there are *two right-to-left shunts*: one across the foramen ovale from right to left atrium, and the other from pulmonary artery (PA) to descending aorta across the ductus arteriosus. Inasmuch as fetal lungs are collapsed, pulmonary vascular resistance is high, and pulmonary blood flow minimal, the desaturated blood returning from the fetal tissues is collected in the cardinal venous system and enters the sinus venosus and right atrium. This blood is largely earmarked for the right ventricle and pulmonary artery, but

pulmonary flow is diverted through the ductus arteriosus to the descending aorta and placenta where it is oxygenated. Well-saturated blood returning across the umbilical veins is delivered by the caudal vena cava to the right atrium where it preferentially crosses the foramen ovale to enter the left atrium, left ventricle and ascending aorta.

These patterns change dramatically with foaling. As the lungs expand, pulmonary vascular resistance falls, and pulmonary blood flow increases. The resultant increase in left atrial pressure *functionally closes the foramen ovale* within the first 24 to 48 hours of life, as confirmed by contrast echocardiography in full-term foals. Similarly, inhibition of local prostaglandins leads to *functional closure of the ductus arteriosus* within 72 hours in most full-term foals (based on auscultation). Persistence of right-to-left shunts, especially at the level of the foramen ovale, can occur in premature foals or those suffering from severe pulmonary disease with associated pulmonary hypertension. In these cases, shunting across the foramen ovale represents an additional mechanism for arterial desaturation and tissue hypoxia. This shunting can be confirmed using saline contrast echocardiography.

The underlying genetic factors guiding normal development of the heart and those leading to cardiac malformation are poorly understood in horses, but are likely related to genetics, infections, exposure to toxins, and other environmental factors. Cardiac morphogenesis is complicated and will be reviewed by other speakers at this conference. It is helpful to understand elementary aspects of cardiac development, especially as these pertain to CHD. The primitive cardiac tube must loop (normally this is a d-loop, to the right), septate, and develop valvular structures that foster unobstructed, one-way blood flow through the heart. Especially relevant to equine CHD are the septation of the atria and their connection to the ventricles; the formation of the ventricular septum; the development of cardiac valves; and the origination and separation of the great vessels.

After looping and migrating the primitive atrium dorsal to the ventricles, the right and left atria are created by incorporation of the right horn of the sinus venosus and development and fusion of the septum primum and septum secundum. Failure of normal development in these tissues can lead to an *atrial septal defect (ASD)*, which is typically designated by the location of the hole and the deficient tissue. Secundum and primum ASDs are the types most often reported in horses. The sinus venosus ASD and the unroofed coronary sinus (connecting the coronary sinus to the left atrium) are considered rare in this species.

The *foramen ovale*, a normal fetal structure, is located slightly dorsally and near the middle of the atrial septum. This creates a passageway for blood flow from right atrium to left atrium and preferentially carries oxygenated caudal vena caval flow into the left atrium to supply the fetal systemic circulation. The equine foramen ovale appears as a fenestrated finger cot at autopsy and appears echocardiographically in newborn foals as a mobile, undulating membrane adjacent to the left surface of the atrial septum. As noted above, this inter-atrial pathway can persist in foals with pulmonary hypertension and elevated right atrial pressures due to pneumonia, immature lung, or persistent fetal circulation.

The endocardial cushions help to close the gap between the atrial septum and developing ventricular septum. These tissues also contribute to the atrioventricular septum, that segment of myocardium filling the gap between the septal (anterior, cranial) mitral valve insertion on the left side and the (more ventral) septal tricuspid valve insertion on the right side. The ventral atrial septum connects to the dorsal parts of the ventricular septum by growth and differentiation of endocardial cushions. These swellings also contribute to formation of the atrioventricular valves. Defective differentiation of the septum primum or endocardial cushions causes various combinations of an ostium primum ASD, an inlet septal ventricular septal defect (VSD), malformation of the atrioventricular valves, or common atrium with a single atrioventricular valve. Most of these CHDs are termed incomplete or complete *atrioventricular septal defects (or endocardial cushion defects)*.

The ventricular septum is a complicated partition that includes a number of regional parts: (1) a small membranous portion located ventral to the aortic root and slightly craniodorsal to the insertion of the tricuspid valve (readily palpated at autopsy by opposing fingers in the aorta and RV and a false source of septal dropout when performing 3D echocardiography); (2) an “inlet” septum immediately ventral to the septal tricuspid valve leaflet; (3) an extensive, curved, muscular or trabecular ventricular septum; and (4) a dorsal outflow septum that separates the subaortic outflow tract from the subpulmonic infundibulum. Defects within these segments are correspondingly named perimembranous, inlet, muscular (trabecular), and subarterial (subpulmonic) VSDs and are further described below. Insufficient development of these embryonic components result in a *VSD, the most common cardiac malformation in horses*.

The aorta and pulmonary artery begin as a single vessel originating in the conus arteriosus. This common vessel, the truncus arteriosus, is eventually partitioned by migration of the conus tissues dorsally and development of subarterial outflow tracts and a spiraling arterial septum. Twisting of this septum produces appropriate alignment (concordance) of the great vessels to their respective ventricular outflow chambers. There has been some controversy regarding naming and development of these segments as discussed in papers by Anderson and colleagues. Malformation of conus or spiral septal tissues can produce complicated congenital heart defects in the horse. These include *tetralogy of Fallot, pulmonary atresia, persistent truncus arteriosus, and double outlet right ventricle*. The aortic arch is another location for potential malformation in the horse as tubular hypoplasia and interruption of the aorta proximal to the ductus arteriosus have been recognized in horses. The descending aorta and pulmonary artery are normally connected in the fetus by the ductus arteriosus (also called the arterial duct), which carries fetal blood from pulmonary artery to descending aorta.

In terms of other congenital heart defects so common in dogs and cats, a number of these are comparably rare in horses. *Persistent patency of the ductus arteriosus (PDA or patent arterial duct)* is unusual, especially as an isolated lesion, although it is persistent in most cases of pulmonary atresia or interruption of the aortic arch. Similarly, *aortic (subaortic)*

stenosis and pulmonary stenosis (PS) are rare, although PS has been reported in horses. *Malformations (“dysplasia”) of the atrioventricular valves* are probably more common than we realize in horses and it is challenging to recognize mitral or tricuspid valve malformations in younger horses with mild valvular regurgitation. Tricuspid valve malformations can range from trivial lesions causing mild regurgitation to severe tricuspid stenosis or atresia of the RV inlet. Apical displacement of the tricuspid valve, similar to Ebstein’s anomaly in children, has been recognized (but not confirmed by EP studies).

Etiology, Prevalence and Classification of Equine Congenital Disease

A failure of normal fetal development can result in congenital malformation of the heart, great vessels, or veins. The etiology of CHD in most cases is uncertain. Causes might include genetic liability or defective genetic control, fetal infection, drug administration to the mare, or fetal exposure to toxins *in utero*. Unlike the situation in dogs, where there are very clear breed associations to CHD, there is scant information about the inheritable nature of CHD in horses. Certainly, it would seem that Arabian horses and perhaps the Standardbred, Quarterhorse, and Welsh Mountain pony are at risk for VSDs. However, no comprehensive surveys of breed associations with cardiovascular malformations have been published. The exact *prevalence* of CHD in the general equine population is unknown, but cardiac malformations were reported in 3.5% of 608 foals examined at necropsy (Crowe and Swerczek, 1985) and others have cited lower prevalence (0.2% or less; see references). Lastly, the prevalence of congenital defects of the impulse forming and conduction system in horses, as with accessory A-V pathways, is unknown.

Theoretically, CHD can involve anomalies of: 1) venous drainage, 2) atrial situs or septation, 3) atrioventricular connection, 4) ventricular development (including formation of the two atrioventricular valves), 5) ventricular outflow tracts, 7) semilunar valves, and 8) great vessels. Furthermore, abnormal segmental connections might occur leading to “discordance” in the path of systemic or pulmonary venous return relative to the pulmonary artery or aorta (Anderson, 2000). These abnormalities include transposition of the great arteries and double-outlet right ventricle, wherein both great vessels exit the right ventricular cavity. The segmental approach to congenital heart disease was popularized by Van Praagh and expanded to include the morphologic (building blocks) of Anderson and colleagues. These approaches have also been applied to the horse by one of the authors (Schwarzwald, 2008). Cardiologists often classify congenital defects using these approaches this way, especially for complex CHD. However, for most equine practitioners, it is more practical to consider the simpler and more common defects by name, accepting that complex malformations do occur. Realistically, the majority of foals with CHD will be affected by lesions causing shunting of blood at the atrial or ventricular levels and less often by malformations of the cardiac valves or great vessels.

The isolated VSD adjacent to the membranous septum represents the most common CHD of the horse. Ventricular septal defect is a component of tetralogy of Fallot, persistent truncus arteriosus, and pulmonary atresia. Most rare forms of equine CHD such as tricuspid valve atresia, double-outlet right ventricle, transposition of the great vessels, and univentricular heart syndromes are also associated with a VSD. The isolated ASD is rare in horses, but secundum defects, primum defects, and atrioventricular septal defects have all been observed. Patent ductus arteriosus as a single defect is rare in horses; however, a PDA can accompany other defects, including pulmonary atresia. In terms of valvular malformations, tricuspid dysplasia and atresia; bicuspid (stenotic) pulmonary valve and pulmonary atresia; fenestrated and quadricuspid aortic valve; and mitral valve dysplasia have been observed. Other reported defects include hypoplastic left heart syndrome, single ventricle/univentricular heart, endocardial fibroelastosis, aortic origin of the pulmonary artery, various coronary artery anomalies, and segmental hypoplasia or interruption of the aortic arch.

Clinical Pathophysiology of Shunts

Fundamental to understanding cardiac malformations in the horse and echocardiographic changes that develop within the cardiac chambers is appreciation of potential responses of the heart and circulation to a shunt. Shunting can be defined as abnormal deviation of blood flow between the systemic (left) and pulmonary (right) sides of the circulation. Possibilities include left-to-right, right-to-left, and bidirectional shunting.

Systemic to pulmonary (left-to-right) shunting is the expected consequence of an ASD, VSD, or PDA so long as systemic pressures and resistance exceed that on the right side. Even in cases of abnormal ventricular-arterial development, as with persistent truncus arteriosus, double outlet right ventricle, or univentricular heart, the clinical findings of a left-to-right shunt can predominate unless there is obstruction to blood flow at the pulmonary valvular or arteriolar levels. The actual shunt volume carried to the lungs depends on the caliber (or “restrictive” nature) of the lesion orifice and the relative resistances between the systemic and pulmonary circulations. Shunting should not be significant immediately after foaling because pulmonary vascular resistance is still relatively high compared to systemic arterial and left ventricular pressures. Eventually left-to-right shunts increase pulmonary arterial flow and augment pulmonary venous return. Small shunts are easily handled through left-sided dilation and eccentric hypertrophy. When the pulmonary to systemic flow ratio ($Q_p:Q_s$) is less than about 1.5:1, the shunt is usually well tolerated. Regardless, a 1.5:1 left-to-right shunt ratio will still result in overt dilation of the left atrium and ventricle as well as the pulmonary trunk. With greater shunts there is more noticeable volume overload of the left heart and the higher the potential for left-sided or biventricular congestive heart failure (CHF) from chronic ventricular systolic or diastolic dysfunction.

Pulmonary hypertension can develop in the setting of left-to-right shunting from combinations of increased flow, inadequate development of pulmonary arterioles, arteriolar remodeling, and left ventricular dysfunction. Thus, consequences of significant left-to-right shunting can include any of the following: exercise intolerance, tachypnea, pulmonary edema, respiratory distress, pulmonary hypertension, atrial fibrillation, pleural effusion, jugular venous distension, or ventral edema. The foal might be smaller than expected, and often has a history of antibiotic therapy for presumed bouts of “pneumonia.”

Right-to-left shunting stems from a different pathophysiologic state and produces a different clinical presentation. When a shunt is complicated by a right-sided obstruction adjacent to or downstream from the defect, right-to-left shunting will develop as right-sided pressures exceed those on the left side. This can occur in the neonate, as in tricuspid valve atresia with ASD or pulmonary valve atresia with VSD. Conversely, elevated right-sided resistance can develop more chronically from pulmonary vascular disease and pulmonary hypertension. For example, a large left-to-right shunt can induce medial hypertrophy and intimal thickening of pulmonary arterioles elevating pulmonary vascular resistance and decreasing the left-to-right shunt. Though uncommon, the resultant pulmonary hypertension might become quite severe (Eisenmenger’s pathophysiology) and reverse the shunt to right-to-left. In these cases, the left heart chambers are small and right ventricle is hypertrophied with thickened walls, to achieve systemic blood pressure. The ventricular septum is likewise affected, especially the “RV” portion.

The entrance of desaturated blood from the right to left side of the circulation causes hypoxemia. Thus, the consequences of a significant right-to-left shunt can include low arterial pO₂, tissue hypoxia, cyanosis, exercise intolerance, mild to moderate polycythemia (erythrocytosis), hyperviscosity of blood, and stunting of growth. Congestive heart failure is rare, but sudden death can occur, presumably from arrhythmia. The degrees of hypoxemia and cyanosis in a right-to-left shunt depend on overall pulmonary blood flow; this is a pivotal concept. If pulmonary flow is diminished, as with tetralogy of Fallot, tricuspid atresia, or pulmonary atresia with VSD, the contribution of the left ventricle to aortic flow (and thus oxygenation) is low. There are instances where right-to-left shunting develops but in the setting of increased pulmonary blood flow, as in some cases of truncus arteriosus or double outlet right ventricle. These lesions cause less hypoxemia because the amount of oxygenated blood reaching the left ventricle is normal to increased. In such situations, cyanosis from right-to-left shunting might be negligible and the clinical condition of left sided or biventricular congestive heart failure can predominate.

Clinical Signs and Diagnostic Approach

Although CHD is relatively uncommon in horses, a diagnosis of cardiovascular malformation can significantly impact the value of the foal and cast doubt on the genetic reliability of the

mare or sire. The clinical impact of a congenital heart defect in an individual horse can vary widely. Some malformations are lethal *in utero* or immediately following parturition; whereas, others cause signs only later in life. Other defects are compatible with a near-normal life expectancy but limit maximum performance and reproductive value. Mild or trivial defects do not affect performance and can escape detection.

The diagnosis of CHD in horses requires an index of suspicion because no clinical findings are specific for cardiac malformation. Certainly, a cardiac murmur is the most common clinical finding in CHD, but other signs such as reduced performance, tachypnea, respiratory distress, or cyanosis might be observed in symptomatic animals. In some severe cases, atrial fibrillation or signs of congestive heart failure (CHF) are evident.

The timing and location of a heart murmur associated with CHD varies with the defect (as discussed later). Ejection murmurs are ubiquitous in horses, and these can pose a diagnostic dilemma. An ejection murmur loudest over the left-cranial cardiac base might be evident with some cardiac defects, but this murmur is even more common in foals and adults without heart disease. In most cases, a left basilar ejection murmur simply represents a functional (flow) disturbance unrelated to heart disease. The usual functional murmur builds in midsystole, ends before the second sound, and varies in intensity with sympathetic activity. Ejection murmurs usually become louder briefly following exercise or after a startle reaction. Echocardiography and Doppler studies should be normal in the horse with a functional murmur. Conversely, a cardiac murmur with one or more of the following characteristics is more likely to be associated with structural heart disease: 1) moderately loud (>3/6) murmur, 2) associated with a precordial thrill, 3) holo- or pansystolic in timing, 4) diastolic or continuous in timing, or 5) loudest over the palpable left apex or over the right side of the thorax. In these instances, echocardiography with Doppler is likely to identify a congenital or acquired lesion of the heart, or at the least, a flow disturbance causing the murmur (such as tricuspid regurgitation in high-performance horse).

Although respiratory signs related to heart failure or to pulmonary hypertension can develop consequent to CHD, these clinical signs are far more likely to stem from airway, lung and pleural diseases. Cyanosis in a foal, especially one without respiratory distress, should alert the clinician to the possibility of CHD with right-to-left shunting; accepting primary lung disease first must be excluded.

The finding of CHF, especially in a foal or yearling, should raise the possibility of CHD. Differential diagnosis should include acquired diseases such as bacterial endocarditis, pericarditis, myocarditis, and tachyarrhythmia-induced cardiomyopathy (from relentless supraventricular or ventricular tachycardia). Infrequently, CHD leads to heart failure in a mature horse, and in these cases, the diagnosis of a congenital etiology is usually accidental, made during echocardiographic examination. Right-sided CHF is a straightforward diagnosis characterized by resting tachycardia, bilateral jugular venous distension, jugular pulses, and ventral edema. If there is pulmonary hypertension, the pulmonic component of the second sound might be tympanic. Clinical findings of left-sided CHF include resting tachycardia, loud

third heart sound, and auscultatory or percussion findings of pulmonary edema, pulmonary hypertension, or pleural effusion. Atrial fibrillation and cardiac murmurs are other common findings. Biventricular CHF is not uncommon in horses with cardiac failure and is most often observed when the combination of a left heart lesion, pulmonary hypertension, and atrial fibrillation occurs.

The most important diagnostic studies in foals with suspected CHD include 1) palpation of the arterial pulse for rate, rhythm and strength, 2) examination of the precordium for thrills or cardiac displacement, 3) careful auscultation of the heart, lungs and pleural space, 4) inspection of mucous membranes, 5) assessment of the jugular venous pulse and pressure, 6) thoracic radiography, thoracic ultrasonography, or airway endoscopy/cytology when there are signs of lung or pleural disease, and 7) echocardiography complemented by Doppler studies. The latter examination represents the noninvasive gold standard for diagnosis of CHD. Should a cardiac malformation be found in a foal, the clinician should at the least auscultate the mare for evidence of CHD. Congenital heart disease, particularly larger or complex defects, should be evaluated on a case-by-case basis in consultation with a specialist experienced in equine cardiology.

ECHOCARDIOGRAPHY IN EQUINE CONGENITAL HEART DISEASE

Atrial Septal Defects

Atrial septal defects are uncommon in foals. An ASD can involve different portions of the atrial septum. Pathologically and by 2D echocardiographic examination ASDs are classified as secundum, primum, and sinus venosum (rare). Secundum defects are located in the fossa ovalis and indicate failure for the septum secundum to properly form. A patent foramen ovale is not the same thing, as the septal tissue is present but higher pressures in the RA have maintained patency of this structure. Primum defects are in the ventral atrial septum. An incomplete atrioventricular septal defect involves a primum ASD with most showing a bridging (common) septal leaflet that serves both ventricles and located on the same plane. Normally, there is a small offset between the mitral (dorsal) and tricuspid (ventral) insertions into the atrioventricular septum. A complete atrioventricular septal defect (AVSD) includes a primum ASD, inlet VSD (directly below the septal TV leaflet), and bridging or common AV valve leaflets (also called an “endocardial cushion defect” or “complete AV canal”). Abnormal atrial septal patency is more likely to be observed with complex congenital cardiac defects, particularly when there is tricuspid or pulmonary atresia.

In the case of a large ASD, left-to-right shunting leads to right-sided volume overload and pulmonary overcirculation with dilation of the pulmonary trunk and main branches. If the ASD is isolated, the left atrium rapidly decompresses and will not typically be enlarged. Most isolated ASDs are visible echocardiographically with 2D imaging in complementary planes;

Doppler echocardiography or saline contrast echocardiography can confirm the direction of the shunt and estimate its severity. As atrial pressures are low on each side, velocity of shunting is low, affected by ventilation, and can be confused with venous return from the caudal vena cava. Atrial fibrillation has been observed in conjunction with ASD and further affects the flow pattern. In cases of complete AVSD there is atrioventricular valve regurgitation that can confusingly start in the contralateral ventricle.

Ventricular Septal Defect

The location of a VSD depends on the embryogenesis of the lesion and influences the designation and even the clinical manifestations of the defect. The nomenclature of VSDs is confusing but most can be remembered by considering the main components of the normal ventricular septum previously described. At autopsy the location of the defect is further defined by the surrounding tissue and fibrous connections. These are also relevant to echocardiographic diagnosis.

In most cases, a VSD is located dorsally (“high”) on the ventricular septum, below the right or noncoronary cusp of the aortic valve on the left side, and craniodorsal to the septal tricuspid leaflet on the right. Such defects maintaining fibrous continuity to both aortic and tricuspid valves are generally termed “perimembranous” or sometimes “paramembranous” or just “membranous”. The older terminology called these holes “subcristal” because the VSD is located caudoventral to the muscular cristae supraventricularis (supraventricular crest) that separates the right ventricular inlet and outlet. However, a very large perimembranous defect can extend under the tricuspid valve towards the inlet septum or advance across the supraventricular crest towards the outlet septum. The septal defects associated with tetralogy of Fallot and with pulmonary atresia are usually very large and fall into the latter appellation.

It is not uncommon for the aortic root to be displaced ventrocranially and to straddle the defect, creating a “malalignment” VSD. This is always evident with tetralogy of Fallot and pulmonary atresia with VSD, but it also can be seen with large perimembranous defects. Malalignment is clinically significant because the right or noncoronary cusp of the aortic valve is likely to prolapse into the defect leading to aortic regurgitation. A less common location for a VSD is immediately ventral to the septal tricuspid valve within the muscular septum. Such “inlet” VSDs are typical of complete atrioventricular septal defects and usually related to a primum ASD (a common atrioventricular canal type defect) that often involves malformation of the septal components of the atrioventricular valves with a “bridging” leaflet. A subarterial VSD communicates with the outlet portion of the ventricular septum directly below the pulmonary valve is variably termed an “outlet,” “supracristal”, “subpulmonic,” “juxta-arterial”, and sometimes “doubly-committed” VSD. The latter term is sometimes confined to defects where there is fibrous continuity with both arterial valves as opposed to just the aortic valve. This lesion places the aortic valve at risk for root or leaflet

prolapse associated with aortic regurgitation. Muscular (trabecular) defects or multiple VSDs are surrounded completely by ventricular muscle on the right side. Some of these are apical and challenging to visualize without color Doppler imaging. These can vary in size from small to enormous.

Many VSDs close spontaneously in people, and one of the authors has observed this with a small apical muscular VSD in a horse; but whether or not this is common in horses is unknown. However, the flow across a VSD can be diminished by imposition of a cardiac valve. For example the rim, or even a major portion of a VSD, might be occluded by scar tissue that ensnares the septal tricuspid leaflet, rendering the defect functionally smaller and possibly creating a hyperechoic aneurysm on the right septal surface. Large defects associated with malalignment of the ascending aorta to the upper border of the remaining septum are often associated with prolapse of an aortic valve leaflet (or of the aortic root) into the defect. Aortic prolapse can effectively close even a large VSD, but at the risk of permitting chronic aortic valve insufficiency over time.

Two-dimensional echocardiography (2DE) and color Doppler imaging establish the diagnosis. M-mode studies and spectral Doppler examinations are useful for assessing the hemodynamic burden of the defect. The 2D imaging successfully delineates the VSD, except for very small or apical defects, provided sufficient imaging planes are obtained. It is important to collect long axis images of the left ventricular outflow tract and aortic valve, as well as a short axis images at the level of the left ventricular outflow tract, just below the aortic leaflets. The typical perimembranous defect appears under the aortic valve and adjacent to the septal leaflet of the tricuspid valve. A true defect is characterized by a relatively echogenic tissue interface; whereas, an area of false echo dropout tends to be gradual. Most ventricular septal defects can be imaged in orthogonal (long axis/short axis) planes. The right coronary artery of the horse is relatively large and might be confused with a subarterial (supracristal) VSD. It also should be noted that an inlet VSD (ventral to the septal tricuspid valve) might not be seen in these standard imaging planes. Tipped or oblique views that show both atrioventricular valves might be required. Similarly, finding a muscular, apical, or small subarterial defect requires more imaging experience and is greatly assisted by color Doppler studies. With the introduction of 3D imaging to horses, it is common for the normal membranous septum to “drop out” so this can create a false-positive diagnosis.

Attempts should be made to identify the largest diameter of the defect in complementary planes, and compare this with the size of the aorta, as orifice size is an important prognostic factor. While there are limitations to 2D sizing of the VSD (as the defects are rarely round), a defect exceeding 2.5 cm in diameter or a VSD/aortic root diameter of >0.4 identifies a larger defect with greater likelihood of clinical signs. Furthermore, 2D or M-mode evidence of left sided cardiac dilatation, right ventricular enlargement, or marked dilation of the main pulmonary artery suggest a hemodynamically significant VSD and one more likely to impact performance or survival.

Identification of shunting across a VSD is best made using color Doppler studies. There will typically be high-velocity, turbulent flow entering the right ventricle during systole with low velocity, uniform color shunting noted during diastole. Color Doppler imaging is extremely helpful for identifying a VSD with atypical location, and also can identify aortic regurgitation in some horses. Continuous wave Doppler is used to estimate the pressure difference between the two ventricles, as velocity (V, in m/sec) is proportional to the instantaneous pressure difference across the ventricles ($\Delta P=4V^2$). A relatively small VSD is “restrictive” to flow and the peak shunt velocity will generally exceed 4.5 m/sec, assuming proper alignment to shunt flow. Should pulmonary hypertension develop related to increased pulmonary flow, left heart failure, or pulmonary vascular injury, the velocity of left-to-right shunting will be lower, and a high velocity jet of tricuspid regurgitation (>3.5 m/sec) might be identified.

Potential outcomes of the isolated VSD include: 1) tolerance of the lesion; 2) partial or complete closure of a VSD by adherence of the septal tricuspid leaflet, fibrous tissue, right ventricular hypertrophy, or aortic valve prolapse; 3) progressive aortic regurgitation, 4) atrial fibrillation; 5) left sided or biventricular CHF, 6) pulmonary hypertension (with left to right shunting); or 7) reversal of the shunt with development of arterial hypoxemia and cyanosis. The last of these is caused by either severe pulmonary vascular disease (Eisenmenger’s pathophysiology) or fibromuscular obstruction in the right ventricular outlet leading to subpulmonic stenosis or double chamber right ventricle. The horse with a relatively small diameter perimembranous defect, high-velocity left-to-right shunt, mild cardiomegaly, relatively normal right ventricular cavity, and normal heart rhythm probably has a restrictive VSD that will be well tolerated. Most of these animals can perform sufficiently in the show ring, as a hunter-jumper, or even as an endurance or racehorse. Large defects that are associated with echocardiographic evidence of moderate to severe cardiomegaly, right ventricular hypertrophy, aortic root prolapse, or Doppler evidence of pulmonary hypertension are prone to complications and carry a less favorable prognosis for performance or life regardless of current clinical signs. Superimposition of atrial fibrillation can markedly decrease cardiac function and promote CHF.

Tetralogy of Fallot

The tetralogy of Fallot is one of the more common congenital cardiac anomalies in foals responsible for right-to-left shunting with arterial desaturation and cyanosis. The four lesions are large perimembranous VSD, right ventricular outflow tract obstruction, cranial and rightward (dextro-) positioning of the aorta with overriding of the septal defect, and right ventricular hypertrophy. The lesion is fundamentally one of abnormal septation. Outflow obstruction can be due to subvalvular fibromuscular obstruction, valvular pulmonic stenosis, or hypoplasia of the pulmonary trunk. Ventricular hypertrophy is caused by right ventricular outflow obstruction and the large, unrestrictive, VSD that functionally creates a

“common ventricle.” Blood leaves the heart along the path of least resistance, and pulmonary flow depends on the severity of right ventricular outflow tract stenosis. As previously discussed, the degree of cyanosis and severity of clinical signs depends on the volume of blood traversing the lungs. In some horses, a PDA is also present (pentalogy of Fallot) and this defect reduces signs by increasing pulmonary flow, left heart filling, and systemic arterial hemoglobin saturation.

Echocardiographic evaluation is diagnostic and reveals a large, unrestrictive VSD, right ventricular outflow tract obstruction, malalignment and overriding of the aortic root, and right ventricular hypertrophy. These are readily observed using conventional long and short-axis images from the right side, optimized for the aorta, tricuspid valve, and right ventricular outflow tract. Shunting can be identified by color Doppler or saline contrast echocardiography. Bidirectional, low-velocity shunting is typical. An injection of agitated saline into the jugular vein results in a positive contrast echocardiogram and similar to the color Doppler study discloses the right-to-left or bi-directional flow across the VSD. Conventional spectral Doppler studies can be used to delineate the shunt (typically bidirectional, low velocity flow of < 2 m/sec) and right ventricular outflow obstruction (high velocity flow exceeding 4 m/sec).

Pulmonary Atresia with Ventricular Septal Defect

Pulmonary atresia is along the spectrum of tetralogy of Fallot. The defect is comparably rare, having been observed most often in Arabian foals but seen in other breeds as well. These are the lesions: 1) the right ventricular outlet is not patent, 2) the right ventricle is hypertrophied, 3) a large malalignment VSD is present, and 4) the fetal truncus arteriosus has been partitioned so unequally that the aorta is markedly dilated and the pulmonary trunk is represented by a small strand of tissue without a patent connection to the RV. This last lesion cannot be identified by echocardiography and without careful ultrasound studies (or necropsy dissection) of the pulmonary circulation, the dilated aorta can be mistaken for a persistent truncus arteriosus, hence the moniker "pseudotruncus arteriosus." Owing to the atretic pulmonary valve, pulmonary blood flow must be derived either from a PDA or systemic vessels off the aorta. In the latter instance, the systemic collaterals are usually from bronchial arteries.

The diagnosis of pulmonary atresia is usually prompted by the findings of cyanosis, cardiac murmur, and stunting in a foal or weanling and is confirmed by echocardiography. Careful echo studies can identify the main lesions: concentric hypertrophy of the right ventricle, unrestrictive VSD, dilated malalignment great vessel (the aorta), and inability to identify the pulmonary valve in the rudimentary right ventricular outflow tract (though a small pouch might be seen). Careful ultrasound examination of the ascending aorta and aortic arch from the right and left sides of the thorax will fail to reveal a normal origin for the pulmonary trunk; however, the bifurcation of the pulmonary artery can usually be found from a cranial imaging position, and continuous flow into the vessel documented by pulsed wave Doppler

echocardiography. The origin of pulmonary flow is typically from the ductus arteriosus or a large collateral systemic artery.

Persistent Truncus Arteriosus

The clinical and echocardiographic findings of persistent truncus arteriosus can resemble those of pulmonary atresia, but this depends on the magnitude of pulmonary blood flow. In this condition, the fetal truncus never partitions and both ventricles continue to develop, communicating with the truncus arteriosus across a large VSD. Systemic, coronary, and pulmonary arterial flows each arise from the truncus, which is guarded by a truncal valve (which can be incompetent or stenotic and consist of three or four leaflets). The truncal valve can appear to be in fibrous continuity with both mitral and tricuspid valves.

Pulmonary blood flow originates from one or more pulmonary arteries, which arise directly from the truncus arteriosus in one of three general ways (Types I, II, III). With careful ultrasound examination, the origin of the pulmonary arteries might be identified provided lung interference does not preclude evaluation of this area. Again, this condition must be distinguished from pulmonary atresia with VSD. Most connections are Type I in foals (a caudal or posterior positioned single pulmonary trunk). Furthermore, in some cases an abnormal truncal valve (with four leaflets) is very suggestive of the diagnosis; this is seen with short-axis imaging. If the pulmonary artery origins are not stenotic and if pulmonary vascular resistance remains relatively low, the clinical condition resembles a left-to-right shunt, except for right-to-left mixing of blood across the VSD.

This defect should also be distinguished from double outlet right ventricle wherein two parallel great vessels exit the right ventricle. There is usually a VSD communicating the left ventricle to the aorta or pulmonary artery, as these great vessels can also be malposed with the aorta cranial and ventral (anterior) to the origin of the pulmonary artery. These can be complicated defects to classify in terms of degree of override needed for the diagnosis of DORV and the requirements for fibrous continuity between AV valves and great vessels.

Valvular Malformations

These are uncommon to rare with the most frequent valvular malformations seemingly affecting the right side of the heart.

Tricuspid Atresia – An important diagnostic consideration for cyanosis in a foal without signs of pneumonia is tricuspid atresia, a malformation that dictates right-to-left shunting of systemic venous blood at the atrial level. The atrial shunt might be across a true ASD or a patent foramen ovale. Because all venous return must mix in the left atrium, this malformation generally causes marked hypoxemia with cyanosis. Affected foals rarely survive to weaning age unless there is a concurrent VSD with a functional right ventricular outflow tract. Otherwise, pulmonary flow must come from a ductus arteriosus or systemic

collaterals (e.g., bronchial arteries). Accordingly, most foals are stunted, nurse poorly, and exhibit severe exercise intolerance and cyanosis at rest. Arterial oxygen tension can be very low (40 to 60 mmHg).

2D echocardiography reveals a severely dilated right atrium and coronary sinus, atretic tricuspid valve with hyperechoic tissue near the A-V connection, and a rudimentary right ventricle (it will be larger if there is a functional left to right shunting VSD). Color Doppler or contrast studies can be used to identify pathways of blood flow. Atrial shunting that allows systemic venous return to empty into the left atrium must be observed; a VSD might be present. Abnormal flow patterns can be verified by contrast or color Doppler echocardiography. Prognosis is grave.

Tricuspid Valve Dysplasia – Severe tricuspid stenosis presents with similar Echo findings to tricuspid atresia, although the right ventricle and pulmonary trunk are likely to be better developed. Tricuspid valve dysplasia can involve the leaflets, insertion points, or support apparatus and often demonstrates abnormal valvular motion. Failure to delaminate the septal leaflet will result in an apically displaced coaptation point, similar to the canine disorder. Doppler studies show valvular regurgitation; less often valvular stenosis with prolonged pressure half-time and prominent A-wave on spectral Doppler recordings. Severe tricuspid regurgitation leads to secondary volume overload of the right atrium and right ventricle.

Pulmonary Stenosis – Congenital narrowing of the pulmonic valve can be diagnosed using 2D imaging and Doppler studies. The severity of obstruction is manifested in the degree of valve fusion, limited opening of the leaflets (doming), and secondary changes that include right ventricular hypertrophy (concentric), right atrial dilation, and pulmonary artery dilation. Bicuspid pulmonary valve might be evident in short axis imaging. Color Doppler shows turbulent flow across the RV outflow tract, starting just proximal to the stenosis. Spectral Doppler shows high velocity systolic flow that can be further assessed by the simplified Bernoulli equation (pressure drop = $4V^2$). The right atrial A-wave is likely to be prominent and a relaxation abnormality (Grade I diastolic dysfunction) is typical in the right ventricle. This is one lesion that is potentially palliated using balloon valvuloplasty and transthoracic echo guidance is likely to be successful for this procedure. As this is a rare defect in horses, this procedure is not refined in this species (and a balloon catheter with a length of 110 cm would likely be needed in a weanling or young adult horse).

Aortic and Mitral Malformations – In terms of left sided malformations, there are sparse reports of aortic or subaortic malformations. Routine imaging of the LV outflow tract and aortic valve should be diagnostic. Severity should be reflected in the degree of secondary left ventricular enlargement. Unless the jet is eccentric, it is difficult to align well to the equine LV outflow tract, therefore, Doppler interrogation of flow velocity and pressure gradient is likely to be underestimated.

Mitral valve malformations are probably under-diagnosed. A significant number of equine cases of mitral regurgitation involve horses <6 years of age, and it is likely some of these represent abnormal leaflet, chordal, or papillary development. Vascular hamartoma has been observed in at least one case of severe mitral regurgitation.

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Surgical Repair of Tricuspid Valve Dysplasia

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Tricuspid valve dysplasia is an uncommon congenital malformation in dogs, accounting for approximately 3% of congenital cardiac malformations (Oliveira et al. 2011). A spectrum of valvular lesions can occur with thickened, immobile leaflets tethered to the ventricular septum amongst the most common (Liu & Tilley 1976). Historically, treatment options for canine tricuspid valve dysplasia have been confined to medical management of right sided congestive heart failure which develops in a proportion of cases (Adin 2008).

Treatment of this disease in human medicine depends on its severity, with surgical management playing a large part. Surgical options consist of both tricuspid valve replacement and repair, with the severity and exact malformations present, as well as patient factors playing a role in deciding upon treatment.

In veterinary medicine to date, surgical treatment has been reported as tricuspid valve replacement. The first published results of this was by Arai et al. in 2011. This report described the results for 12 dogs, of which 10 survived the surgery. In the medium to longer term, two were euthanised at 10 and 13 months post-operatively due to inflammatory pannus formation and consequent failure of the bioprosthesis. Formation of pannus affecting the function of the bioprosthesis was reported in a further three dogs from another centre undergoing TV replacement, as well as the result of failure of mitral valve replacement in six of seven dogs (Bristow et al 2017, Orton et al 2005).

Due to these suboptimal results and the progression of our mitral valve repair programme, it was decided to pursue tricuspid valve repair at the Royal Veterinary College. Between October 2017 and March 2019 six dogs who were diagnosed with tricuspid valve dysplasia underwent tricuspid valve repair under cardiopulmonary bypass. Four of the six dogs had experienced at least one episode of congestive heart failure pre-operatively and were receiving diuretic medications. Breeds consisted of three Labradors, two boxers and one Spanish Waterdog. Two dogs were in atrial fibrillation pre-operatively. Two dogs required additional malformations treating at the time of surgery; one dog had a cortriatriatum dexter lesion resected and another dog had closure of a common atrium with a GoreTex patch as well as ligation of a persistent left cranial vena cava.

The tricuspid valve was accessed via a transverse right atriotomy. The abnormalities of the tricuspid valve were very similar between dogs with direct insertion of the papillary muscles onto the mural valve leaflet. In 4/6 cases these papillary muscles were very thickened and short, creating substantial leaflet tethering. In these cases, the papillary muscle attachments were cut and the mural leaflet resuspended using GoreTex sutures. All dogs had numerous

short primary and secondary chordae tendinae to the septal valve leaflet, causing severe restriction of leaflet movement. These attachments were cut and the leaflet resuspended with GoreTex sutures in all cases. A partial annuloplasty was then performed in all cases using a strip of GoreTex and simple interrupted TiCron sutures.

All dogs survived surgery and survived to discharge from the hospital. Two dogs developed AV block intra-operatively. In the first case temporary pacing leads were placed but replaced with an epicardial pacemaker the following day when these became dislodged. In the second case an epicardial pacemaker was placed under the same anaesthetic as the tricuspid valve repair.

The first dog undergoing this surgery developed a soft cough followed by haematoptysis post-operatively. After a series of investigations and treatments, he ultimately ruptured an artificially placed chordae tendinae at 4.5 months post-operatively with recurrence of congestive heart failure. He was euthanised on veterinary recommendation at 5 months post-operatively due to a poor quality of life and grave prognosis.

One dog developed atrial fibrillation approximately 11 months post-operatively and despite appropriate management, ventricular arrhythmias were documented 5 months later. Eighteen months post-operatively this dog underwent sudden death whilst on a walk, thought to be due to a fatal ventricular arrhythmia. The remaining four dogs are still alive at the time of writing (range 8-15 months post-operatively), with a good owner assessed quality of life.

Tricuspid regurgitation was categorised as torrential pre-operatively in all dogs and although reduced post-operatively, reduction was to a moderate to severe level in all dogs. Right sided cardiac dimensions reduced but remained above reference range in all cases. This concept is an expected outcome following a proportion of cases of tricuspid valve repair in human medicine, unlike that seen with mitral valve repair in both humans and dogs. The exact reasons as to why tricuspid regurgitation does not reduce further in more cases is largely unknown. It is possible that this reduction is enough to ensure a sustained and long term improvement in clinical signs and quality of life in the majority of dogs, however these cases will need to be followed closely over a longer time frame in order to ascertain this. Further work is also required to explore why reduction in tricuspid regurgitation is not more substantial in dogs, and ultimately, to establish if refinement of the technique can result in a more marked reduction in tricuspid regurgitation.

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Mitral valve dysplasia in English springer spaniels

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Introduction: To describe the signalment, physical examination and echocardiographic findings of a series of English Springer Spaniels (ESS) diagnosed with congenital mitral valve dysplasia (MD).

Animals: Fourteen client-owned ESS with congenital MD referred for murmur investigation and/or suspected congestive heart failure (CHF).

Methods: Retrospective case series. Medical records and echocardiograms were reviewed to collect relevant data.

Results: All fourteen dogs showed the typical echocardiographic lesions associated with MD: tenting of the valve apparatus (12/14 dogs), thickened valve leaflet tips (9/14), 'hockey stick' appearance to the one or both leaflets (8/14), elongated anterior leaflet (4/14). Tethering of one or both leaflets to the interventricular septum or papillary muscles was seen in 13/14 dogs. Three dogs had evidence of concurrent mitral valve stenosis. Twelve of the 14 dogs presented in CHF. Cardiac remodeling, i.e. left ventricular dilation (12/14), left atrial dilation (9/12), increased sphericity (10/14) was common. Four dogs had or went on to develop atrial fibrillation.

Conclusions: Congenital MD is seen in ESS and frequently results in marked cardiac remodeling and CHF. Affected patients may have concurrent mitral valve stenosis and may develop AF. It may be difficult to distinguish MD from myxomatous mitral valve disease and/or dilated cardiomyopathy. MD should be considered in ESS with a left-sided apical systolic murmur, particularly in young dogs.

General congenital morphology and echocardiography

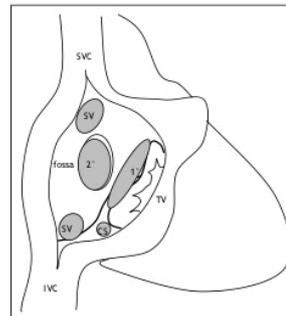
Dr. Navroz Masani, Consultant Cardiologist
 Cardiff and Vale University Health Board, Cardiff, U.K.

Vi-Hue Tran
 Institute of Cardiovascular Science, UCL, London, U.K

General congenital morphology in echocardiography

Nav Masani

ASD

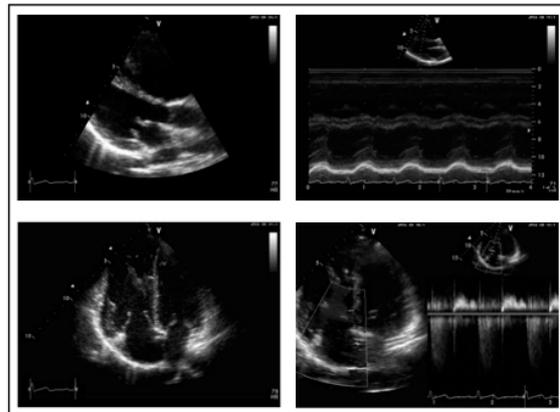
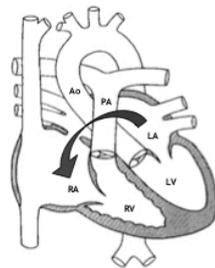


- Secundum
- Primum - AVSD
- Sinus venosus
- Unroofed coronary sinus

Secundum ASD

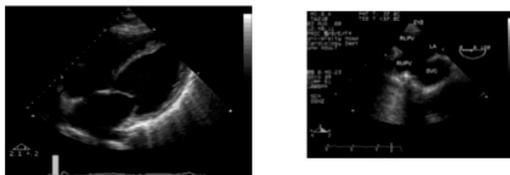
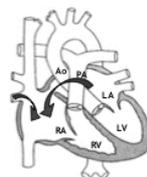
RH overload

- RV volume overload
- source of embolus
- associated lesions
- suitability for device

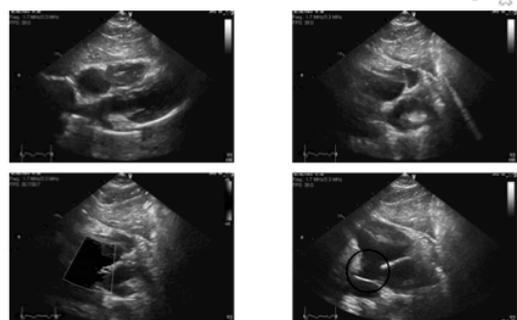


Sinus venosus ASD

- lx of RH overload
- difficult by TTE
 - close to SVC
 - close to IVC
 - close to coronary sinus
- associated with
 - partial anomalous pulmonary venous drainage



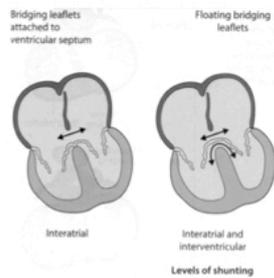
Sinus venosus ASD



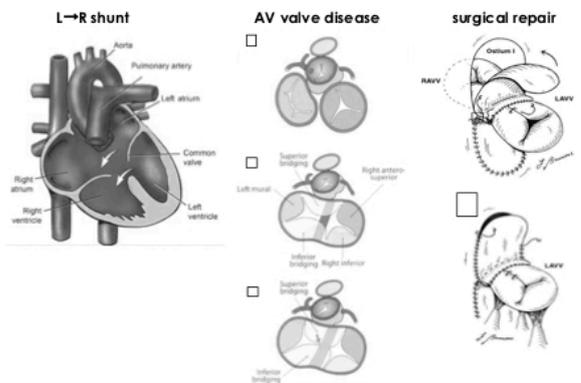
Atrioventricular Septal Defect

AV canal defect
Endocardial cushion defect

- ASD
- partial AVSD
- primum ASD
- inlet VSD
- intermediate AVSD
- complete AVSD
- common AV valve
- AVV regurgitation

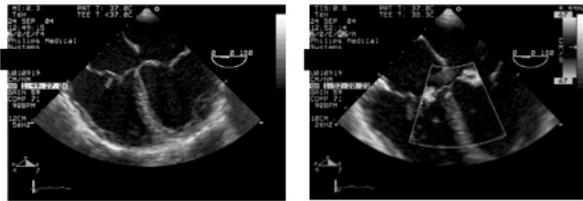


Consequences of AVSD (repair)

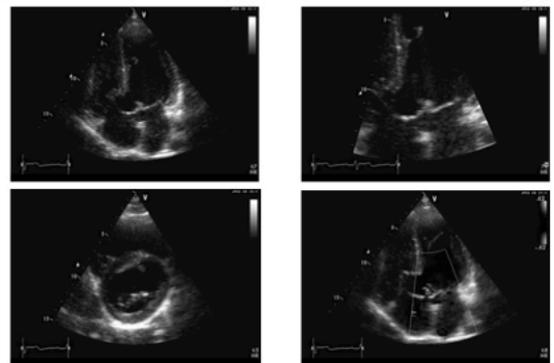


Partial AV Septal Defect

- Lack of A-V offsetting
- Primum ASD
- No VSD
- LAVV - "cleft MV"

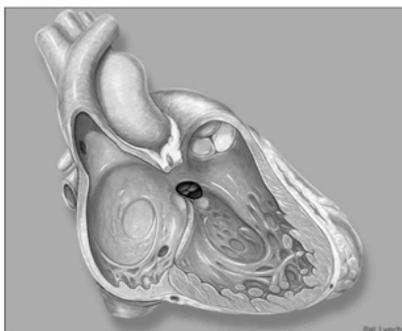


Complete AVSD

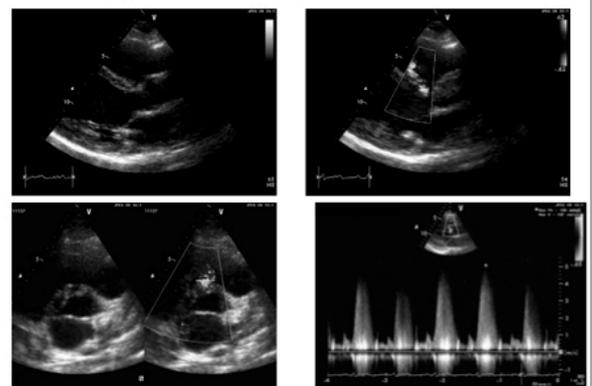


Perimembranous VSD

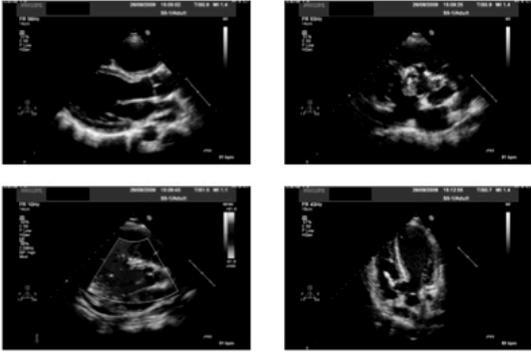
80%
TV plugging



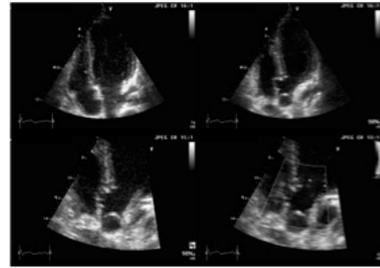
Perimembranous VSD



Perimembranous VSD



TV plugging



Sub-pulmonic VSD

Sub-arterial, Supra-cristal, Doubly committed
5%
AV prolapse



Sub-arterial VSD

TOE LAX

TOE SAX



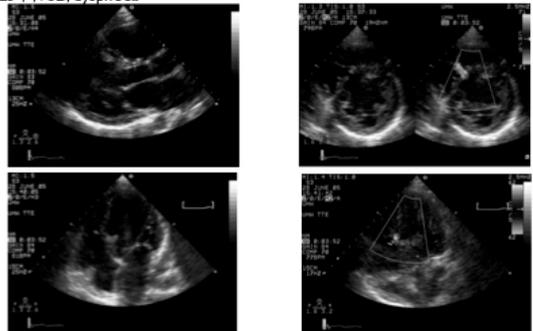
Muscular VSD

15%
Muscle overgrowth



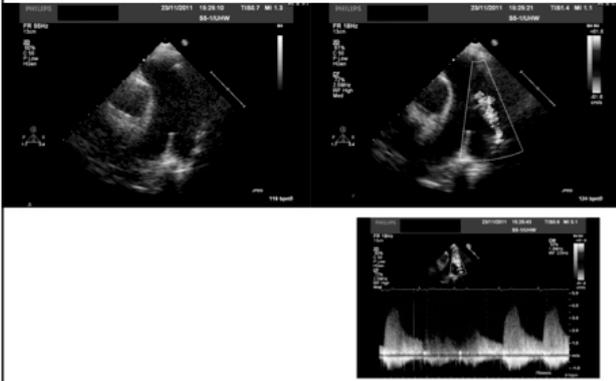
Muscular VSD

25 ♀, VSD, dyspnoea

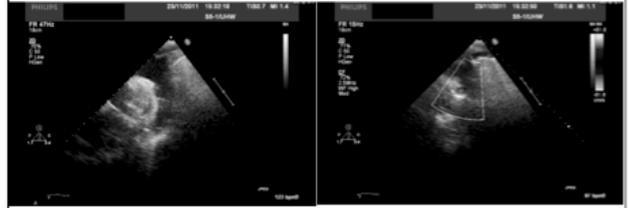


Suitable for device closure?

PDA

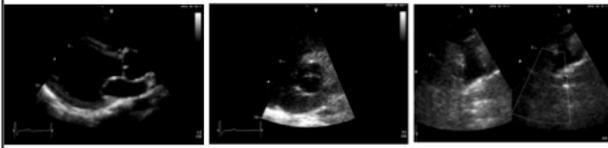


Patent Ductus Arteriosus



Coarctation of the aorta Operated or unoperated

- stenosis in juxtaductal region
- secondary aneurysmal dilatation
- 80% assoc'd with bicuspid AoV
- aortic root dilatation
- hypertension



Aortic coarctation

Modified Bernoulli equation

$$PG = 4(V_2^2 - V_1^2)$$

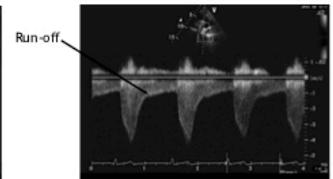
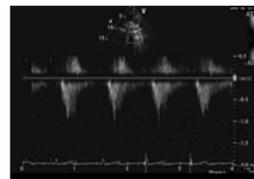
$$PG = 4(4.2^2 - 1.0^2)$$

$$PG = 4(17.6 - 1)$$

$$PG = 66\text{mmHg}$$

V_1 (PW)

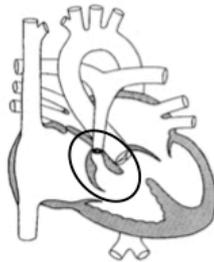
V_2 (CW)



Tetralogy of Fallot

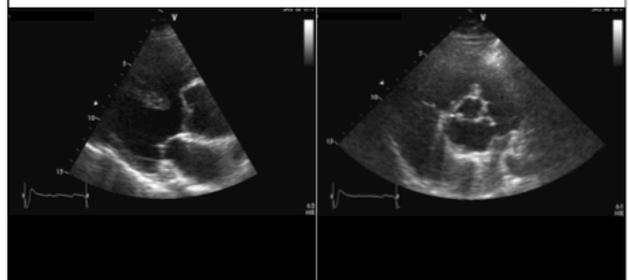
Failure of cono-truncal development

- RVOTO
- RVH
- VSD
- overriding aorta



Tetralogy of Fallot

VSD, overriding aorta, RVH



Morphology and Echocardiography of PDA and PS

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Echocardiography (**Echo**) is the most common method for confirming and evaluating suspected cases of patent ductus arteriosus (**PDA**) and pulmonic (pulmonary) stenosis (**PS**). These are two of the most common congenital heart defects identified by veterinary cardiologists. Additionally, most cases of PDA and PS can be definitively treated or palliated through an interventional catheterization technique or surgical procedure. Understanding the morphology of these diseases and related cardiac responses are critical to diagnosis, staging and effective treatment. This presentation will focus on Echo imaging in these two disorders. The morphology of PDA & PS will be described in this session by Vi-Hue Tran, MoP.

Ultrasound imaging, angiography, and examination of post-mortem specimens are integral to understanding PS and PDA in dogs and cats. Images from these sources demonstrate key features relevant to cardiologists, catheter interventionists, and surgeons. These notes are a supplement to the case studies presented during this Veterinary Cardiovascular Society scientific meeting. Most of the following comments pertain to dogs; however, some feline examples will be presented. (Abbreviations used in this notes set are initially **bolded**.)

The echocardiographic evaluation can include transthoracic (TTE) or transesophageal (TEE) imaging (which is invariably performed under general anesthesia in veterinary practice). Most cases are evaluated by TTE from right and left parasternal (intercostal) windows. Most veterinary cardiologists perform 2D grayscale imaging exclusively along with standard Doppler studies. A small number working in referral practices have employed 3D imaging (real time or full volume acquisitions with subsequent analysis). Currently, 3D transducers are relatively large for most of our PS and PDA patients and the incremental value provided requires better delineation. 3D imaging does offer useful information in some cases, particularly in larger dogs or those with poor transthoracic windows. Hemodynamics are further explored by Doppler Echo. The author obtains initial Doppler images of blood flow across the stenosis while the patient is fully conscious, as sedatives can alter heart function, hemodynamics, and blood pressure. However, TTE image quality is usually enhanced by conscious sedation as the typical dog and cat hold little tolerance for protracted imaging. Accepting that withdrawal of sympathetic tone will likely reduce systolic ventricular function, RVOT velocities, and systemic blood pressure, the overall study quality and stress on everyone is reduced following sedation/tranquilization. One might argue, especially for PS, that a baseline study under sedation creates a better comparison for follow up examinations

by mitigating some of the adrenergic effects on gradients. Although clinician preferences vary, the author uses combinations of butorphanol (0.2 to 0.25 mg/kg, IV or IM for dogs and for cats) or buprenorphine (0.005 to 0.0075 mg/kg, IV, IM, SQ for dogs) and usually mixes the opioid with acepromazine (0.025-0.03 mg/kg for dogs; 0.05 to 0.1 mg/kg for cats depending on IV or IM route of administration). Alfaxalone (1-2 mg/kg IM or IV) following butorphanol is another consideration for intractable feline patients.

A TEE examination is typically conducted during an interventional catheterization procedure, and in some cases, offers additional information regarding the morphology of the lesion. Compared to the TTE study, the anatomic delineation for guiding an effective and safe pulmonary balloon valvuloplasty is sometimes better with TEE. There is little doubt that ductal anatomy is clearer with TEE; it can be with TTE studies. However, evaluation of the pulmonary valve by TEE can be challenging, especially in smaller dogs. Furthermore, the availability of specialized endoscopes (human adult, pediatric; with or without 3D capabilities) is pertinent. Even in well-equipped practices, patient size and cost limit the use of TEE, especially for dogs <3 to 3.5 kg.

PULMONIC STENOSIS

Canine PS is a genetically predisposed congenital heart disease, affecting many breeds. Congenital PS is less common in cats, but is observed sporadically. Clinically significant PS is recognized by identification of a systolic ejection murmur. This is generally followed by an echocardiogram (**Echo**) study with complementary Doppler imaging. Definitive diagnosis can be achieved in nearly every case by Echo, and the severity of the condition assessed by evaluating the valve morphology and motion, quantifying hypertrophy of the right ventricle (**RV**) and recording suitable Doppler images. Additional comorbidities such as a patent foramen ovale (**PFO**), ventricular septal defect (**VSD**), PDA tricuspid valve incompetency, tetralogy of Fallot or cor triatriatum dexter might be identified. Tetralogy and CTD are not addressed in this presentation.

Table 1 – Echocardiographic Evaluation of Pulmonic Stenosis

- 2D Evaluation of the pulmonary trunk & branch arteries (anatomy, size, length & diameters)
 - Rule out concurrent branch stenosis, patent ductus arteriosus, or systemic to pulmonary shunts
- 2D (\pm 3D) Evaluation of pulmonary valve morphology, thickness, attachments, and mobility
- Measurements of the minimal pulmonary valve and annular plane diameters
- 2D (\pm 3D) assessment of the right ventricular outflow tract (RVOT) for:
 - Supravalvular obstruction or leaflet tethering
 - Discrete or tunnel-like fixed subvalvular obstructions
 - Dynamic subvalvular obstruction
 - Prominent (single origin, right or left) coronary ostium (rule out prepulmonic CA)
 - Subarterial or muscular ventricular septal defect
- Color & Spectral Doppler echocardiographic assessment of systolic & diastolic flow | velocities
 - Color Doppler assessment of flow in RVOT (aliasing, vena contracta, turbulence)

- CW Doppler recording of maximal and mean velocities across the RVOT
 - Calculate maximal instantaneous and mean RVOT gradients (Bernoulli)
 - Estimate influence of stroke volume on PGs by calculating ratio of PV/AV VTIs, or estimating LV stroke volume using PW Doppler and 2D imaging
 - Identify levels of fixed or dynamic obstruction (V_1 , V_2 velocities & timing)
 - Estimate of severity of pulmonary regurgitation
 - 2D & M-mode evaluations of the right ventricular cavity and walls
 - Hypertrophy of walls and papillary muscles
 - Ventricular radius of septal curvature (diastolic and/or systolic septal flattening)
 - Estimation of right ventricular systolic function, diastolic function, and ventricular filling pressures using multiple imaging modalities
 - 2D & M-mode methods (e.g. Fractional area change; TAPSE)
 - Doppler methods (e.g., transtricuspid filling; tissue Doppler of the RV wall)
 - Potentially speckle tracking or 3D off-line analysis
 - 2D & Doppler evaluation of the right ventricular inlet to identify perimembranous ventricular septal defect or a double-chambered right ventricle
 - 2D and Doppler Evaluation of the right atrium
 - Evaluation of the atrial septum for a patent foramen ovale
 - Evaluation of the caudal right atrium for obstructive membranes (cor triatriatum dexter) or abnormal pulmonary or systemic venous drainage
 - 2D and Doppler assessment of the left ventricular outflow tract for systolic anterior motion of the mitral valve and dynamic obstruction due to altered ventricular geometry
 - Identification of the putative right and left coronary artery ostia and branches (when feasible)
 - Determination of aortic root size as a comparator the annular plane of the pulmonary valve
 - Inspection of the Echo ECG for rhythm abnormalities and the QRS complexes for hypertrophy patterns or preexistent bundle branch block (if uncertain, obtain a diagnostic ECG).
 - Balloon valvuloplasty: Additional TTE or TEE guidance regarding valve morphology, dimensions, coronary anatomy, & potentially TEE monitoring of balloon positioning & inflation. Imaging of immediate complications: cardiac perforation, pulmonary dissection, or intra-pericardial bleeding.
 - Post-procedure: focus on valve mobility & swelling; fixed/dynamic outflow tract gradients; changes in pulmonary regurgitant jets; & procedural complications such as pericardial effusion, pulmonary dissection, acquired aortopulmonary communication, and pulmonary & tricuspid regurgitation.
-

General Points Echo imaging of congenital PS is usually centered on the valve proper and includes 2D (\pm 3D) imaging, color Doppler imaging (**CDI**), pulsed-wave Doppler (**PWD**) and continuous wave Doppler (**CWD**) imaging. The segments adjacent to the right ventricular outflow tract (**RVOT**) can also be involved in the lesion, producing subvalvular or supra-valvular stenoses. Additionally, hypertrophy of the RV is common and often leads to a mid-to-late systolic, dynamic obstruction readily seen by CWD imaging.

Although Echo is the noninvasive diagnostic test of choice, a thorough medical history of clinical signs should be obtained prior to imaging (accepting most clients consider their dogs and cats “normal”). Patients should undergo a complete physical examination. Noninvasive blood pressure should be recorded just prior to the Echo, and an ECG tracked throughout the study. A systematic approach to the assessment of PS is recommended to avoid missing an important lesion (see **Table 1** for an example).

In many veterinary patients, a complete Echo assessment is facilitated by including a sedated examination. As noted previously, TEE can provide some additional morphologic detail, but the vast majority of PS cases are not assessed this way in veterinary practice. When TEE is not possible, and initial images are suboptimal, the author has found that performing TTE immediately prior to balloon valvuloplasty while the patient is under anesthesia can be instructive. The slower heart rate and immobility provided by anesthesia often improves the delineation of valve anatomy while offering another chance for measurements and completing a saline contrast study. Of course, outflow gradients are markedly reduced under general anesthesia and therefore unreliable. This procedure should be brief and focused on pulmonary valve and coronary anatomy as the longer these patients are anesthetized, the greater the risk of anesthetic complications later in the operation.

2D (3D) Imaging of PS The diagnosis of PS is usually straightforward with 2D imaging. In addition to the right-sided images with the aorta visualized in short axis, the left cranial position and less conventional pulmonary valve-focused image planes should be obtained. As originally described by Patterson and colleagues in Beagle dogs, key aspects of PS include valvular thickening, commissural fusion, and varying degrees of annular plane and leaflet hypoplasia. These are not the only morphologic features of canine PS, but are a good starting point when evaluating the RVOT by 2D (or 3D) Echo. It is noteworthy that despite a wide veterinary experience with Echo, there are relatively few *correlative* studies between morphology at autopsy and Echo findings. Real-time Echo studies capture varying degrees of leaflet mobility, with doming and supravulvular tethering common findings. Combinations of obstructive lesions are relatively common in canine cases. For example, in French bulldogs, complex obstructions at multiple levels have been observed with some so severe as to challenge the value of any catheter-based procedure for palliation. Cats with PS can have valvular or subvalvular PS. The distinction between double outlet right ventricle and infundibular PS in cats is unresolved and the location of the obstruction relative to internal structures should be considered. Overall, the canine disease seems more similar to atypical PS in children, or valvular dysplasia. However, some dogs have relatively thin, well developed, mobile, but fused leaflets of “typical” PS. Canine valvular PS has also been classified as Type A (fused leaflets with mobile valve and doming) and Type B (thickened leaflets, hypoplastic leaflets, and reduced mobility). There are likely breed and geographic differences in the prevalence of these various lesions and there is no consensus statement of classification of PS in dogs or cats.

Coronary Anatomy in PS Emphasis is placed on the coronary circulation in PS, especially over of concerns of a single origin of the two coronary arteries. The major issue is a single origin trunk stemming from the right sinus of Valsalva and distributing both the right and left

coronary artery (CA) but with a pre-pulmonic left CA coursing over the subpulmonary infundibulum before dividing into the paraconal (cranial interventricular descending) and circumflex branches. This has been observed mainly in English bulldogs with a concurrent subvalvular component of PS; however, other brachycephalic breeds can be affected, as well as dogs of unrelated somatotype. It has been suggested that the "RIIA" coronary anomaly is less related to in-utero development of subvalvular PS, and more of a genetic liability in English bulldogs. Regardless, its presence affects the approach and aggressiveness of pulmonary balloon valvuloplasty as most cases have a distinct subvalvular obstruction with the CA nestled in that space. Less common is the single ostium coronary artery from the left sinus of Valsalva with a prepulmonic course of the right CA. Even rarer is a CA with an interarterial path. Generally, the left CA is relatively easy to visualize by TTE; however, the right CA is more challenging and discrepancies have been noted between Echo and angiographic assessments (which the author still performs prior to balloon valvuloplasty). In breeds at high risk, a CT angiogram might circumvent further anesthesia if the owner is unwilling to proceed with a "gentle" balloon procedure should a pre-pulmonic CA be identified. Identification of coronary anatomy has been reported to improve with TEE. The coronary arteries are identified initially by viewing the aorta in short axis. While obtaining this image, also remember to use the aortic diameter as a general "ruler" to decide if the pulmonary annulus is hypoplastic (the normal ratio is about 1:1 measuring the aorta in short axis and the adjacent pulmonary valve annulus in long axis). Using fine plane angulation from the short axis plane, the noncoronary cusp of the aortic valve can be seen straddling the atrial septum, the origin of the normal left CA is closest to the left atrial appendage, and the right coronary artery originates closer to the right side of the thorax. Both left and right ostia normally "face" the PA. The left CA is also potentially visible from parasternal long axis images. In true short lines of CW velocity spectra should be optimized to identify a clear modal velocity and in many cases, a fainter velocity spectrum emphasizing grayscale is preferable to a "bright" filled-in spectrum. The examiner should aim for clear envelope and peak velocity profile. Commonly the assessment is confounded by measuring the "whiskers" instead of the "chin" of the spectral velocity profile. These errors occur at the highest velocities and therefore have clinical impact. Multiple spectral signals should be measured and averaged, with attention to the physiological state and heart rate (prior cycle lengths). Both maximal instantaneous and the mean pressure gradient (PG) should be determined. The usual relationship is about 0.55 to 0.60 mean PG to maximal PG.

The flow pattern across a stenosis is characterized by conversion of potential (pressure energy) to kinetic energy. Proximal to the obstruction there is a rapid acceleration of flow within the zone of proximal convergence. Occasionally a proximal isovelocity area is evident on color Doppler imaging (CDI) and it can be "tracked" by discretely moving a small PWD sample volume toward the valve. However, more often, there is a rapid acceleration, a number of aliasing wraps (depending on the Nyquist limit settings) and activation of the turbulence algorithm if "on". Properly gained CDI images often show an abrupt narrowing at the vena contracta but over-gained studies simply show turbulence everywhere along with color bleeding into adjacent pixels. Filling of the sinuses behind the valve leaflets can be confusing, as the valve can appear angled with leaflet anchor points at different levels relative

to the long axis of the pulmonary trunk. Additionally one leaflet often appears more mobile than the other two. Distal to the obstruction marked turbulence is evident by CDI or pulsed-wave Doppler (PWD). Some degree of pressure recovery might occur in the pulmonary trunk in PS although this is better studied in aortic stenosis. Mild cases of PS must be distinguished from the normal impulse gradients exaggerated by sympathetic tone, but clinically significant PS causes high velocity flow (and Doppler Echo was never designed to screen dogs for breeding :). The severity of PS is usually estimated by combining evidence of valve morphology and ventricular remodeling with Doppler estimated PG. This is discussed more below.

Frequently CWD imaging uncovers a velocity profile proximal to the valvular stenosis that is substantially augmented from dynamic RVOT obstruction. This situation creates a subvalvular velocity profile (V1) superimposed on the peak velocity envelope (V2). If the second peak is late in systole it should have little effect on the maximal PG estimate based on squaring the peak velocity (V2); however, if the velocity is >2 m/s in mid-systole, the simplified Bernoulli equation should not be used as it will overestimate the valvular obstruction. The mean pressure difference across the stenotic valve can be estimated by tracing each profile and subtracting the mean gradient of V1 from the mean gradient of the peak envelope. Additionally, the longer form of the Bernoulli equation can be used that subtracts from the peak velocity (V2) the instantaneous V1, before squaring. It is re-emphasized that Doppler PGs recorded from unsedated dogs are substantially higher (often twice) compared to peak-to-peak pressures recorded by catheter during general anesthesia. The two cannot be meaningfully compared unless recorded simultaneously.

Thus, identifying and quantifying isolated, moderate to severe PS by Doppler Echo is relatively straightforward so long as the examiner obtains good alignment to the jet and optimizes the velocity spectrum. Additionally, serious obstructive lesions are accompanied by abnormalities of 2D/3D imaging along with cardiac remodeling. Conversely, identification of trivial to mild outflow tract stenosis is more problematic. Normal transvalvular flows frequently alias and activate turbulence mapping in CDI; thus, 2D imaging and spectral Doppler studies must be used to confirm any suspected obstruction.

Ventricles The response of the RV to outflow obstruction is concentric hypertrophy, readily seen by 2D and M-mode imaging. Similar to severe subaortic stenosis, hyperechoic segments are sometimes observed with severe RV hypertrophy, suggesting prior subendocardial ischemia and replacement fibrosis. The hypertrophy usually includes the right side of the ventricular septum and papillary muscles. The increased RV pressure flattens the septum and reduces the LV size; when systemic pressures are present, the septum remains flat throughout the cardiac cycle (easily seen with M-mode studies). Severe RV hypertrophy often leads to a second level of functional, dynamic obstruction in the infundibulum. This obstruction is often relieved by beta-blockade, although the value of that treatment is unproven.

Diastolic dysfunction of the RV is common and heralded by RA dilation and E/A reversal with prolonged isovolumetric relaxation times in tricuspid inflow and tissue Doppler imaging

(TDI). Systolic function of the RV is generally preserved based on tricuspid plane annular systolic excursion (TAPSE), RV fractional area change, and the s' velocity in TDI. Subtle reductions in RV systolic function have not been sufficiently addressed with invasive gold standards, but it is likely that TDI and myocardial strain imaging can identify contractile dysfunction if serial examinations are performed. With long-standing, severe PS, overt dilation develops in the right ventricle and right atrium (RA) usually accompanied by tricuspid regurgitation (TR). This can be complicated by atrial flutter or fibrillation, which are poorly tolerated in severe PS.

The left ventricle (LV) shows a reduced cavity size in severe PS along with the aforementioned changes in septal motion. A prominent rightward systolic movement of the ventricular septum suggests that RV systolic pressure are less than systemic. Changes in geometry predispose to systolic anterior motion of the mitral valve with dynamic LV outflow tract obstruction and mitral regurgitation. This might improve with beta-blockade or following successful balloon valvuloplasty.

Comorbidities within the RV can include subarterial or perimembranous VSD. In cases of tetralogy of Fallot or a large muscular VSD, the defect will be obvious by 2D imaging. If systolic pressures equilibrate across the ventricles, the velocity of shunt flow is low and a smaller perimembranous or muscular VSD can be overlooked. Another potential finding with PS is the presence of fibrous muscle or fibromuscular bands. The distinction between infundibular PS and double chambered RV is not clear-cut, especially in cats where one cardiologist's DCRV as another's subvalvular PS. VSD is sometimes associated with DCRV in dogs and cats; however, it is unknown if an isolated VSD is likely to cause progressive subvalvular PS.

Tricuspid Valve The normal canine and feline tricuspid valve includes a short septal leaflet, inserting just apical to the anterior (cranial) mitral leaflet on the atrioventricular septum and a more expansive parietal or lateral leaflet that in humans is subdivided into anterior and posterior cusps. The apical papillary muscles can be multi-headed and this becomes more obvious when there is RV enlargement. Tricuspid regurgitation is a common finding in PS and offers another chance to quantify the RV systolic pressure using CWD. With severe RV hypertrophy, the papillary muscles and even the chords appear to thicken so it is possible that any TR is just "functional" and related to the geometric changes within the RV and high systolic pressure found in that chamber. However, the potential for concurrent tricuspid malformation should be considered and the valve apparatus inspected from multiple planes. Sometimes the distinction between primary and secondary TR due to valve dysplasia is not clear-cut.

Right Atrium Most cases of moderate to severe PS are accompanied by some degree of right atrial dilation with a prominent atrial kick evident in the PW Doppler and CW Doppler recordings. This late diastolic outflow velocity is often captured by the nearfield segment of the CW cursor and can be quite prominent. The atrial septum should be carefully screened for a patent foramen ovale. 2D imaging might show a mobile valve of the foramen ovale in the left atrium, CDI (with low velocity filters and velocity scales turned "down") might identify right to left shunting across the foramen ovale. The color appearance of shunting is typically

one of a low velocity (dark blue), curved flow moving from atrial septum into the dorsal left atrium. This must be distinguished from pulmonary venous return, which enters immediately dorsally (to the “right” of a PFO) in the parasternal long axis plane. Short axis images can be confusing as the atrial septum is often foreshortened and a right to left shunt might appear to be located in the primum septum as opposed to the oval fossa. The finding of right-to-left shunting PFO is common and found in 39% of 31 cases in one canine study. This comorbidity can influence the long-term clinical features of the disease in terms of risk for CHF or cyanotic heart disease. Hematocrit should be followed in patients with a functional PFO and access to water insured to prevent dehydration and additional hyperviscosity. (The author has clients add salt or bouillon to the food to encourage drinking.)

Contrast Echocardiogram Some cardiologists routinely perform a saline contrast study in cases of PS because CDI can be ambiguous for the presence of a PFO. This procedure can be performed at the time of initial diagnosis (facilitated by sedation) or prior to cardiac catheterization if TTE or TEE will be done. Infrequently multiple levels of right-heart obstruction are identified in a young animal that includes cor triatriatum dexter, tricuspid valve malformation with TV stenosis, hypoplastic RV, and under-developed RVOT with PS. There is usually a PFO in these cases. High pressure in the caudal right atrial chamber can prevent saline contrast from entering the caudal RA chamber but contrast might be diverted into the left atrium through a PFO or enter the cranial RA chamber via an azygous vein (as pressures cranially are lower).

Assessing Severity of PS The term critical stenosis is probably not too relevant to veterinary patients that are unlikely to see a veterinarian prior to 6 or 7 weeks of age. It is likely that dogs and cats with truly critical PS succumb before that first veterinary exam. However, the severity of PS ranges widely from trivial gradients to peak PG values over 240 mm Hg. Although PG relates to severity, clinical signs including impaired exercise capacity, tiring, and development of right-sided CHF may not be clearly linked to the Doppler gradient. The author has observed more than a handful of small breed dogs with severe PS (PGs >100 mm Hg) survive to 8 years of age. Other factors might influence the natural history including the size of the patient, presence of a PFO or VSD, concurrent tricuspid valve dysplasia, coronary anatomy, reflex responses and cardiac rhythm. Regardless, the logical tendency is to observe and follow mild to moderate asymptomatic patients, and to intervene when the PS is moderately severe or worse.

A comprehensive approach to evaluating lesion severity should be undertaken, using findings from all Echo imaging modalities. Estimating valve cross sectional area by continuity equation or 3D Echo is rarely done in dogs or cats with PS. This might in part stem from the challenge of indexing the CSA to body size or somatotype. PGs only tell part of the story as flow across the valve exponentially affects the PG. As noted above, some estimate of stroke volume should be made, be it VTI ratios (PV/AV) or a volumetric estimate of averaged LV stroke volume with attention to prior cycle lengths. When there is multi-level RVOT stenosis, the peak PG can overestimate the severity of stenosis. If there is a clear V1 and V2 within the velocity spectra, the difference in PGs should be calculated to assess the valvular (balloon-target) component. Additionally, the RV systolic pressure should also be assessed from any

TR jet. If the patient is not in CHF, the pressure drop measured by TR (RV systolic pressure minus zero) should equal the sum of the CW gradient across the RVOT + the estimated PA systolic pressure of 22 mm Hg (PA pressure from personal observations from catheterizations). There should be commensurate levels of RV hypertrophy in most cases, although the degree of PR or TR and chamber volume can also affect apparent RV wall thickness.

Traditionally maximal (“peak”) RVOT gradients <50 mm Hg PG were considered mild; 50 to 80 mm Hg “moderate”, and maximal PGs >80 mm Hg classified as “severe”. However, in one study of 55 untreated dogs, a peak PG >60 mm Hg was the best cut-off for predicting death (AUC 84.1%). This study was retrospective and relatively small, but it might be noted that in children mild/moderate/severe Doppler PGs “cutoffs” are lower than for dogs, with a moderate severity PS at a peak PG of > 35 mm Hg and PGs >64 mm Hg (mean >40 mm Hg) considered “severe”. Currently there is no consensus regarding this in veterinary medicine, and more outcome data with untreated controls and standardized methods of measurement are needed.

Other factors are relevant. Clinical signs should also be considered in the assessment, as well as comorbidities such as tricuspid valve dysplasia or PFO. Some larger breed dogs seem experience more signs once PGs exceed 60 mm Hg and these patients might benefit from balloon valvuloplasty if experiencing exertional issues or syncope. Age and breed are additional factors, as a moderate PS gradient in a large breed puppy is likely to progress to more severe PS at maturity.

Procedural Guidance The value of TEE for guiding balloon valvuloplasty of PS is unclear based on personal conversations with other cardiologists. Some operators consider TEE useful and others find it a study that adds little to decision making (and creates a distraction that delays the procedure, prolongs anesthesia, and interposes another structure onto the vascular entry and fluoroscopic fields). In the author’s hospital, TEE is most often done as the anesthetized patient is prepped and anesthetic monitors are attached in the cath lab (with a 10 to 12 minute timer imposed by the technicians[⊗]). Digital images are captured for subsequent review by the operators prior to balloon selection, and the scope is then withdrawn and replaced by a marker catheter. As noted previously, TTE also can be performed immediately after induction of anesthesia further to evaluate the stenotic area, coronary ostia, and shunting lesions. The author does not find balloon positioning particularly difficult using fluoroscopy and the angiographic road map, but there might be times when TEE offers value-added, especially for operators trying to perform procedures without (or with minimal) fluoroscopy. In the rare case of guidewire or catheter perforation, having 2D imaging close by can assist with identification of intrapericardial hemorrhage, cardiac tamponade, volume depletion, or thrombus formation.

After an interventional procedure, the Echo should be focused on valvular mobility and swelling and the outflow tract gradient. This is typically measured the following day, often with a mild level of sedation in puppies and kittens. One should be mindful of fixed versus dynamic obstructions. Any changes in pulmonary regurgitant or tricuspid regurgitant jets should be noted. Uncommon procedural complications including flail tricuspid leaflet, pulmonary intimal dissection, and acquired aortopulmonary communication have been observed.

Follow up How to follow a case of untreated PS and to assess the “success” of a balloon valvuloplasty requires more longitudinal data. In a dog with overt CHF or repeated bouts of syncope, the success can be measured in part by the ability to reduce or end cardiac medications or exertional spells. However, in the largely asymptomatic patient most clinicians rely on the residual PG, which in isolation is probably insufficient as a benchmark. As noted previously, some estimate of stroke volume across the valve should be made, whether comparing pre- and post-procedural pulmonary to aortic VTI ratios or trying to calculate LV stroke volume from aortic VTI and cross sectional area. Attention to cardiac cycle length and to heart rate should be part of a meticulous assessment. Rarely is the PG eliminated in dogs with valve dysplasia, but right heart remodeling might improve and rarely revert to normal. Most clinicians accept that ~50% reduction in maximal PG is indicative of procedural benefit.

Often a “successful” technical procedure creates more pulmonary regurgitation. This is a concern in human patients for eventual RV failure, but whether it is a serious issue in dogs (that are smaller, quadrupeds, and have a somewhat more compressed RVOT) is uncertain. Increasing the degree of PR usually causes some degree of RV dilation, so wall thickness might decrease for this reason alone. Overall, PS balloon valvuloplasty in dogs (and likely in many cats) is palliative and retrospective studies have reported better outcomes than dogs not similarly treated. A large, prospective, multicenter trial or registry is needed. Restenosis is probably more common than we realize but challenging to assess without some estimate of transvalvular flow or cross-sectional area. Currently a veterinary definition of restenosis is not available, but in some dogs, the maximal PG approaches that of the original, pre-balloon valvuloplasty value.

Comprehensive assessments of the RV – chamber size, area changes, wall thicknesses, systolic function (TAPSE, tissue Doppler s’ fractional area change, possibly RV free-wall strain & strain rate), diastolic function, and flow and velocities across the tricuspid and pulmonary valves – should be undertaken during follow-up examinations. Measuring the pressure half-time of PR or the PR index might be instructive. Serial Echo exams combined with patient evaluation are likely to offer the best long-term assessments.

PATENT DUCTUS ARTERIOSUS

The patent arterial duct is one of the most common cardiac malformations in dogs and prevalence is underappreciated in cats. Diagnosis in the dog is suspected from auscultation of a continuous murmur over the PA and confirmed by echocardiography. In cats, the continuous murmur is less obvious and there is a greater tendency to develop progressive pulmonary vascular disease and pulmonary hypertension (PH). In typical left-to-right, shunting PDA, aortic pressures exceed PA pressures by ~5 times over the duration of the cardiac cycle. This PG drives the shunting that is readily detected by Doppler studies.

General Points Compared to PS, the echocardiographic assessment of PDA is somewhat less complicated. 2D Echo can demonstrate the ductal ampulla and secondary changes in the great vessels and cardiac chambers. Doppler studies can illuminate left-to-right (or right to left) shunting across the ductus, estimate arterial and intracardiac pressures, and assess heart size and ventricular function. The introduction of TEE to catheter-based occlusion procedures has been helpful and some centers rely extensively on this approach for procedural guidance, while other operators continue to use angiography/fluoroscopy exclusively. In the author's hospital, both imaging methods are used to guide transcatheter ductal occlusion procedures.

Definitive treatment of PDA is possible with surgery or a catheter-delivered occlusion device, exceeding >95% of cases in experienced centers, and the prognosis for a normal life is excellent. A small percent of dogs with PDA develop severe pulmonary hypertension (PH) from pulmonary vascular injury. In most of these cases, PH this occurs early in life (before 4 to 6 months of age) and it can be associated with necrotizing pulmonary arteritis. This results in nearly equal pressures across the great arteries and allows for bidirectional shunting, the so-called "reversed PDA." When it does occur, the shunt reversal occurs abruptly and early in life, although it might not be recognized until later. Gradual development of mild to moderate PH is sometimes observed in older dogs, and the influence of altitude certainty affects the likelihood of increased PA pressures. Progressive onset of moderate to severe PH is more common in cats with PDA, and some even develop signs of right-sided CHF. Concurrent cardiac malformations are also common in cats. **Table 2** lists some of the considerations involved with diagnosis, assessment, and intervention of PDA.

2D (3D) Imaging of Left-to-Right PDA Owing to the extracardiac location of the defect – between the descending aorta and the origin of the left PA – 2D imaging of ductal anatomy can be challenging. The PDA can be visualized from a right parasternal or a left cranial parasternal images optimized for the PA. Determining ductal morphology and size is important if a catheter intervention is planned and this is discussed further below.

As outlined in **Table 2**, 2D (3D) and M-mode imaging are used to quantify the secondary responses of left-to-right shunting. These include LA and LV dilation, increased LV sphericity, enlargement of the ascending aorta, and dilation of the main PA. Both LA and LV enlargement occur in direct relationship to the shunt size and pulmonary blood flow. The LA can be extremely enlarged when there is moderate to severe MR secondary to cardiac remodeling or from concurrent valvular malformation.

Systolic LV function is often in the low-normal range, especially if fractional shortening is

evaluated, but ejection fraction is more often normal if a 2D or 3D volumetric estimate is performed. It is worth remembering that volume is a function of the cube of the radius; therefore, in a dilated LV, less minor axis shortening is required to eject a large stroke volume. Additionally, fractional shortening likely overestimates LV volumes in cases of canine LV dilatation. Speckle tracking studies of dogs with PDA have shown that 24h post ACDO closure the minor axis shortening, radial strain, and circumferential strain are all reduced; whereas, normal longitudinal systolic function were evident. Some of these changes are likely due to reduced preload and increased afterload and the clinical importance of mild changes is debatable. However, in some dogs, LV function is clearly reduced and it persists after PDA closure. This is more common in larger dogs. Overall mildly depressed LV function does not appear to influence long-term survival unless complicated by myxomatous mitral valve disease or a dietary or genetic cardiomyopathy. (Consider: how many dogs with mildly depressed LV function have you seen after successful PDA closure return in congestive heart failure?). A DCM phenotype might be identified in some older and larger breed dogs and this is presumably the “cardiomyopathy of volume overload”. Advanced methods have been used to evaluate LV function in dogs with PDA. Whether these change outcomes or the way we manage patients with PDA is uncertain.

Otherwise, the RV is usually normal in PDA except in the situation of “reversed” PDA or a concurrent lesion such as an ASD, PS or tricuspid valve dysplasia.

Doppler Findings in Left-to-Right PDA The diagnosis of PDA is confirmed by examining the PA from cranial imaging positions and observing a continuous, high-velocity, turbulent flow pattern entering near the bifurcation of the PA and extending towards the pulmonary valve. Flow coming from the descending aorta but confined to the ductal ampulla is encoded in red by CDI, but this mapping abruptly changes to turbulence as it reaches the low-pressure side of the defect. The appearance of the shunt flow in the PA is variable: it might track eccentrically along the arterial wall; assume a serpentine path that leaves and reappears within the imaging plane; or completely fill the PA. The left cranial position is often superior for identifying the ductus. In this position, the left PA is positioned almost “on top” of the ductal ampulla and fine-plane imaging is needed to identify the minimal ductal diameter at the entry point into the left PA. Often the arterial duct can be visualized between the two branches, but rotation and tilt are needed to obtain this image.

When a PWD sample volume is advanced from the pulmonary valve toward the ductus the signal becomes turbulent, aliased, and continuous and exhibits a greater intensity and duration in the audio channel, not unlike the sound of moving the stethoscope from the left apex to the left base. Turbulent flow signals in the main PA are disrupted briefly in early systole because of PA ejection. The CWD examination demonstrates continuous, high-velocity flow directed mainly above the baseline, but often with a negative RV ejection component superimposed. The peak velocity occurs near the end of systole, just after the T-wave and trails off slightly during late diastole. Good alignment with the ductal flow will yield a peak velocity of 4.5 to 5 m/s, indicating preservation of the normal aortic to PA pressure

difference (and ruling out significant PH). The PG can be roughly estimated using the simplified Bernoulli relationship. Velocities <4 m/s are most often from suboptimal alignment or lower systolic pressure due to tranquilizers or physiologic hypotension encountered in some in very young animals. Dilation of the main PA often leads to pulmonary regurgitation, which is of low-velocity in left-to-right shunting PDA.

It is possible to estimate left to right shunting (Q_p/Q_s) using Doppler methods or combinations of 2D or ventricular volume estimates and PW Doppler of the aortic valve. This has not been validated in dogs with PDA, and there seems to be little added to this assessment in dogs earmarked for ductal closure. One potential value of estimating shunt ratio might be for the dog or cats with concurrent subaortic stenosis and PDA, where the relative contribution of flow across the SAS to the measured PG might be clarified.

Most cases of PDA also demonstrate MR and AR to varying degrees, related to the LV and aortic dilation. As venous return increases, the mitral and aortic inflow and outflow velocities become higher, often exceeding 1 m/s and 2 m/s, respectively. Pulmonary venous flow can be enhanced, with prominent D-wave observed. The aortic ejection velocity can exceed 3 m/s simply from high flow volume in the ascending aorta. This makes the diagnosis of concurrent, mild SAS difficult in predisposed breeds such as Newfoundland dogs. After ductal closure, LV outflow velocities are normal although aortic dilation can persist.

Imaging in Reversed PDA In the author's experience, when PA pressures are measured an intervention; it is uncommon to observe systolic pressure exceeding 35 mm Hg in a dog catheterized at sea level. In cases evaluated at altitude (>2,000 meters) mild to moderate pulmonary hypertension (**PH**) is more common, and that is also true when older dogs with PDA or cats are studied. However, true reversed PDA is not very common and for practical purposes can be excluded if the cardiac murmur is continuous and the RV cavity is not hypertrophied.

When severe PH does complicate PDA, the LA and LV appear normal to small in size while the RV is thickened, the RA enlarged, and the PA and its main branches dilated. As a reverse PDA is generally unrestrictive, the duct is often imaged clearly by 2DE as it extends. Shunting in this syndrome is most always bidirectional, low in velocity, and often difficult to separate from antegrade PA flow. With careful PWD mapping and imaging from each side of the thorax, the diagnosis is usually straightforward.

Placing the sample volume on the ventricular side of the pulmonary valve will often yield a positive diastolic flow signal of high-velocity pulmonary insufficiency that can be quantified using CW Doppler. Conversely, placement of the sample volume at the PA bifurcation records low-velocity (<2 m/s), bidirectional flow. Left-to-right shunting might be facilitated by oxygen inhalation or administration of sildenafil. This is especially likely in cats where reactive vasoconstriction appears to be a major contributor to PH.

Table 2 – Echocardiographic Evaluation of Patent Ductus Arteriosus

- 2D and Doppler Evaluation of the ductus arteriosus, aorta, pulmonary trunk and branch arteries
 - Identify the PDA and associated cardiac malformations (especially in cats)
 - Rule out aortopulmonary window, coronary artery to PA shunt, arteriovenous malformations
- 2D (\pm 3D) Evaluation of ductal morphology (multiple classification systems)
 - Subjective evaluation of the ductal origin, “angle” towards the PA, and length
 - Subjective evaluation of distal ductal tapering & entry into the PA (from right & left thoraces)
 - Optional: classify the ductus (which classification should be used in Vet Medicine?)
 - Objective measurements of the ductal ampulla and minimal ductal diameter
 - Subjective evaluation of ductal flow and turbulence by CDI
 - Objective measurement of shunt velocity during systole & diastole using CW Doppler
 - In right-to-left PDA careful measurement of shunt direction(s) and velocity by PWD
- 2D assessment of the right ventricular outflow tract and pulmonary trunk for:
 - Current PS or changes in the PA ejection profile (from precapillary pulmonary hypertension)
 - CDI evidence of pulmonary regurgitation
 - In right-to-left PDA careful measurement of pulmonary regurgitant velocity by CW Doppler
 - In cases of pulmonary hypertension, assess the RV fully and tricuspid valve for TR (velocity)
- Evaluation of the left ventricle, mitral valve, and aortic valve by 2D, M-mode & Doppler imaging
 - Identify subjective evidence of volume overload (sphericity)
 - Measure ventricular size (linear dimensions)
 - Estimate LV diastolic and systolic volumes (Simpson’s method of discs or 3D volumes)
 - Calculate LV shortening fraction (linear) and Simpson’s LV ejection fraction
 - Scan the septum for a ventricular septal defect
 - Evaluate the subaortic vestibule & aortic valve; measure the aortic annulus by 2D methods
 - Evaluate the aortic valve for regurgitation & record the LVOT velocities by CW Doppler
 - Expect increased velocities in a large left-to right shunt
 - Rule out (if possible) aortic stenosis
 - Evaluate mitral valve anatomy, motion & function with 2D (\pm 3D) imaging & Doppler studies
 - Evaluate mitral valve competency with CDI and CW Doppler
 - Measure transmitral inflow velocity (expect increased velocities from shunting)
 - Evaluate for functional MR versus concurrent mitral valve malformation
 - Optional: estimate Qp:Qs using Doppler or combined Doppler 2D methods (value?)
 - Optional: record tissue Doppler of ventricular function (value with increased preload?)
- Evaluation of the left atrium by 2D & Doppler Imaging
 - Subjectively evaluate for rounding and remodeling
 - Measure LA diameter; index to aortic root; optionally estimate LA volume
 - Optional: measure pulmonary venous flow patterns (often prominent diastolic wave)
- In bidirectional or reversed PDA
 - Perform saline contrast study with imaging over the abdominal aorta if diagnosis is uncertain

- Evaluate the right heart including PA pressures based on TR and PR jets
 - Transcatheter ductal closure:
 - TEE guidance if available to determine ductal morphology (“classification”), dimensions of the ampulla & minimal ductal diameter at opening to left PA.
 - Guide delivery of the guiding catheter or sheath, and positioning and deployment of the Amplatz canine ductal occluder (ACDO), vascular plug, or thrombogenic coil.
 - Assess ductal closure by CDI.
 - Post-procedure:
 - Assess closure and residual flow. Evaluate for thrombosis or movement of devices.
 - Assess left heart size and function. Include long-term follow up if LV function was moderately reduced, or if there was congestive heart failure, atrial fibrillation, VSD, or concurrent mitral or aortic valve disease.
-

Dogs with established, reversed PDA often improve clinically on PDE-V inhibitors, but there seems to be little impact on the direction of shunting. If fortunate enough to capture the actual reversal in a young dog, sildenafil effect on vascular resistance can be substantial and stabilize the patient sufficiently to allow ductal closure. If the diagnosis is uncertain, it can be helpful to perform saline contrast echocardiography using a cephalic venous injection while recording images over the heart and then the abdominal aorta. The appearance of saline contrast in the aorta, but not in the RA or RV, points to right-to-left shunting at the level of the great vessels.

Interventional Guidance Minimal ductal diameters typically range from 2.0 to 6 mm (with a wider overall range) as measured by TTE or TEE. Unfortunately, artifacts created by lung tissue can obscure the lesion when imaging by TTE. Moreover, and the duct is often truncated using TTE (and when in doubt most examiners guess 3.5 mm minimal ductal diameter ☺). Experienced centers do report good correlation with angiography and TTE; however, this is not a universal experience and considering the “average” PDA is about 3.0 to 4.5 mm even a 1 mm difference could be meaningful. Some studies have shown ampulla measurements are more likely to diverge from angiographic measures. Typical ampulla dimensions are around 7 to 9 mm, but these values depend very much on breed and patient size. Changes in arterial blood pressure during general anesthesia might also influence these comparisons.

When there is uncertainty about minimal ductal diameter, angiography or TEE can be used to provide a clearer image and guide device selection. Determining the diameter of the ductal ampulla is also relevant for delivery of the Amplatz canine ductal occluder (ACDO) or a vascular plug. Most operators select a minimal device diameter of approximately twice the minimal ductal diameter but this can be modified by the shape and size of the ampulla. Generally, this structure is very flexible and can carry a much larger proximal (aortic side) disc. For example, placing a 14 mm proximal disc in an 8 mm diameter ductal ampulla would not be surprising (so long as the device assumes its preformed shape). However, too large of a device can potentially stent the duct open, obstruct or dissect the left PA, or theoretically

tear the ductus.

The availability of a TEE Echo system, matched to the patient size, influences the approach taken for an interventional catheterization. As device delivery is usually from the femoral approach, the scope can be maintained during the procedure without complicating the surgical field. Minor manipulations are often needed to facilitate angiography or allow insertion of a marker catheter. In the author's hospital, the typical TEE image plane shows the aorta, ampulla, duct, pulmonary artery and pulmonary valve from near to far field. Some alternative views are obtained likely related to ductal anatomy, thoracic confirmation, and position of the scope crystals. Extreme flexion of the scope is not usually needed and general manual rotation of the scope along its long axis is often the critical maneuver. No consistent angle has been used in terms of biplane rotation.

With TEE imaging, ductal morphology is clearer and the higher resolution images permit measuring ductal dimensions with more assurance. Although color bleeding and filling must be avoided, CDI can also be helpful for determining ductal diameter. Biplane or 3D imaging also permits delineation of the "shape" of the PDA in cross section (round, oval, or something else), which might influence device selection. Multidimensional imaging has been suggested as a more objective classification system than some prior proposals based only on angiography.

TEE can monitor delivery of the guiding catheter or sheath (this passage sometimes makes the edges of the duct clearer). Echo can monitor the positioning and deployment of the ACDO discs, a vascular plug (delivered in the ampulla), or a thrombogenic coil. Ductal closure is assessed by CDI. The author uses both angiography (through the sheath) and CDI to determine closure. The first assessment after delivery of both ACDO discs (or a plug) is made after 5-minutes (the PDA is usually closed); and if necessary again at 10 minutes (most always closed); and if needed at 15 minutes (PDA is "always" closed or one better reassess the device type, size and placement). After flow is assessed, the restraining cable is then unscrewed and retracted back to the sheath. This often results in slight repositioning of the device ventral and caudal and can remove any distortion on the aortic side disc.

After the procedure/Follow up TEE and TTE (the next day) can verify closure and document any residual flow (common with surgical closure and coil embolization; infrequent with an ACDO). Complications such as thrombosis, PA dissection or obstruction, or movement of the device should be ruled out. The assessment of left heart size and function can be somewhat confusing post-PDA closure. Again, it is important to measure ventricular function in a number of ways and not rely on M-mode recordings. Mild depression of LV systolic function is relatively common post closure, but is rarely an issue. As previously noted, a longer follow up is justified if there was CHF or atrial fibrillation, another shunt, or concurrent valvular disease.

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Plugging the gap between ACDOs and Coils: Experience with the Amplatz Vascular plug-2

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The Amplatz Vascular Plug-2 (AVP-2) is a self-expandable nitinol mesh occlusion device, designed to be introduced in a minimally invasive fashion, through a catheter similar to the Canine ACDO. The main difference of the AVP-2, when compared to the canine ACDO, is its symmetric design comprising three disks (small disk, large disk, small disk) and absence of a distinct “waist” dimension between the disks. This symmetric design allows for both venous and arterial access for device positioning within the PDA, which may be advantageous in certain circumstances (e.g. very small patients).

I will briefly describe the technique of the minimal invasive PDA closure using the Amplatz Vascular Plug-2, with an emphasis on using the percutaneous trans-venous approach. The device was used for PDA closure in 26 dogs with body weights between 1.2-53.8kg over the last 2.5 years. Experience and outcome will be discussed..

Development and use of interventional clinical audit – a tool for everyone?

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Clinical audit was introduced by the NHS in 1989. It is a process of improving the quality of healthcare, by reviewing the delivery of care and making change to continually achieve best practice. It is now part of the clinical governance in the NHS. Whilst there is clinical audit within the veterinary practice, it is very much still in its infancy with a need for progress and development.

Clinical audit is often confused with research. Particularly by professionals with a research knowledge. Research aims to answer a hypothesis with particular aims and objectives and patients allocated to different groups. Audit asks the questions “does this service reach a predetermined standard?” and thus determine improvements are required.

There are many types of audit from monitoring processes targeting quality improvement and patient safety within a hospital through to significant event audit, peer review, adverse events, patient surveys etc. Each type of audit is likely to be attractive to different mindsets. The type of audit we wish to discuss is ‘outcome clinical audit’, a standards-based audit. This can apply to many aspects of our clinical work, in this example we discuss its use in cardiac interventions.

One of the hurdles to undertaking clinical audit is the perception of its difficulty or perceived usefulness. Not unlike the perception GPVets might have in their opinion of ECGs! It’s often more of a psychological barrier that needs to be overcome.

The British Cardiac Interventional Society’s website publishes the results of clinical audit from their members, cardiac interventionalists, which is open access to the public and their patients. <https://www.bcis.org.uk/patient-area/> Clinical audit data is gathered and analysed by NICOR (National Institute of Cardiovascular Outcomes Research) <https://www.nicor.org.uk/>

In developing a clinical audit tool within our cardiology unit, it needed to be easily accessible, easy, quick and simple to use and produce useful measures. This was achieved using an Excel document, with a tab for each of the procedures performed. The table simply included a patient identifier with basic information, and then outcome measures. Each of the outcomes required a definition of the standard, which was established from known publications. Once each dataset is entered, the top-row automatically changes to give the percentage success or complication on a continuous and rolling basis. Once the audit tool

was up and running, it became remarkably easy to complete, taking only a minute at the start of a procedure to enter the patient details, some information was then entered on complication of the procedure such total radiation dose, and the rest of the information was entered at the time of discharge. All entries within a column with a heading of a measure (an automated percentage) must be entered in solely numerical binary information (1 = yes 0 = no).

With expansion of our cardiology team and recruitment of junior colleagues, the clinical audit tool has allowed us to internally-audit, self-audit and provide benchmarking that can be compared to other centres and published literature, to then allow implementation of measures to improve success and/or reduce complications and/or risk factors.

The key outcomes determined prior to clinical audit collection included patient outcomes, operator safety data and procedural complications. In this talk we will present this data and both its applicability to clinical cases, including for referring clinicians and well as other tertiary referral centres.

The use of this clinical audit tool could readily be implemented across interventional referral centres nationwide and provide a registry for all veterinary cardiac interventions and provide a strong body of evidence that the veterinary world has yet to see. This is common practice in the NHS and has been used to audit PCI (Primary Coronary Interventions) as the British Cardiovascular Intervention Society Database.

Acknowledgements

The Clinical Audit Support Centre were instrumental in helping me understand and develop a clinical audit process in interventional cardiac procedures. They have a very informative and useful website, which is an excellent first port of call and more importantly run a number of training days that are worth attending. <http://www.clinicalauditsupport.com/>

<https://www.dropbox.com/sh/x91mee308hv6r0c/AAAu8XIKRvS6rZJAo6Aieip-a?dl=0>

ACVIM 2019 Update

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Abstract Summaries:

Hemodynamic, Echocardiographic, and Sedative Effects of Oral Gabapentin in Healthy Cats

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Gabapentin is being used more frequently to decrease perceived anxiety in cats during veterinary visits. Many sedative and anxiolytic drugs have haemodynamic effects that may bias data acquired during the cardiology investigation. However, the haemodynamic effect of gabapentin in cats is currently unknown.

The aims of this study were to assess the effects of gabapentin on blood pressure measurement, heart rate and echocardiographic parameters in healthy cats.

Twenty-four cats were investigated for healthy status and cats that met these criteria received either gabapentin or placebo. Between 30 and 120 minutes after capsule administration, complete physical examination, Doppler BP, and echocardiography were performed. Following a washout period of 7 days, the same cats then represented having been given the alternative capsule and were assessed in the same manner. Vital parameters and echocardiographic measurements at baseline, post-gabapentin, and post-placebo were compared statistically. Sedation score was calculated independently by two observers and interobserver agreement was calculated. Finally, correlation between variables was assessed.

Following health assessment, 14 cats were excluded, and the study population consisted of 10 adult healthy cats. No cats experienced adverse events during the study. Seven of 10 cats exhibited sedative effects post-gabapentin administration within 120 minutes. Near perfect interobserver agreement on sedation score was obtained and no significant correlation was found for gabapentin dosage with sedation scores at any time point.

With regard to the parameters heart rate, respiratory rate and Doppler BP measurement, there was no significant difference between those recorded at baseline, post-placebo or post gabapentin. However, there were echocardiographic parameters that differed significantly between baseline measurement and post-gabapentin administration including left ventricular fractional shortening, left ventricular diameter in systole and left atrial volume. However, these measurements remained within established reference ranges. For the

remaining echocardiographic parameters, which included Doppler measurements, no significant differences were found between the three time points baseline, post-placebo and post-gabapentin.

These results suggest a possible reduction in systolic function following administration of gabapentin to healthy cats. However, a further study with a larger population is needed to fully evaluate the haemodynamic effects of gabapentin.

Diagnostic Utility of Point-of-Care Lung Ultrasound for Monitoring Congestive Heart Failure in Dogs.

Shane Murphy¹; Teresa DeFrancesco²; Melissa Tropf¹; Jennifer Fowler¹; Rebecca Walton¹; Wendy Ware¹; Jessica Ward¹

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Point-of-care lung ultrasound is a commonly used technique in the diagnosis of left-sided congestive heart failure (CHF) in dogs. The use of this technique is predicated on the assumption that the co-presence of severe left sided heart disease and B-lines is indicative of cardiogenic pulmonary oedema.

The objective of this study was to determine whether LUS could be used to monitor the resolution of cardiogenic pulmonary oedema following initiation of therapy.

Twenty-four client-owned dogs that presented with CHF were prospectively enrolled. A LUS protocol was performed at hospital admission, discharge and at subsequent recheck examinations. LUS findings were compared between these time points. The correlations between LUS and other measures of response to therapy including respiratory rate, thoracic radiography, and NT-proBNP concentration were also calculated.

Between hospital admission and discharge, the median number of sites strongly suggestive of B-lines (>3 B-lines per site) reduced from 5 to 1 and the median total number of B-lines reduced from 39 to 4.5. LUS indices continued to decrease to the time of recheck and remained low during subsequent rechecks. There was a positive correlation between LUS indices and respiratory rate at both admission and discharge. Radiographic pulmonary oedema score was also correlated with LUS indices at admission and decreased over subsequent time points. NT-proBNP decreased between hospital admission and discharge but was not correlated with respiratory rate or LUS findings at any time point.

In conclusion, B-lines decrease significantly within 24 hours of CHF treatment and therefore LUS can be a useful tool for monitoring improvement of left sided CHF in dogs.

Consensus Statement: Pulmonary Hypertension

C. Reiner, (Chair), H. Kellihan, I. Masseur, E. Rozanski, L. Visser

This session consisted on the presentation of the report for the "Guidelines for classification, diagnosis and treatment of pulmonary hypertension in dogs", generated by an ACVIM consensus panel.

The proposed classification scheme for dogs has been designed based on the human classification of pulmonary hypertension. Following its guidelines , pulmonary hypertension can be subdivided into five groups according its primary cause: (1) pulmonary arterial hypertension, (2) pulmonary hypertension secondary to left heart disease, (3) pulmonary hypertension secondary to lung disease and/or hypoxia, (4) pulmonary thromboembolism and (5) pulmonary hypertension with multifactorial and/or unclear mechanisms.

These clinical groups are useful for diagnostic evaluation and appropriate therapy.

In order to determine the probability of pulmonary hypertension, the velocity of the tricuspid regurgitation should be assessed, however not exclusively. The following echocardiographic anatomic sites should also be assessed, and the following pathological findings detected:

- 1) Right and Left ventricle
 - a. Flattening of the interventricular septum (especially systolic flattening)
 - b. Underfilling or decreased size of the left ventricle
 - c. Right ventricular wall thickening (Hypertrophy with or without dilatation)
 - d. Right ventricular systolic dysfunction
- 2) Pulmonary Artery
 - a. Pulmonary artery enlargement ($PA/Ao > 1.0$)
 - b. Peak early diastolic pulmonic regurgitation velocity > 2.5 m/s
 - c. Right Pulmonary Artery distensibility index $< 30\%$
 - d. RV outflow Doppler acceleration time ($< 52-58$ ms) or acceleration time to ejection time ratio (< 0.30)
 - e. Systolic notching of the Doppler RV outflow profile (Caution: False positives are possible)
- 3) Right atrium and caudal vena cava
 - a. Right atrial enlargement
 - b. Enlargement of the caudal vena cava

The panel proposes that the probability of a dog having pulmonary hypertension can be classified as low, intermediate or high, by combining the Tricuspid regurgitation velocity and the number of anatomic sites with echocardiographic evidence of pulmonary hypertension.

Regarding treatment of the pulmonary hypertension, the panel has advised to start by addressing the underlying cause. To aid the stabilisation of these patients, the following medications can be considered: Nitric oxide, phosphodiesterase 5 inhibitors, milrinone,

tyrosine kinase inhibitors, L-arginine and pimobendan (if there is evidence of right ventricular systolic dysfunction).

Consensus Statement: Diagnosis and Classification of Feline Cardiomyopathies

V. Luis Fuentes (Chair), J. Abbott, V. Chetboul, E. Cote, P. Fox, J. Haggstrom, M. Kittleson, K. Schober, J. Stern

In this session, the classification based on echocardiographic findings and treatment recommendations for feline cardiomyopathies were presented.

The need for a specific classification for feline patients resides on the difficulty to apply the human classification to cats, predominantly because in the feline patients the underlying cause of the cardiomyopathy is unknown, because there is heterogeneity of the different phenotypes of cardiomyopathies and due to considerable overlap between the phenotypes. Furthermore, there is disease progression over the years, which is often associated with remodelling, and therefore of a change in the echocardiographic appearance and function of the heart.

In order to appropriately stage the disease, the panel proposes dividing the cases into the following stages:

A: cats predisposed to feline cardiomyopathies

B1: Subclinical cats, with normal to mildly enlarged atria. These cases are considered to have a lower risk of congestive heart failure and aortic thromboembolism.

B2: Subclinical cats with moderate to severe atrial enlargement. These cases are considered to have a higher risk of congestive heart failure and aortic thromboembolism.

C: Cats with current or previous history of congestive heart failure and aortic thromboembolism

D: Cats in congestive heart failure that are refractory to treatment.

This staging applies to all forms of cardiomyopathies, however the panel has proposed that different phenotypes are considered when classifying feline cardiomyopathies.

The first one is the Hypertrophic cardiomyopathy (HCM) phenotype, which includes cats with increased left ventricular wall thickness in the absence of congenital aortic stenosis or systemic hypertension. Within this phenotype, dynamic left ventricular outflow obstructions or abnormalities of mitral valve and papillary muscles can be commonly found.

Following, there is the Restrictive cardiomyopathy (RCM) phenotype, characterised by echocardiographic evidence of atrial enlargement with diastolic dysfunction and reduced left ventricular compliance. This phenotype can be subdivided into a Myocardial form (with left ventricular wall thickness and chamber within normal limits, left atrial or bilateral atrial enlargement and restrictive diastolic function) and an Endomyocardial form (with endocardial scar bridging between the left ventricular septum and free wall, possibly causing left obstruction and apical aneurism and left atrial or bilateral atrial enlargement).

The third phenotype described by the panel is the Dilated cardiomyopathy (DCM) phenotype. This form presents with left ventricular systolic dysfunction and an increase in ventricular and atrial dimensions.

An Arrhythmogenic right ventricular cardiomyopathy (ARVC) phenotype has also been described, consisting in severe right ventricular and atrial dilation, with right ventricular systolic dysfunction and wall thinning. Arrhythmias are commonly described in cats with this phenotype.

The former Unclassified group was replaced with a Non-specific phenotype, that should be accompanied by a description of the cardiac morphology and function.

Regarding the diagnosis of these cardiomyopathies, this the panel recommends the use of echocardiography, ideally by a cardiologist although a non-specialist echocardiographic examination is recommended when there is no possibility of achieving the first. The use of bedside echocardiography was recommended for unstable patients. An electrocardiographic exam is recommended in the presence of arrhythmia. Other tests that can be performed include cardiac biomarkers, thoracic radiography and further blood analysis (to rule out complicating comorbidities, such as anaemia, and hyperthyroidism).

Regarding treatment, the panel has made recommendations for patients in the following stages:

- B2
 - clopidogrel and/or aspirin for thrombi prevention in cats with left atrial enlargement;
 - pimobendan and/or ACEI if in the presence of systolic dysfunction;
 - atenolol can be considered in cases with severe left ventricular outflow obstruction;
 - antiarrhythmic medication can also be used for severe tachyarrhythmias;
- C and D
 - all of the previously mentioned medication;
 - sedation, oxygen supplementation, furosemide and thoracocentesis as required for acute onset congestive heart failure.
 - pimobendan or dobutamine for hypotensive, hypothermic and low output disease
 - Torasemide in cases of refractory congestive heart failure

Acute pharmacodynamic Effects of Pimobendan in 22 Client-owned Cats with Hypertrophic Cardiomyopathy

Maureen S. Oldach, Yu Ueda, Eric S. Ontiveros, Samantha L. Fousse, Samantha P. Harris and Joshua A. Stern

This abstract followed a randomized, placebo-controlled cross over study, using colony cats that showed improved left atrial function and no change in left ventricular outflow tract after a single dose of pimobendan. The population of cats from the previous study were very sympathetically driven, requiring sedation and this was considered a limitation, having prompted the present study.

Pimobendan increases sensitivity of cardiac troponin C to calcium (having positive inotrope properties) and simultaneously inhibits phosphodiesterase 3 (causing vasodilation). Its benefit in the management of dogs with dilated cardiomyopathy and myxomatous mitral valve disease has been well documented. When it comes to the use of pimobendan in cats, there is very little pharmacodynamic data in the literature.

Feline Hypertrophic Cardiomyopathy (HCM) is the most common cardiac disease in cats. It is characterised by diastolic dysfunction and can occur with preserved or hyperdynamic systolic function. As a complicating factor, 47.5–67% of HCM cats presenting to referral hospitals have concurrent left ventricular outflow tract obstructions (LVOTO).

Considering Pimobendan's mechanism of action, there is a concern that this medication may exacerbate or induce these dynamic obstructions in cats with HCM, however, some clinical benefit has been reported in cats with CHF that were receiving pimobendan.

The main objective of this study was to assess the acute pharmacodynamic effects of a single oral dose of pimobendan in cats with HCM using echocardiography, with a specific aim to investigate whether pimobendan could induce or exacerbate LVOTOs. The authors hypothesized that pimobendan would not exacerbate LVOTO and would have positive left atrial function effects and that it would be well tolerated by cats with HCM.

The population of this study consisted in 22 client-owned normotensive, euthyroid adult cats with HCM. The authors have included cats with left ventricular hypertrophy in the absence of other forms of cardiac disease, that were amenable to handling without sedation and were not receiving medication that could affect cardiac function.

This was a prospective study. An echocardiographic examination and Doppler blood pressure assessment were performed before (pre-treatment) and 90-minutes after (post-treatment) oral administration of 1.25 mg of pimobendan. Echocardiographic measures were performed by a single investigator blinded during off-line analyses.

Heart rate, systolic blood pressure, left auricular blood flow velocity, left ventricular fractional shortening, and tissue Doppler peak diastolic velocity of the lateral mitral annulus were not significantly different between pre-treatment and post-treatment groups. The number of cats with LVOT obstructions was not significantly different between groups. No adverse effects were observed in this study. Median LVOT velocity was not significantly higher following pimobendan [1.9m/sec (1.5 - 3.4) vs 3.08m/sec (1.98 – 3.99); $p = 0.07$]. Mean (\pm SD) left atrial fractional shortening was significantly higher following pimobendan ($28.9\% \pm 6$ vs. $32\% \pm 7$; $p = 0.016$). Mean tissue Doppler peak early systolic velocity of the lateral mitral annulus was significantly higher following pimobendan ($7.4 \text{ cm/s} \pm 1.5$ vs. $8.5 \text{ cm/s} \pm 1.6$).

One clinical benefit from pimobendan could be the observed improvement of left atrial function, as this can be important in reducing the risk of thrombi formation. The observed mild increase in LVOT and RVOT velocity occurred simultaneously with an increase in heart rate, which could be due to sympathetic stimulation caused by the repeated examination.

The limitations of this study include the timing of the single time-point assessment of the echocardiographic changes with pimobendan administration, as this may have not coincided with the time of peak effect of the medication, as we are currently unaware of when this may be. Another limitation, likely related to their efforts of having non-sedated cats, was the difficulty to assess the diastolic function when they displayed tachycardia. Furthermore, there may have been a considerable amount of sympathetic stimulation in the second exam that may have biased the results, and the fact that they did not use a control population meant that they were not able to observe this in a cat that did not receive pimobendan.

In conclusion, in cats with HCM, pimobendan acutely increased echocardiographic measures of left ventricular function, left atrial function and LVOT velocity without causing adverse effects.

Comparative atrial arrhythmias

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OVERVIEW

Atrial rhythm disturbances are common in dogs, cats, horses and humans, with a substantial veterinary experience in managing these arrhythmias, particularly in equine and canine patients (see reference list). While some differences in epidemiology, underlying causes, comorbidities, complications, and management approaches are evident across species, there are also many commonalities. The human literature regarding atrial arrhythmias is extensive. In the ATRIA study of nearly 2 million patients, the overall prevalence of atrial AF was 1% with 70 percent of the cases at least 65 years old; the prevalence was even higher in aged adults. In contrast, AF is comparably rare in cats with the largest multi-institutional survey including just 50 cases over a 23-year period. In a 1969 survey of UK horses conducted by Professors Holmes, Darke, and Else, the incidence of AF was about 2.3% of 1187 horses with different groups sampled. The percentage of (presumably healthy and younger) Army horses with AF in that study was lower, at 0.93% of 431 while that from an abattoir sample was 1.8%. The prevalence of AF in dogs depends strongly on the study sample (i.e., a cardiology clinical population versus a general practice population). Patient age, body size, and breed or race are additional factors (see below). Although it is logical to assume that equine and canine breed differences are largely weight-related, that might not be the case as racial differences in the prevalence of AF have been reported in people as well as familial clusters and genetic mutations predisposing to atrial arrhythmias. Systemic inflammation as measured by C-reactive protein has been associated with AF in humans. This relationship in animals is unknown, but in one study of horses with AF this biomarker was not increased.

This presentation will offer a high-level consideration of atrial arrhythmias and address premature atrial depolarizations or complexes (**PACs**), focal atrial tachycardia (**FAT**), atrial flutter, and atrial fibrillation (**AF**). Other forms of supraventricular tachyarrhythmias, specifically multifocal atrial tachycardia and atrioventricular (**AV**) reciprocating tachycardias will not be considered. The major emphasis for this presentation is atrial fibrillation. Dr. Van Loon will also be considering equine atrial arrhythmias during his presentation. (Logically, the species not treated by the author will be used only for comparison.)

Clinical Pathophysiology Across ranges of age, the clinical outcomes and prognosis for sustained atrial arrhythmias in dogs, cats, horses and humans relates largely to cardiac comorbidities, although the prognosis of idiopathic (lone) AF is not always favorable. Sustained atrial arrhythmias can affect patients in a number of ways. One overarching principle pertains to the negative effects of losing *atrial pump function* when there is *structural cardiac disease especially when the heart rate (HR) increases* with activity, exercise,

or sympathetic tone. The reductions in preload, stroke volume and cardiac output, along with increases in left atrial (LA) pressure, contribute to the exercise intolerance observed with chronic or paroxysmal AF. We also recognize that cardiac decompensation is prompted by AF in the setting of serious structural heart diseases; this includes congenital and acquired valvular diseases and various forms of cardiomyopathy. For example, the major reason for congestive heart failure (**CHF**) in horses is the combination of AF superimposed on valvular or congenital heart disease. Similarly, stable dogs with myxomatous mitral valve disease (**MMVD**) or dilated cardiomyopathy (**DCM**) frequently develop CHF once atrial flutter or AF supervene. In the setting of ventricular diastolic dysfunction, as with hypertrophic cardiomyopathy, AF can precipitate pulmonary edema. Atrial fibrillation is also notorious for causing deterioration of patients with mitral or tricuspid stenoses, where the loss of atrial kick, irregular diastolic cycles, and inappropriate increases in exercise heart rate conspire to increase mean gradients and atrial pressures.

Conversely, idiopathic or lone AF – that occurring without overt structural heart disease – can be well tolerated, especially in horses, dogs, and humans provided the cardiovascular system is untaxed by exercise or pregnancy and (for humans) the risk of thrombotic stroke is managed. However, the long-term outcome of lone AF in dogs is less certain, and atrial arrhythmias clearly represent an early stage of impending cardiomyopathy in some breeds, with Irish wolfhounds offering a prominent example. Some reports in humans suggest that lone AF might also carry a higher risk for morbidity and mortality if untreated (see references).

Not surprisingly, the least is known about atrial arrhythmias in cats, where even an accurate rhythm diagnosis of AF can be a challenge due to their tiny P-waves, unapparent fibrillation waves, and propensity to develop intraventricular conduction disturbances with widened QRS. Although lone AF has been reported in cats, their small stature dictates that sustained atrial arrhythmias will mainly occur in the setting of severe structural disease. Typically, this is end-stage hypertrophic cardiomyopathy, restrictive cardiomyopathy, or a congenital malformation such as mitral stenosis. Similar to people, but unlike horses and dogs, AF and atrial dilation present substantial risks for atrial thrombosis and thromboembolic complications.

The hemodynamic consequences of AF – reduced ventricular filling despite increased venous pressures and decreased cardiac output – pose clinical consequences. The impact of AF on exercise performance and rider safety is at the forefront in performance horses, and the potential for further electrical instability during exercise has been raised through clinical reports and exercise electrocardiogram (**ECG**) tracings. Similarly, in people lone AF can limit functional capacity. Holter ECG monitoring of dogs with lone AF is variable inasmuch as some have nearly normal average daily heart rates (~70 to 80/minute) with reasonable increases in exercise HR; whereas, other dogs have inappropriate daily and exercise rates. High ventricular rate responses to a sustained FAT, atrial flutter or AF poses a small but finite risk for tachycardia-induced cardiomyopathy.

Etiology & Clinical Associations Awareness of the causes and risk factors for atrial arrhythmias is central to the patient evaluation and workup. In most veterinary cases, atrial

arrhythmias are caused by structural heart disease, including congenital malformations, chronic valvular diseases, myocardial disorders, and pericardial conditions. These lead to atrial stretch, ischemia, and fibrosis of atrial myocardium. Consequences include longer atrial conduction times and altered electrophysiology (**EP**) that includes afterdepolarizations, macro- and micro-reentry, and myocardial fibrillation. Both electrical and mechanical remodeling of the atria also develop with protracted atrial tachyarrhythmias. These can reduce the effectiveness of antiarrhythmic drugs and increase the risk for recurrence of the arrhythmia.

In some patients, atrial arrhythmias develop due to medical or iatrogenic disorders. Some examples include potassium or magnesium abnormalities, thyrotoxicosis (spontaneous in humans & cats; iatrogenic in dogs and horses), or from drug effects, including a risk associated with general anesthesia (in giant breed dogs). Atrial tumors and pericardial effusion – common diagnoses in older dogs – are an under-recognized cause of atrial arrhythmias. Increasing age and body size as well as male sex are associated with a higher risk of AF in humans, dogs, horses, and cats; sex also predisposes to some of the disorders increasing risk of AF. Body size presents an obvious predilection as seen by the rarity of AF in cats, the low incidence of AF in small breed dogs with even severe MMVD, the greater risk of AF in large canine breeds (with or without DCM), and the common diagnosis of lone AF in giant breeds of dogs, humans and horses. As an example, in the study by Westling and colleagues the odds ratio for the Irish wolfhound breed for AF was 41.65! Cor pulmonale and systemic thromboembolic disease are risk factors for AF in humans, but aside from case reports, pulmonary disease is not as clearly associated with AF in animals. Similarly, associations of human AF including coronary heart disease, systemic hypertension, alcohol ingestion, obesity, open heart surgery, and emotion are less important for our patients (but quite relevant to their caretakers). Strenuous exercise often precedes the recognition of AF in horses, but it is likely that AF was preexisting in many of these cases. Heart failure is both a cause and effect of atrial tachyarrhythmias.

Sometimes there is no obvious reason for the development of AF and it is therefore termed idiopathic or lone AF, accepting that the main diagnostic criterion for lone AF in veterinary medicine is a “normal echo”. Lone AF might represent a “primary” electrical disease, and it is certainly common in humans, horses and giant breeds of dogs. However, it is likely that many cases are simply due to myocardial disorders below our diagnostic limits of detection, and lone AF can foreshadow the eventual development of genetic cardiomyopathy. Larger atrial mass and perhaps muscular walls of veins entering the heart predispose to the development and perpetuation of atrial arrhythmias, which helps to explain the aforementioned influence of species and breed. Additionally, high vagal tone can open some potassium channels and shorten the atrial action potential, facilitating reentry. This is likely operative in horses and dogs, and has been identified following reflex mediated syncope in dogs. Vagotonia is also a putative mechanism for AF seen in extreme male athletes. Drugs (opiates, anesthetics) and genetic predisposition might also be factors, but are undefined in our patients.

Presumably, there are triggers and substrates operative in the induction and maintenance of atrial tachyarrhythmias. These might arise from cells in the atrium or muscle of the

pulmonary veins that exhibit abnormal automaticity, triggered activity (afterdepolarizations), or micro-reentry. Clinically there is a suggestion that canine and equine patients with frequent PACs seem to have a higher risk for developing AF. Many horses converted from AF to NSR continue to experience PACs or short runs of atrial tachycardia and later revert to AF.

Atrial tachyarrhythmias can be paroxysmal, recurrent, persistent, or permanent. They can also be short-lived and convert spontaneously to normal sinus rhythm, as often seen horses, dogs and people. However, in the majority of veterinary cases, these arrhythmias can persist and might require treatment. Many patients experience permanent AF, unresponsive to any form of antiarrhythmic therapy aside from heart rate (HR) control. This is the usual scenario with severe MMVD or DCM or in cats with cardiomyopathy associated with severe LA dilation. The author has observed long-standing atrial flutter in dogs with marked right atrial dilation from pulmonary stenosis, pulmonary hypertension or right ventricular cardiomyopathy. Persistent or permanent AF is especially likely if there is enlargement, stretch, or microscopic disease of the atria. Moreover shortening of the atrial cell refractory period can occur from electrical remodeling or perhaps an underlying channel disease. There are numerous EP studies on animal models of atrial arrhythmias, which are instructive, but currently only scant EP studies of *spontaneous* atrial arrhythmias in dogs and horses. Van Loon and colleagues have evaluated the cycle length of equine atrial arrhythmias using number of catheter based and novel (echocardiographic) methods and have begun mapping atrial arrhythmias with advanced imaging systems, (Some of these approaches will be presented later in this conference.) Similarly, Santilli has reported on atrial flutter and FAT in dogs using catheter-based techniques for recording and ablation (see references).

The underlying cause of an atrial arrhythmia is probably the most important factor determining the short and long-term prognosis and clinical management. When there is an identifiable cause for the rhythm disturbance, treating the condition and tincture of time might resolve the issue. When acute in onset, in-hospital antiarrhythmic therapies and electrocardioversion can be effective for converting atrial tachyarrhythmias. However, long-term maintenance of normal sinus rhythm (**NSR**) after spontaneous, drug-induced, or electrocardioversion is hampered by our lack of approved and effective drugs, the cost and availability of electrocardioversion, the frequent coexistence of structural heart disease, and a fundamental lack of understanding about the electrophysiology, triggering, and perpetuating factors operative in AF in our veterinary patients. Although physicians have published an extensive literature regarding atrial tachyarrhythmias in people, there is only limited information available for horses and dogs. As the author is not an electrophysiologist, the present discussion will center mainly on the clinical issues facing veterinary cardiologists in practice.

ATRIAL ARRHYTHMIAS – DIAGNOSTIC CRITERIA

This section offers a diagnosis summary with some pointers based on surface ECG traces (mainly from dogs and horses). Atrial rhythm disturbances include premature atrial complexes (PACs or APCs), focal (micro-reentrant) atrial tachycardias (FAT), macro-reentrant

atrial tachycardias (atrial flutter), and atrial fibrillation (AF). These must be distinguished from sinus tachycardia, and the less common (we believe) atrioventricular reciprocating tachycardias and nodal (junctional) tachycardias (see **Table**). As more Holter and EP studies are performed in animals, our assessment of arrhythmia prevalence could change. The availability of implantable micro-recorders (e.g. Reveal Linq®) should facilitate recognition of paroxysmal atrial arrhythmias.

The conduction sequence provides valuable diagnostic clues. Subtle electrical (QRS) alternans is common with regular SVTs regardless of mechanism, often appearing at the onset. This finding can help to separate a pathologic SVT from a “fast” sinus tachycardia (in which electrical alternans is uncommon) so long as the alternation is not an artifact from an exercise ECG. Supraventricular tachyarrhythmias also can be conducted with gross ventricular aberrancy and the resultant QRS complexes can be confused with PVCs or ventricular tachycardia (VT). A wide and irregular QRS tachycardia, without obvious P waves, might represent antegrade conduction of AF impulses down an accessory pathway. Physiologic AV block is commonly observed with FAT and atrial flutter and this can reveal the underlying atrial arrhythmia. This can be spontaneous, follow a vagal maneuver, or be induced by diltiazem or a beta-blocker. Importantly, an AV nodal response to adenosine typically seen in people is not observed in dogs, where very high doses are needed to achieve AV block.

Premature atrial complexes | Nonsustained atrial tachycardia The electrocardiographic (ECG) diagnosis of *premature atrial complexes* (PACs) and of nonsustained or *paroxysmal atrial tachycardia* is relatively straightforward. These arrhythmias are characterized by premature P' waves with a morphology that usually differs from sinus P-waves. The premature P' wave is followed by a normal to prolonged P-R interval that precedes a narrow QRS complex. Isolated, nonconducted PACs are common in horses. The P' is often blocked on the T-wave of the previous supraventricular complex whereas other P' waves conduct with first-degree AV block. Repetitive ectopic atrial activity (paroxysmal atrial tachycardia) might also be seen; often these are very short runs of 3 or 5 PACs and can end with a Wenckebach period and AV block.

Cycle-length dependent *ventricular aberrancy* in the form of widened QRS complex with ST-T changes or a manifest bundle branch block is observed with PACs in all species. Sometimes this is long-short cycle aberrancy, but this is variable. Subtle QRS aberrancy of conducted PACs is common along with secondary ST-T changes. Sometimes the first premature QRS complex of nonsustained FAT exhibits aberrancy, leading to confusion with premature ventricular complexes (PVCs). Ventricular aberrancy is quite common in horses with atrial arrhythmias. In the case of isolated PACs or short runs of atrial tachycardia, the diagnosis of aberrancy can be straightforward; however, widened QRS complexes are often observed with short cycle lengths on exercise ECGs, including atrial fibrillation, and it is very difficult to separate these from PVCs.

Sustained Atrial Tachycardia Sustained atrial tachycardias in dogs and horses often fit human criteria for *focal atrial tachycardia* (FAT) and usually exhibit positive P-waves in the caudal limb leads (in dogs) and the base-apex lead (in horses). The lower rates of FATs overlap

with those of sinus tachycardia in these species, and a normal P-wave axis and narrow QRS complex can foster diagnostic confusion. Focal atrial tachycardia can be induced experimentally in models of heart failure and likely represent a tachycardia stemming from afterdepolarizations or micro-reentry. In dogs most FATs originate along the terminal crest of the right atrium or within the triangle of Koch or atrial septum, but perhaps one-third are of left atrial or pulmonary venous origins. Mean(SD) cycle length of 238(69) were reported by Santilli in anesthetized dogs, for an average atrial rate of 252/minute. (It should be mentioned that cycle length and the resultant heart rates reported might be slightly depressed by anesthetic agents.) The author has frequently observed FAT in dogs at a rate of ~300/minute although most are somewhat lower. Electrical alternation of QRS amplitude is common if sought.

Table – Diagnostic Criteria for Atrial Arrhythmias

Cardiac Rhythm	Comments
Sinus tachycardia Normal mechanism with heightened sympathetic tone	Normal P-QRS relationship; often rate dependent changes in the P-wave (larger), PR interval (shorter assuming normal AV conduction) Secondary repolarization changes common (Ta wave; ST-T changes) Subtle R-R interval variation; response to increased vagal tone or beta-blocker Rate usually <290/minute in dogs
Premature atrial complexes (PACs) Atrial “echoes” (retrograde activation)	Can be challenging to distinguish from sinus arrhythmia with wandering atrial pacemaker or with “paired beating” from atria Premature P’ often buried in prior ST-T; Prolonged P-R interval is common Ventricular aberrancy might be seen, especially with PAT Retrograde (negative) P’-waves with atrial echoes from accessory pathways or from retrograde AV conduction of ventricular ectopics or paced beats
Atrial tachycardia Focal atrial tachycardia	Can resemble sinus tachycardia if origination is from the crista terminalis Typically: greater RP/PR with P-waves positive in left, caudal leads QRS alternans less common after initial start-up (dogs) Might terminate with Wenckebach periodicity or second degree AV block
Atrial flutter Macro-reentrant atrial tachycardia	Typical (counterclockwise) or Atypical macroreentrant form of tachycardia Wide range of manifest atrial rates; Often conduct 1:1 & 2:1 Regular conduction sequences cause difficulties in diagnosis (use Bix’s rule) AV node block usually reveals regular flutter waves AV conduction sequence can change rapidly
Atrial fibrillation	Resting ventricular rate variable; normal in lone AF; high in CHF Readily observed f’ waves in horses, usually in dogs; obscure in cats

	<p>Irregular ventricular response is expected except in cases of concurrent AV conduction disease (complete AV block with escape activity)</p> <p>QRS variation common in cats</p> <p>Autonomic tone the major factor in determining the rate response</p>
<p>Orthodromic atrioventricular reciprocating tachycardia (accessory pathway)</p>	<p>Labrador retrievers predisposed; can have tricuspid dysplasia</p> <p>Wide range of rates; can be >300/min in dogs; QRS alternans is common</p> <p>PAC or long PR prior to SVT with retrograde P'-waves in ST-segment.</p> <p>Smaller ratio RP/PR; absence of second degree AV block</p> <p>Positive P' waves in leads aVR and aVF</p> <p>Presence of ventricular pre-excitation an (uncommon) clue to diagnosis</p> <p>Electrophysiologic study often needed for diagnosis</p>
<p>Nodal/Junctional tachycardia</p>	<p>Rare except with digitalis toxicity</p> <p>Sometimes seen in Labrador retrievers</p> <p>Can be confused with "high", narrow-complex ventricular tachycardia especially in horses (look for A-V dissociation)</p>

Distinguishing a sustained (focal) atrial tachycardia from atrial flutter can be difficult from the surface ECG as the ECG features of FAT and flutter sometimes overlap and large Ta waves can mimic flutter waves. Van Loon has indicated a similar situation in equine cases. This confusion relates in part to the wide ranges in both atrial cycle lengths and ventricular rates reported with these arrhythmias and the fact that atypical atrial macro-reentry might not present with distinctive saw-tooth waves (see references). Similar to atrial flutter, is the potential for physiologic first-degree and second-degree AV block; the latter often occurs spontaneously, after a vagal maneuver or administration of a beta-blocker or calcium channel antagonist. Orthodromic atrioventricular reciprocating tachycardia also can be confused with both paroxysmal and sustained FAT, but ectopic atrial tachycardias typically shows a shorter P-R compared to R-P interval.

Atrial Flutter Atrial flutter represents a (counter-clockwise) macro-reentry tachycardia circuit involving the right atrium. The ECG baseline in classic atrial flutter is characterized by saw-toothed waves and a regular or irregular ventricular response rate. The atrial rate of 170 to 275/minute has been reported in horses, and flutter rates approaching 400/minute have been observed in spontaneous canine cases. Loss of the isoelectric shelf is not a requirement for the diagnosis. Both right and left atrial activation can influence the appearance of the baseline on the surface ECG. A number of atypical atrial flutter patterns are also recognized in humans. The A:V conduction sequences in atrial flutter can vary widely from 1:1 to 8:1 and can change rapidly.

Whereas the ventricular response is nearly "always" irregular in AF, with FAT or atrial flutter the diagnosis can be challenging if there is a regular AV conduction sequence, especially with 1:1 or 2:1 A:V conduction. In these cases, ectopic P' or F-waves are buried in the QRS or ST-T. Inspecting multiple lead traces for buried deflections (including leads aVR

and aVL where the waves are usually negative for FAT and positive for reciprocating tachycardias) or inducing sudden depression of AV conduction might be required for identification. In humans with supraventricular tachycardia, the finding of an apparent P-wave spaced halfway between two QRS complexes is very suggestive of atrial flutter with 2:1 conduction (the other flutter wave is hidden within the QRS). “Bix’s rule” has been observed in some canine cases.

Atrial Fibrillation Atrial fibrillation represents the best-studied and most important atrial tachyarrhythmia in dogs and horses and affects millions of humans worldwide. It is a straightforward diagnosis in these species characterized by a lack of consistent P waves, baseline fibrillation waves, and an irregularly-irregular ventricular rate response of supraventricular complex morphology (with rare exceptions of patterned AV conduction demonstrated in horses). Based on human studies and research models, the genesis of AF is likely from electrical rotors in the pulmonary veins or abnormal focal automaticity or micro-reentry within the pulmonary venous entries or atrium. However, this supposition requires EP delineation in cases of *spontaneous* AF in veterinary patients. Varying conduction across the atrial mass can lead to chaotic activation (with measured atrial impulses of ~275 to 500 per minute in horses; faster in dogs & cats). Sometimes AF seemingly alternates with atrial flutter (“flutter-fib”), particularly in horses. Fibrillation waves are challenging to identify in cats, but ventricular rate in sinus tachycardia and FATs in this species are usually regular.

The ventricular rate response to any atrial tachyarrhythmia depends largely on sympathetic tone to the AV node. In lone AF, the resting HR is often normal; however, inappropriately fast responses to sympathetic stimulation as with exercise – or even a sudden loud noise – can evoke an inappropriate tachycardia. This is best evaluated by ambulatory (Holter) ECG or an exercising ECG for which the equine literature is most complete. Ventricular rate responses exceeding 250/minute can be observed in horses, rates approaching 300/minute seen in dogs, and rate responses can exceed 300 in cats. In veterinary patients with untreated CHF, the typical standing rates are often 60 to 90 for horses, 140 to 240 for dogs (median about 195), and in cats the mean rate response is about 220/min. QRS morphology is narrow unless there is aberrant ventricular conduction. This can be subtle variation in complexes, common in cats, or marked widening as in horses with rapid response to AF or dogs with right or left bundle branch block. Occasionally an Ashman-like ventricular aberrancy (long-short cycle) is evident.

Conceptually it can be helpful to consider that atrial arrhythmias (PACs, FAT, atrial flutter and AF) appear to be interrelated. For example, horses can exhibit more than one of these rhythm disturbances at one point or another over a relatively short period. With quinidine cardioversion of AF it is common to observe a period of atrial flutter and later what appears to be a sustained atrial tachycardia. After successful conversion, many horses continue to manifest isolated or repetitive PACs.

Differential Diagnosis of Atrial Tachyarrhythmias When presented with a regular, narrow-QRS tachycardia, the ECG differential diagnosis is usually one of the following: (1) sinus tachycardia; 2) focal (ectopic) atrial tachycardia; 3) macro-reentry atrial tachyarrhythmia

(flutter); 4) orthodromic atrioventricular reciprocating tachycardia (using an accessory pathway or longitudinal dissociation of the AV nodal pathways); or 5) automatic junctional (high ventricular) tachycardia. Some of the key characteristics of these are summarized in the previous **Table**.

Sinus tachycardia typically exhibits a subtle irregularity, and might slow transiently with a vagal maneuver. As this rhythm is secondary to beta-receptor stimulation, searching for and managing reasons for adrenergic drive can be instructive. Anxiety, pain, hypotension, hypovolemia, anemia, fever, colic, thyrotoxicosis, toxicosis (methylxanthine, cocaine, adrenergic drugs), and other likely causes should be ruled out. Fast-scan of the heart and estimating cardiac chamber and venous volumes can be useful. In sick patients without overt fluid overload, a crystalloid challenge (10-20 ml/kg of balanced solution, IV) might reduce the sinus rate. If the diagnosis is still in doubt, and FAT with 1:1 AV conduction cannot be ruled out, a beta-blocker can be given. For example, 25 to 50 mcg/kg/minute infusion of esmolol will usually slow a sinoatrial rate.

Once sinus tachycardia has been excluded, blocking of atrial impulses in the AV node is especially helpful in confirming the diagnosis. This can be spontaneous (inspect the baseline during any pauses) or induced with diltiazem, esmolol, sotalol, or digoxin as well as sedatives, vasoconstrictors, or maneuvers that increase blood pressure and augment reflex vagal tone. (Digoxin is not recommended for this purpose, except perhaps in a horse). As previously noted, adenosine is not a standard drug for veterinary patients due to lack of efficacy (dogs) or lack of information.

FAT, atrial flutter and AF are supraventricular tachycardias that do not depend on AV nodal conduction for their maintenance, and the atrial rhythm should be revealed with increased AV nodal block. Conversely, orthodromic AV reciprocating tachycardia using an accessory pathway should be temporarily extinguished by AV nodal block. When a wide-QRS tachycardia is observed with grossly irregular R – R intervals, and without demonstrable P-waves, the potential for antegrade conduction of AF impulses across an accessory pathway (with fully “pre-excited” QRS complexes) should be considered. This is well appreciated in humans but only sporadically reported in veterinary patients. A similar situation might develop if AF is conducted down the AV node but propagated with aberrant ventricular conduction related to drug therapy or preexistent bundle branch block.

MANAGEMENT

General Points Human patients with atrial arrhythmias are often classified based on the presence or absence of valvular heart disease, as this influences the selection of antithrombotic therapy. In veterinary patients, a suggested categorization of atrial tachyarrhythmia might include: (1) *idiopathic or lone* atrial tachyarrhythmia – without clinical or echocardiographic evidence of structural heart disease; (2) Atrial arrhythmia associated with *organic (structural) heart disease*; or (3) Atrial arrhythmia *secondary to a noncardiac disorder* such as a metabolic disturbance or drug. These are arbitrary categories because some dogs, horses (and likely humans) with AF have microscopic or cellular cardiac disease

beyond our detection. In addition to searching for structural heart disease such as mitral regurgitation through clinical examination and echocardiography, predisposing causes for atrial arrhythmias previously mentioned should be excluded. The workup will be guided by signalment, history and physical examination. Echocardiography is generally performed.

In terms of hemodynamic consequences, sustained or relentless SVTs can lead to hypotension and collapse. The onset of FAT or AF in the setting of structural heart disease is especially destabilizing. Relentless tachyarrhythmias can induce arrhythmia-induced cardiomyopathy, creating a “chicken and egg” dilemma regarding which problem occurred first.

Two general approaches are taken in the management of sustained or recurrent FAT, atrial flutter and AF: rhythm control with electro- or drug induced-cardioversion or ventricular rate control. The issue of underlying structural disease is relevant in terms of prognosis and treatment. Despite reports of successful cardioversion of AF in dogs with CHF, the general experience is that most dogs revert to AF in a relatively short time. Similarly it would be rare to see a horse with AF and CHF cardioverted. Accepting differences on the subject, the short-term benefit of AV synchronization is hard to justify with the attendant anesthetic risks and costs of electrocardioversion. If the heart disease is potentially amendable to surgery or a catheter based procedure, cardioversion can be reconsidered. We need controlled, prospective studies in veterinary medicine to identify the best candidates.

Premature Atrial Complexes Although PACs or short runs of atrial tachycardia are unlikely to be a management concern (except in people where they might cause palpitations), understanding the *underlying cause* of the arrhythmia is important. This might be as benign as the sympathetic stimulation of a hospital visit. Hypokalemia from acute diuresis, potassium wasting, bicarbonate drenching, thyrotoxicosis, anemia or another cause of sympathetic stimulation should be excluded. PACs in horses after an exercise test are usually considered normal, especially during acute cool-down, but that might not always be the case. In most species, PACs and nonsustained atrial tachycardias can trigger paroxysmal AF. In horses and dogs converted from AF to NSR, recurrent PACs are often a harbinger of recrudescence to AF. When PACs are associated with structural cardiac disease the usual management approach will involve treating the underlying condition (volume overload, heart failure, pericarditis, etc.). This could reduce the number of atrial ectopics but such patients are at higher risk for development of atrial flutter or fibrillation. Atrial fibrosis is considered important in the development and perpetuation of AF and experimental studies show that blocking of the renin-angiotensin-aldosterone system are effective in reducing the inducibility of AF. Of course, this needs to be tested in clinical trials. Beta-blockers are often used to suppress frequent PACs in human patients, and this approach (atenolol or sotalol) can be adapted to dogs and cats if deemed important. It is quite common to administer sotalol to horses after successful conversion to normal sinus rhythm in an attempt to suppress triggering PACs. (Unfortunately, this therapy does not seem particularly effective, especially in warmbloods).

Sustained Atrial Arrhythmias: FAT, Atrial Flutter & AF – Dogs & Cats

CHF – In the setting of CHF, rate control is recommended. Diltiazem is most effective but can suppress cardiovascular function. Nevertheless, the slowing of heart rate can outweigh the negative inotropy of the drug, and if initial CHF therapy (furosemide, pimobendan, butorphanol, oxygen) does not promptly decrease the rate to <200/minute judicious IV doses of diltiazem (0.05 to 0.1 mg/kg over 5 minutes with BP monitoring) can dramatically slow the rate and improve ventricular filling and reduce oxygen demand. When CHF is present, digoxin (starting at approximately 0.005 mg/kg, PO b.i.d.) can be added for dogs; serum levels should be measured in about 7 to 10 days. The combination of digoxin and oral, long-acting diltiazem (2-4 mg/kg PO bid) better slows heart rate than either drug alone. If optimal rate control cannot be obtained with these two drugs at appropriate dosages, small doses of carvedilol will usually control the rate. Although rate control can be achieved in some dogs with amiodarone, the potential for adverse effects (hepatotoxicity mainly) outweigh the benefit for routine (chronic) use.

Cats with AF are usually in heart failure and careful doses of compounded diltiazem or atenolol (if CHF is controlled) can be used along with heart failure drugs. The author no longer prescribes digoxin to cats due to the challenging kinetics of the drug in this species. These patients also should receive clopidogrel or another agent (LMW heparin; Factor Xa inhibitor) to reduce the risk of arterial thromboembolism.

Higher dosages of digoxin or diltiazem definitely provide better rate control, but at the expense of anorexia or other adverse effects. Maintain the trough level of digoxin (10 to 12 hour post-pill) at 0.7 to 1.2 ng/ml. Be aware the diltiazem is not well tolerated in some dogs and formulations vary across countries (insure the correct Rx).

The optimal heart rate for dogs with AF is unknown. Generally a HR of <140 has more effective cardiac cycles, but many aim for lower rates, especially a lower average rate at home. For a patient with CHF, an average daily rate of 100/minute is not unreasonable (similar to sinus in CHF); in lone AF the target daily rate should be lower, around 75/minute. Exercise rates targets are also uncertain but should ideally be <200/minute in dogs. The in-clinic ECG heart rate overestimates the average home heart rate in dogs with AF. In the author's experience, an in-hospital ECG rate over 60 seconds of 120-150/minute often predicts good control; optimally a Holter ECG should be recorded once the clinic ECG rate seems appropriate. It may take some weeks to achieve optimal rate control. In one study an in-hospital rate of 150 or more predicted poor rate control in dogs.

Lone AF – For idiopathic (lone) AF or atrial tachyarrhythmias unrelated to structural disease, electrocardioversion is considered. If declined or ineffective, atenolol (0.5 to 1.5 mg/kg PO b.i.d.) is a consideration especially if Holter monitoring shows an excessive daily average or exercise heart rate. Diltiazem long acting is an alternative treatment for heart rate control lone AF. Atenolol is potentially cardioprotective in dogs with preclinical DCM.

Rhythm Control/Cardioversion – Treatments that can suppress ectopic rhythms or result in cardioversion to normal sinus rhythm (NSR) include lidocaine (sometimes effective in acute vagally-mediated AF); sotalol (generally safe but of low efficacy), IV amiodarone (Nexterone®) or oral amiodarone, procainamide, and flecainide (but do not use in heart failure or impaired

heart function!). Dofetilide and ranolazine also have been successful in experimental models of canine AF and (equine AF) but are untested in clinical cases. Intravenous diltiazem sometimes converts atrial tachyarrhythmias and infrequently esmolol will do the same. These drugs are most likely to be successful in atrial arrhythmias of recent onset and when the echocardiogram is normal. Despite the potential, most cases of FAT are relatively resistant to drug suppression and bested treated with heart rate control. Again, drugs that block the AV node – diltiazem, beta-blockers, and digoxin – are the mainstays.

Rhythm control in atrial tachyarrhythmias is appropriate for well selected cases. Intravenous drug therapy or synchronized DC cardioversion for atrial flutter/fibrillation are reasonable in dogs if the arrhythmia is known to have begun during a perioperative period or has been witnessed. A structurally normal heart should first be assured via echocardiography, and hypokalemia and hyperthyroidism excluded as predisposing factors. As in horses and many humans, when the onset of AF is acute, spontaneous cardioversion is common within a few hours is common. However, lidocaine (2-8 mg/kg over 10 minutes) can be tried, especially if AF is vagally-mediated. Other options for intravenous cardioversion include 1) procainamide (2 mg/kg IV over 2 minutes; up to 20 mg/kg cumulative dosage with QRS, Q-T, and BP monitoring; currently very expensive); 2) IV amiodarone (use the preservative-free Nexterone® from Baxter Healthcare only! Begin at 4 to 6 mg/kg IV infusion over 60 to 120 minutes with monitoring repeat in 6 to 12 hours if tolerated); 3) or diltiazem (start with 0.1 mg/kg over 5 minutes; repeat up to 0.3 to 0.4 mg/kg IV cumulative dosage; with BP monitoring). Although diltiazem is used mainly for ventricular rate control when the heart rate becomes dangerously high (>250/minute) or the BP too low; it will occasionally convert AF to normal sinus rhythm. Calcium antagonists (verapamil, flunarizine, ryanodine) have been shown to terminate atrial tachycardia and suppress inducibility in canine models of atrial arrhythmias.

Biphasic, DC cardioversion is very effective in treating lone AF of recent onset. If the cardioversion is planned, oral amiodarone (loading dose of 4-6 mg/kg PO b.i.d.) can be given for one week prior to conversion; alternatively intravenous Nexterone® can be administered at 4-6 mg/kg IV 2 hours prior to electrocardioversion. If cardioversion is successful in a dog, the author prescribes generic amiodarone (4-6 mg/kg PO twice daily for 2 weeks and then once daily for 3 months before switching to sotalol (1 to 2 mg/kg PO twice daily for one year); this is to suppress atrial ectopy and maintain sinus rhythm.

Sustained Atrial Arrhythmias: FAT, Atrial Flutter & AF – Horses

The three *general approaches for the management of atrial fibrillation*. The first is to *do nothing and simply evaluate the impact* of the rhythm on the horse. This is probably most relevant to retired animals or those used for light work. Economics sometimes force this issue or in some cases horses have been treated for rhythm control but failed. Those horses with lone AF who are past their peak-exercise years often do well with watchful waiting and curtailing of excessive exercise. Many of these horses can be ridden safely, although an Echo and exercise ECG are recommended if permanent AF is accepted instead of sinus rhythm. Even in horses with AF and normal resting rates, there is a likelihood that an excessive

ventricular rate response will occur once under sympathetic provocation. For example, the rate response to exercise is usually excessive for the work performed, and the irregular cardiac cycles often lead to relatively short intervals between complexes. Such rhythms hemodynamically inefficient and potentially dangerous electrically, sometimes creating a form of “R on T” in the cardiac rhythm. These can be challenging electrocardiograms to interpret because distinguishing an aberrantly conducted atrial impulse from a ventricular ectopic at high heart rates is challenging.

The second approach involves *heart rhythm control*. This includes cardioversion of atrial flutter or AF using either drugs or electrocardioversion. Quinidine, procainamide, amiodarone, and flecainide each have the potential to suppress atrial ectopy or convert atrial flutter or fibrillation. There is substantial experience with quinidine in horses but progressively less experience in the hospital therapy of AF using the other listed drugs. Some specific approaches are discussed below.

The third approach is *heart rate control* which involves depressing conduction of atrial impulses across the AV node. In general, horses with structurally normal hearts tend to control the ventricular rate response themselves, especially at rest. Most cases of lone AF maintain a normal resting heart rate. As mentioned above, finding a persistent resting tachycardia in a horse with presumed “lone AF” should prompt a more thorough investigation that includes echocardiographic evaluation of cardiac size and ventricular function and a search for hidden reasons for sympathetic activation. Heart rate control is most often considered for horses with CHF (see below) and involved administration of drugs that block AV nodal transmission to control the ventricular rate response. This can be accomplished with digoxin (IV or PO), diltiazem (IV), or a beta blocker such as the class III antiarrhythmic sotalol. The major caveat regarding heart rate control is the potential for depression of ventricular function, and for horses with CHF, only digoxin is a positive inotrope within this group of rate-slowing drugs.

Congestive Heart Failure – Patients with incipient or overt congestive heart failure (CHF) should be identified due to their risk of use and potential for adverse effects if cardioversion is attempted. Although the diagnosis of CHF is usually straightforward, horses with incipient failure might only exhibit a more rapid resting heart rate with no other excuse for sympathomimetic activation. Some foaling mares with lone AF are stable until late in gestation, at which time the demands for higher cardiac output can precipitate congestive heart failure (CHF) and the need for medical therapy (summarized earlier).

These patients are likely to have adverse reactions to the negative inotropic effects of drugs like quinidine or class IC agents like flecainide (as well carrying a heightened risk for general anesthesia needed for electrocardioversion). Horses with overt CHF are treated medically and rarely undergo cardioversion in veterinary medicine. The treatment plan should be aimed at controlling fluid retention with furosemide, blunting neurohormones with an ACE-inhibitor (benazepril), and supporting contractility and controlling resting heart rate with digoxin (and potentially increasing inotropy with pimobendan if it becomes practical economically and better studied).

Digoxin can be initiated using IV injections for the first one to three days and maintained with oral therapy at the farm provided the owner is willing to treat. The ACE-inhibitors are potentially teratogenic, but are probably safe for a late-term fetus when treating pregnant mares. Clinical monitoring of the horse with AF and CHF should include periodic measurements of serum digoxin concentration, renal function, and serum electrolytes as well as inspecting the horse for tachypnea, jugular venous distension or the accumulation of ventral edema. Exercise should be limited to pasture walks and the horse should never be ridden or driven.

Cardioversion – Although acute, witnessed AF is usually observed for at least 24 hours before any treatment (in hope of spontaneous conversion to NSR), the rhythm persists in many horses and will sustain unless treatment is given. Rhythm control of lone AF can be achieved in the vast majority of cases. Given motivated clients, the precise success of cardioversion of lone AF depends largely on the patient population and the persistence of the clinician. Cardioversion of Standardbred racehorses promptly diagnosed at the track is likely to exceed 95% with either quinidine or electrocardioversion. Conversely, treatment of a warmblood with chronic AF of >6 months duration can be more challenging and require prolonged therapy, additional drugs (e.g. adding in digoxin) or if failing drugs require electrocardioversion attempts. In our hospital, where quinidine is the agent of choice, the success rate is approximately 85% for all cases; this is similar to other reports. Our success of electrocardioversion is slightly higher but we there has been the rare failure and more importantly a number of successful electrocardioversion horses have reverted back to AF.

Cardioversion is typically performed in one of three ways. The two most accepted approaches are administration of **quinidine sulfate** (nasogastric tube) for drug-induced cardioversion to sinus rhythm. The second is **transvenous, synchronized, biphasic electrocardioversion (TVEC)** done under echocardiographic and radiographic guidance via two specialized electrode catheters. This is a referral procedure and is well described in the references (See McGurrin papers and the Van Loon chapter in the VCNA-Equine Practice). TVEC requires standard general anesthesia or a constant infusion of an alpha2 agonist like dexmedetomidine. Generally quinidine and TVEC are considered equally appropriate first-line treatments and will be selected based on experience, facilities, availability of quinidine, client preferences, and responses to previously treatment with the alternative method.

The third approach to converting AF is the administration of an alternative antiarrhythmic drug with the potential for conversion of AF to normal sinus rhythm. These include the aforementioned procainamide, flecainide, amiodarone, or propafenone. Newer agents are also potentially useful, such as dofetilide and ranolazine. Small clinical studies and personal experience indicate variable results with these drugs when compared to quinidine or synchronized transvenous electrocardioversion. In the largest flecainide series, involving Thoroughbred horses in Japan, quinidine was far superior to flecainide for primary cardioversion of AF with flecainide, which as still successful in 41% of cases used. Although often used in humans with lone AF, flecainide has been proximally associated with sudden death from proarrhythmia in humans, dogs, and horses, especially in the settings of impaired ventricular function or heart failure.

Quinidine therapy – The most common treatment of lone AF in North America is with administration of quinidine sulfate by nasogastric tube using a variety of dosing intervals. In our hospital quinidine sulfate is administered every two to six hours until the horse converts to NSR or develops signs of toxicosis. This is most likely to occur at a cumulative dosage of 88 to 132 mg/kg. In practice, most horses convert after two or three doses and we rarely extend the first-day treatment past four doses q2h before starting a q6h treatment regimen either later that day or on the second day of treatment. If cardioversion has not occurred after two (or three) days of treatment, quinidine is usually discontinued or the treatment suspended. While it is ideal to draw a plasma sample for determination of quinidine plasma concentrations at the point of “failure”, virtually no laboratories run this analysis anymore or return results in a timely manner. Therapeutic plasma quinidine concentrations for conversion from AF to sinus rhythm are 2 to 5 micrograms per mL and readily achieved with these regimens. A number of horses will convert to NSR *after* treatment is suspended and high plasma levels decline.

Most horses demonstrate some signs of toxicosis, but mild colic or soft feces are not necessarily reasons to stop treatment. Walking the horse can sometimes help the symptoms and the patient should be constantly monitored for more serious signs of toxicosis. If there is an increase in heart rate to >100/minute, marked widening of the QRS complex, signs of abdominal distress, fluid-like diarrhea, symptomatic nasal edema, or clinical signs of hypotension therapy should be aborted and an ECG and vital signs carefully monitored. When the ventricular rate response increases gradually with administration of quinidine, this vagolytic effect can be blunted with a dose of IV digoxin (typically one mg) daily. We consider this when the resting ventricular rate response approaches 80 to 90/minute or the horse seems uncomfortable with an increasing rate response. As in other species, there is a digoxin-quinidine interaction that can effectively double the serum concentration of digoxin with long-term use of both drugs. Interestingly, in one retrospective study, the success of quinidine cardioversion was higher in horses also receiving digoxin (see references).

As indicated above, despite the potential for serious adverse effects with quinidine, most referral practices experience an excellent treatment response with >85% conversion rates. The more persistent clinician will likely achieve slightly greater success depending on the patient population treated. Horses with recurrent or long-standing (persistent or permanent) AF or those with structural heart disease (without CHF) can be more challenging to convert.

Details of quinidine therapy, monitoring, and toxicosis are available in standard textbooks (see Marr & Bowen or the recent VCNA-Equine Practice for good reviews). In terms of proarrhythmia, the most common ECG change after quinidine administration is an acceleration of AV nodal conduction related to the vagolytic effect of quinidine. Rapid supraventricular tachycardias with ventricular rate responses of over 250/minute have been observed, begging the question of whether the rhythm has changed to atrial flutter with 1:1 conduction or constitutes a rapid ventricular tachycardia. True polymorphic ventricular tachycardias of the torsade de pointe variety also occur with severe toxicity or as in people, as an idiosyncratic reaction. In these cases, quinidine should be stopped, intravenous fluids initiated to improve perfusion, and IV sodium bicarbonate administered to enhance quinidine

binding and elimination. A phenylephrine drip can restore blood pressure if there is critical hypotension. It can be difficult to decide on the course of treatment of the critically fast tachyarrhythmia because distinguishing atrial flutter/fibrillation with rapid AV conduction from fast ventricular tachycardia can be difficult. Regularity of rhythm is not always a helpful feature due to the rapid rate and the fact that some equine VTs are somewhat irregular, especially if multiform. Digoxin, diltiazem or esmolol could potentially be useful by blocking AV nodal conduction of flutter or fibrillation waves. However, an intravenous magnesium sulfate infusion is often a better choice if the rhythm is thought to be ventricular in origin. Lidocaine HCl also can be administered. If there ventricular rate response is very irregular and wide, the possibility of conduction of AF down an accessory pathway with a short refractory period should be entertained and drugs that block the AV node avoided.

Follow up and Outcomes – Reversion to AF occurs in at least 25% of horses and in one study was closer to 35% of cases in a year. Although some time off is desirable after conversion to NSR, the imperative for racing horses to return to training essentially starting work within 48 hours after conversion. This is inadvisable as it can take a month or longer for the atria to regain more normal mechanical and electrical properties. Mechanical atrial function can be quantified a number of ways using echocardiography, but this is usually confined to referral practices. Overall, most horses return to their previous performance level if sinus rhythm can be sustained. With the advent of economical phone-based ECGs, it is reasonable for the trainer or farm manager to record and transmit follow up traces to the veterinarian for her or his review. Horses with repeated episodes of AF can be converted multiple times with quinidine sulfate or TVEC. Recurrence of AF can be immediate or develop years later and is often portended by the persistence of PACs or short runs of FAT.

The finding of persistent PACs after cardioversion is a concern for precipitating paroxysmal AF. Although drugs such as quinidine or procainamide are effective for converting atrial tachyarrhythmias, these are not practical long-term therapies. Sotalol appears to be relatively well-tolerated in horses without ventricular dysfunction and the drug can be administered long-term. However the success of sotalol for preventing AF has not been impressive in the author's experience. Ideal post-cardioversion monitoring would also involve an ambulatory (Holter) ECG and exercise ECG, but these are infrequently obtained beyond the hospital telemetry unit.

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Suspected intra-hisian (infranodal atrioventricular) block in an 8 year old jumping horse

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An 8 year old jumping horse stallion was presented because it had showed severe epistaxis after mild exercise. The examination at home showed a heart rate of 30/min with frequent 2nd degree atrioventricular (AV) block and a 2/6 holosystolic murmur over the tricuspid valve.

Clinical exam revealed a normal body condition. Mucous membranes and capillary refill time were normal; there was no oedema. Heart rate was 28/min with a regularly irregularity. The right-sided systolic murmur was confirmed. Echocardiography revealed trivial tricuspid and aortic valve regurgitation without clinical importance. Size of the cardiac chambers and vessels was normal. No structural abnormalities in the region of the atrioventricular conduction system and his bundle could be identified.

ECG at rest initially showed a heart rate of 28/min with predominant 2/1 atrioventricular conduction during the exam (2 P waves for 1 QRS complex). Morphology and duration of P, QRS and T waves and the PQ interval were within normal limits except for frequently occurring 2nd degree AV block. When the horse calmed down, ventricular rate went up to 44/min due to a 1/1 AV conduction. Due to stress or exercise, more AV block occurred due to decreased AV conduction that resulted in 2/1, 3/1, 4/1 atrial to ventricular conduction. Maximal heart rate during exercise was 92/min.

Serology indicated high and increasing antibody levels for borreliosis. Right ventricular biopsies were taken but PCR was negative for *Borrelia*.

In the standing sedated horse, intracardiac electrogram recording was performed under ultrasound guidance, using a decapolar diagnostic catheter. The AH interval was 208 ms and the HV interval was 80 ms. Due to sedation, however, the slow sinus node rhythm was no longer associated with AV block whereby the exact point of block could not be confirmed.

Due to the fact that increased sympathetic tone impaired AV conduction and alfa-2 agonists improved AV conduction, a presumptive diagnosis of infranodal or intra-hisian block was made.

The horse was treated with anti-inflammatory drugs, antibiotics, vitamin E and vitamin B1. Although atrioventricular conduction improved with more episodes of 1/1 AV conduction, it did not return to normal. Repeated follow-up exams did not show further improvement and therefore a grave prognosis for a sport career was given. The owner therefore elected castration of the stallion. The procedure was performed under general anaesthesia with temporary right ventricular pacing. Recovery was uneventful. The horse was subsequently retired.

Segmental septal dysplasia – is it (pre-) exciting?

Omri Belachsen

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A 4-year-old neutered female Golden Retriever presented to the cardiology service at Southern Counties Veterinary Specialists for investigation of asymptomatic heart murmur and arrhythmia. Twelve-lead surface electrocardiogram revealed a wide QRS rhythm with a fixed, short P-R interval and slurred upstroke, consistent with delta waves. Transthoracic echocardiography showed segmental thinning of the basal interventricular septal wall, measuring 3 mm in thickness at end-diastole. The abnormal segment demonstrated dyskinetic motion with paradoxical wall motion during diastole. Based on the electrocardiographic findings, manifest ventricular pre-excitation secondary to an accessory pathway was suspected. The patient was referred to Davies Veterinary Specialists for an electrophysiology study (EP), where two distinct right-sided accessory pathways (APs) were confirmed, one in right caudal position and another in mid-septal position. Both pathways were capable of fast anterograde and retrograde conduction.

A 3.5-year-old neutered female Golden Retriever presented to the small animal hospital at the Royal (Dick) School of Veterinary Studies in Edinburgh with paroxysmal tachyarrhythmia, short P-R interval and slurred upstroke of the QRS complexes. Echocardiographic examination revealed segmental septal dyskinesia and wall thinning with striking resemblance to the first case. EP study was not performed in this dog to confirm the presence of an accessory pathway, although the electrocardiographic findings were consistent with this diagnosis.

Accessory pathway with manifest pre-excitation is a rare condition in dogs. Fast antegrade conduction through the accessory pathway causes premature electrical and mechanical activation of myocardial segments close to the ventricular insertion of the accessory pathway. Large pre-excited areas may lead to significant dyssynchrony in the ventricular motion and thereby cause ventricular dysfunction. In addition, early activation of the pre-excited segment causes it to contract under low local pre-load conditions, which can lead to decreased myocardial work and hypotrophy. The hypotrophy is likely the reason for the extreme systolic bulging of the pre-excited segment towards the right ventricle as seen in both dogs in this case report.

Echocardiographic findings similar to those described in this case report, have been well documented in people with manifest Wolf-Parkinson-White syndrome. However, to the best of the authors knowledge, this is the first report of such findings in dogs with manifest pre-excitation due to an accessory pathway.

3D electro-anatomical mapping and ablation in horses

Gunter van Loon

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Electrical mapping of the heart indicates a technique to identify the electrical activation pattern of the myocardium. In case of a tachyarrhythmia this technique aims to identify the focus of arrhythmia or the pathway of a macro-reentry. The ultimate goal is to ablate (destroy) the myocardium at the level of the focus or at a narrow isthmus of the re-entry path. Ablation is performed by heating (radiofrequency ablation) or cooling (cryo-ablation) of the myocardial tissue. These techniques require very precise mapping and ablation in order to be successful and in order to avoid ablation of important structures such as atrioventricular node or his bundle.

Electrical mapping is performed by placing multiple catheters, each with multiple electrodes, at very specific locations in the heart. A frequently used technique in human and small animal patients is the 'four wire' technique with catheters in the coronary sinus (CS), at the high right atrium, his bundle and right ventricular apex. The coronary sinus catheter or the QRS from the surface ECG can be used as timing reference for atrium and ventricle, respectively. For every recorded electrogram, the timing can be compared to this reference so that a timing sequence can be built up from different positions in the heart. Positioning of the catheters is crucial and fully performed by fluoroscopy. CT or MRI before the mapping procedure help to identify the 3D anatomy of the mapped chamber.

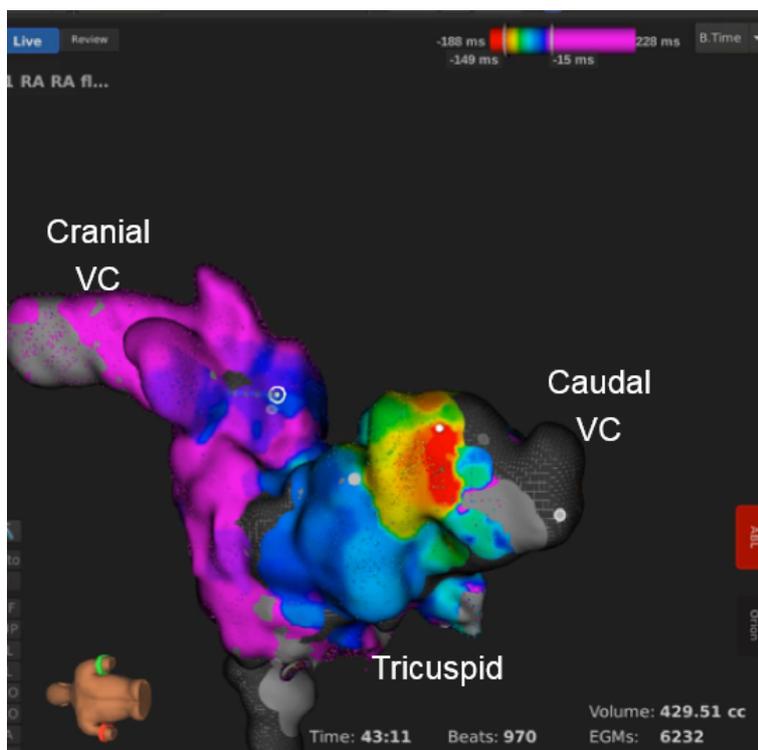
In adult horses, fluoroscopy and even radiography does not provide sufficient detail to allow precise positioning of catheters and the gantry of an MRI or CT is too small to fit the thorax of a horse. Ultrasound is helpful but so far limited knowledge is available to guide mapping procedures. As such, there are no adequate imaging modalities to perform mapping in adult horses.

Recently, we successfully applied the Rhythmia[®] system (Boston Scientific) in adult horses in sinus rhythm for mapping of left and right atria and ventricles. This high density 3D electro-anatomical mapping system uses a magnetic field in combination with impedance tracking to locate the mapping catheter (Intellamap Orion[®]) in 3D in the equine heart. The mapping catheter is an 8.5F bidirectional deflectable catheter with a deployable mini-basket (diameter ranging 3–22mm) consisting of 8 splines. Each spline contains 8 printed, small (0.4mm²) low impedance electrodes (64 electrodes in total) with an interelectrode spacing of 2.5mm (centre-to-centre). During the procedure, at any time, the Rhythmia station records all intracardiac electrograms in combination with the precise location of the catheter in 3D space. The catheter is manoeuvred along the endocardial wall of the entire cardiac chamber. The Rhythmia station records the 'outer shell' of all catheter

positions which results in a very detailed 3D copy of the inside of the heart. At the same time the local electrogram of each point is available. By comparing the timing of the local depolarisation with a fixed reference (CS electrogram or QRS), the activation sequence of the entire cardiac chamber can be shown on the 3D heart providing a realistic, colour-coded 3D activation map. In case of a tachyarrhythmia, this map shows the rapidly firing focus (in case of focal tachycardia) or the re-entry path (in case of macro-reentry) which is the target of ablation.

In three horses with atrial tachycardia originating from the right atrium, the technique showed the precise mechanism of tachycardia that allowed to perform successful radiofrequency ablation.

First a (custom developed) 12-lead surface ECG recording was used to identify the most likely location of tachycardia based upon vector-electrocardiography. The origin was located in the caudal right atrium. Subsequently, under general anaesthesia, the magnetic field generator was fixed over the heart. A 12-lead surface ECG and a decapolar catheter positioned in the CS were connected to the Rhythmia® system. With ultrasonographic aid, the right atrium was mapped with the Intellimap Orion® catheter. The activation map confirmed the origin of tachycardia in the caudal right atrium in all 3 horses.



High density 3D electro-anatomical map (view from the left) of a horse with focal atrial tachycardia originating from the caudo-medial right atrium. The colour bar on top shows the timing colour codes. The red area is the earliest activation and therefore the focus of the tachycardia. The other colour codes show how the electrical depolarisation spreads over the right atrium.

After mapping of the tachyarrhythmia, an Intellanav OI® (open irrigated) catheter was introduced to perform radiofrequency ablation, which terminated the atrial tachycardia in all 3 horses. The advantage of this catheter is that I can also be tracked magnetically so that

it is continuously visible on the 3D electro-anatomical map. As such it allows precise manoeuvring to the target tissue.

These first cases of successful mapping and radiofrequency ablation of tachyarrhythmias in horses are very promising for a new, targeted approach of atrial tachyarrhythmias in adult horses. The 3D mapping system proved to be applicable to the large equine heart and allows the whole technique to be performed without any fluoroscopy. Future experience needs to be built up and must also point out the applicability of the technique for tachyarrhythmias coming from other locations within the heart.

An Update on Heart Testing

Hannah Stephenson

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The Veterinary Cardiovascular Society is responsible for coordinating and regulating veterinary heart testing prior to breeding in dogs and cats. The VCS is trying to move towards a more standardised approach to heart testing and we also want to encourage interested members to consider applying to be on the auscultation and Doppler teams. We have recently launched a new heart testing scheme for the Cavalier King Charles Spaniel in association with the Kennel Club, and there is certainly appetite to develop more schemes. This short presentation will provide an overview of the VCS role in heart testing and the roles of the breed-related sub-committee.

ECVIM 2019 Update

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Left atrial tear in dogs with myxomatous mitral valve disease – clinical presentation, echocardiographic features and long-term survival.

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This case-control matched multicentre retrospective study aimed to firstly describe the clinical presentation and short- and long-term treatment of left atrial tear (LAT) in dogs, and secondly to compare the survival of LAT patients with an echo-matched control group of dogs with myxomatous mitral valve disease (MMVD) without LAT. Data from 15 dogs with advanced MMVD-associated LAT, including signalment, clinical presentation, echocardiography, selected laboratory findings, the cause of death and survival data were collected and assessed at the time of LAT diagnosis (T0), and at the resolution of pericardial effusion (T1). Data were compared to a control group consisting of 15 dogs with similarly advanced MMVD without LAT. Commonly observed clinical signs included dyspnoea, syncope and weakness. No significant differences in age, body weight, gender distribution serum creatinine concentration or echocardiographic variables were identified between the groups. The mean survival time for LAT dogs was 53 days, however when animals that died within the first week (5/15 dogs with LAT; 1/15 control group) were excluded, the survival times increased to 427 days for dogs with LAT and 371 days for the control group. In summary, the authors stated that dogs with MMVD associated LAT bear a higher cardiac mortality risk, especially in the first week post the event, however long-term survival does not seem to be significantly impacted by the presence of LAT past this point.

Point of care ultrasound of the caudal vena cava in canine degenerative mitral valve disease

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This retrospective study aimed to assess if caudal vena cava (CVC) parameters, evaluated via point of care ultrasound (POCUS), were correlated with different ACVIM stages of degenerative mitral valve disease (MVD) in dogs. The CVC diameter (CVCD) and collapsibility index (CVCCI) are widely used in human medicine to assess intravascular volume status, with CVC POCUS examinations helping to identify heart failure patients that are at risk of decompensation. In this study the CVC was observed via a longitudinal

subxiphoid view in eighty-one dogs diagnosed with MVD in ACVIM stages B1, B2, C or D. The CVC maximal and minimal diameter were measured and indexed on aortic diameter, and the CVCCI was calculated. The CVC was also subjectively assessed as flat, normal or fat by the same observer, who was unaware of MVD status. The authors reported that CVC parameters were associated with ACVIM stage. Particularly a significantly larger CVCD/Ao and reduced CVCCI in dogs with ACVIM stage C or D compared with ACVIM stage B1 or B2 (p value <0.01), and a significant association between a subjectively fat CVC and ACVIM stage C or D (p value <0.0001). The findings indicate that CVC POCUS parameters could be useful in identifying dogs at increased risk of decompensation or that require hospitalization,

Changes in Renal Endothelin Activity with Cardiac, Renal and Other Chronic Diseases in Dogs

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Overall, cardiovascular risk factors in dogs are unknown, with mechanisms that disrupt the cardiovascular-renal axis being incompletely defined. This study aimed to investigate the effect that heart disease may have on the urinary excretion of renal endothelin-1 (ET-1), a ubiquitous peptide that has increased circulatory levels in cardiac disease in people. Urinary ET-1 (UET-1) is a marker of renal vascular and tubular ET-1 activity. The urinary concentrations of UET-1 and cystatin C (a marker of renal injury/dysfunction) were measured and compared between six groups of dogs; 1. healthy control, 2. non-cardiac non-renal-disease, 3. chronic kidney disease (CKD), and lastly cardiac disease at ACVIM stages B1, B2 and C representing groups 4 – 6 respectively. Results revealed that UET-1 excretion markedly increased in dogs with stage C heart disease compared with healthy dogs (p value =0.02). Furthermore, excretion increased with increasing severity of canine heart disease, particularly in MMVD. Increases in UET-1 were not associated with renal injury or a chronic disease state. In contrast, urinary cystatin C was markedly increased in CKD groups (p value <0.0001), mildly increased in dogs with chronic disease (p value =0.03), and not increased in heart disease stages B1-C. Therefore, the authors state that it is possible that renal ET-1 may mediate pathophysiological cardiovascular-renal interactions in MMVD at the time of cardiac remodelling, but these interactions are different to those in CKD and non-MMVD cardiac disease.

Retrospective Evaluation of the Safety and Tolerability of Pimobendan in Cats with Obstructive versus Nonobstructive Hypertrophic Cardiomyopathy

Dr. Jessica Ward of Iowa State University

Pimobendan is frequently used off-label for treatment of cats with congestive heart failure (CHF) secondary to hypertrophy cardiomyopathy (HCM). Concerns exist regarding the safety of pimobendan in the subset of cats with HCM and dynamic outflow tract obstruction

(HOCM). The purpose of this study was to evaluate safety and tolerability of pimobendan in cats with CHF secondary to HOCM compared with nonobstructive HCM. Medical records from 94 cats with CHF (47 with HOCM, 47 with nonobstructive HCM) at two tertiary referral hospitals were reviewed. Demographic, clinicopathologic, echocardiographic, and treatment data were collected and compared between groups, including information regarding possible adverse effects of pimobendan. Average age of cats (9 ± 4 years) did not differ between HOCM and HCM ($p=0.12$). Compared to cats with HCM, cats with HOCM were more likely to manifest CHF as pulmonary edema (44/47 versus 32/47; $p=0.003$) and less likely to have pleural effusion (13/47 versus 25/47; $p=0.02$). Other than a higher incidence of heart murmurs in cats with HOCM ($p<0.001$), clinical variables did not differ between groups.

Pimobendan was typically initiated on the date of CHF diagnosis (median time from diagnosis of CHF to initiation of pimobendan was 0 days). Initial dose of pimobendan was 0.25 ± 0.07 mg/kg every 12 hours; dose (or frequency) was escalated at some point during CHF management in 31/94 (33%) of cases, with no difference between HOCM and HCM cats.

Clinical signs that could potentially represent adverse effects of pimobendan (vomiting, diarrhea, anorexia, lethargy, new-onset arrhythmias) were noted in 13/47 (28%) HCM cats and 9/47 (19%) HOCM cats ($p=0.34$). Based on patterns of timing and resolution, these signs were generally ascribed to recurrence of CHF rather than pimobendan administration. Pimobendan was discontinued due to adverse effects in only 1 cat with nonobstructive HCM that experienced lethargy and nausea 2–3 hours following pimobendan administration (resolved when pimobendan discontinued). Pimobendan was discontinued in 7 additional cats, either because owners were unable to administer the medication ($n=2$) or because CHF had resolved ($n=5$ cases where CHF was precipitated by an acute external event, such as fluid overload or injectable glucocorticoid administration).

No cats experienced acute adverse hemodynamic effects (hypotension, cardiovascular collapse) following pimobendan administration.

Results of this study suggest that pimobendan is well tolerated in cats with cardiomyopathy and CHF, regardless of presence of dynamic outflow tract obstruction.

Delayed Electrolyte Depletion and Azotemia in a Furosemide Rate Continuous Infusion Model

Dr. Darcy Adin of University of Florida

Intravenous furosemide is the mainstay of treatment for acute congestive heart failure in dogs, however, the potential for delayed effects on hydration, electrolytes and renal function have not been studied. This study sought to evaluate these parameters in normal dogs receiving furosemide continuous rate infusion (CRI) with or without renin-angiotensin-aldosterone system inhibitors.

Ten healthy dogs were studied in a 3-way randomized, cross-over design. Dogs orally received either placebo, benazepril, or benazepril+spironolactone for 3 days prior to 5-hour furosemide CRI 0.66 mg/kg/hr. Body weight (BW), renal values, serum electrolytes, packed cell volume and total protein were measured before oral medications, hour 0 and 5 of the furosemide CRI, and hour 24. Variables were compared between time-points and treatments.

Loss of BW during the CRI exceeded recovery at 24 hours and hemoconcentration occurred, with incomplete return to baseline at 24 hours. Blood urea nitrogen and creatinine were unchanged during the CRI but increased $24 \pm 12\%$ at 24 hours. Serum sodium did not change during the CRI but decreased at 24 hours. Serum chloride decreased at hour 5 and did not return to baseline at 24 hours. Hypochloremic metabolic alkalosis and increased anion gap present at hour 5 did not normalize at 24 hours. No differences between treatments were found.

Some furosemide CRI-related biochemical changes were delayed for 24 hours while others evident at hour 5 only partially improved at hour 24 in these normal dogs. These findings have implications for clinical patients with renal dysfunction, or receiving higher doses or longer furosemide infusions.

High-Grade AV Block and Third-Degree AV Block in Cats: A Retrospective Study of Epicardial Pacemaker Implantation (2006–2018) Focusing on Signalment, Presentation and Survival

Dr. Ilaria Spalla of Royal Veterinary College

Third-degree atrioventricular block (AVB) is characterised by complete atrioventricular (AV) dissociation, causing independent atrial and ventricular rhythms. Persistent third-degree AVB (PAVB) is most commonly described. Another form of AVB, where AV dissociation is intermittent (IAVB) is also recognised. In cats, AVB can be associated with underlying cardiac or systemic diseases. When present, clinical signs associated with these forms of AVB can include weakness, lethargy and syncope. If clinical signs and in particular syncope are present, epicardial pacemaker implantation represents an effective treatment.

The aim of the study was to retrospectively assess presentation, echocardiographic data, comorbidities and outcome from cats diagnosed with AVB (PAVB or IAVB) in a single referral hospital, including those that underwent pacemaker implantation. Non-parametric testing and Kaplan-Meier curves with log-rank testing were performed. Sixty-four cats were included over a 12-year period. Forty-three cats had PAVB; 21 had IAVB. Median age of presentation was 13 years, with no difference between AVB type ($p=0.752$). Thirty-five cats were male, and 29 female. Forty-four cats were referred for cardiac complaints (syncope, arrhythmia or dyspnoea), 8 cats had non-specific signs (lethargy) and in 12 cats AVB was an incidental finding. Cats with IAVB were more likely to present with syncopal events ($p=0.005$). The median duration of clinical signs prior to presentation was 21 days (1–1138). Twenty-nine cats had echocardiographic changes, left ventricular hypertrophy (17),

chamber dilation (12); 13 cats presented with congestive heart failure (CHF). Forty-five cats had one or more comorbidities, the most common were hyperthyroidism (16), diabetes mellitus (9), azotaemia (8). Fifteen cats underwent epicardial pacemaker implantation, mainly cats with IAVB (9/15). Five cats had minor complications (lead dislodgement, pacemaker undersensing, exit block), and 12 cats showed no further clinical signs since implantation. Forty-seven cats died; all-cause mortality median survival time was 799 days (0–2965) and no difference in survival was observed in cats that presented with CHF ($p=0.052$), IAVB ($p=0.082$), had comorbidities ($p=0.683$) or pacemaker placement ($p=0.089$). Death due to cardiac cause occurred in fewer cats (17/47), with shorter survival than all-cause mortality (65 days, $p=0.003$). CHF on presentation was associated with cardiac death ($p<0.001$). The results of this study showed a variable outcome in cats with AVB. Cardiac death occurred in the minority of cats and was associated with CHF at presentation. Most cats have comorbidities, which did not affect all-cause mortality. Pacemaker implant controlled clinical signs in the majority of cats.

Effect of spironolactone and benazepril on time of onset of clinical signs in dogs with advanced preclinical degenerative mitral valve disease: The DELAY study

Luca Ferasin

Lumbry Park Veterinary Specialists, Alton, UK

DElay of appearance of sYmptoms of canine degenerative mitral valve disease treated with spironolactone and benazepril: The DELAY study

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The efficacy of angiotensin converting enzyme inhibitors (ACEi) in dogs with preclinical myxomatous mitral valve disease (MMVD) is controversial. However, there are no studies available to test the hypothesis that combined administration of spironolactone and benazepril in dogs with preclinical MMVD, not receiving any other cardiac medications, can delay the onset of heart failure (HF) and cardiac-related death, as well as reducing disease progression based on echocardiographic parameters and level of cardiac biomarkers.

To test this hypothesis, 184 dogs with pre-clinical MMVD and echocardiographic signs of cardiac remodelling (LA:Ao \geq 1.6 and LVEDDn \geq 1.7) were enrolled in the present prospective, randomized, multicentre, single-blinded, placebo-controlled study. Primary outcome variable was time to onset of first occurrence of HF or cardiac death. Secondary endpoints included effect of treatment on progression of the disease based on echocardiographic and radiographic parameters, as well as variations of concentration of cardiac biomarkers.

The median time to primary endpoint was 902 days (95% CI 682-NA) for the treatment group and 1139 days (95% CI 732-NA) for the control group (p=0.45). Vertebral heart score (p=0.05), LA:Ao (p<0.001), LVEDDn (p<0.001), trans-mitral E peak velocity (p=0.011), and NT-proBNP (p=0.037) were lower at the end of study in the treatment group.

Based on the above results, this study failed to demonstrate that combined administration of spironolactone and benazepril can delay the onset of HF in dogs with preclinical MMVD. Nevertheless, this treatment appeared to reduce the progression of cardiac remodelling in this population, indicating a potential clinical benefit of this medical intervention.