Proceedings of the November Meeting of the

Veterinary Cardiovascular Society

Friday 7th and Saturday 8th of November

Officers of the society

Chair	Proceedings Editor / Web Manager
Gavin McAulay North Downs Specialist Referrals, Surrey Brighton Veterinary Cardiology, Brighton chairperson@vet-cardio.co.uk	**Mattia Basili** ChesterGates Veterinary Specialists, Chester proceedings@vet-cardio.co.uk
Joint Secretaries	**Treasurers**
Julie Hamilton Elliott Northern Ireland Veterinary Specialists Co. Down secretary@vet-cardio.co.uk **Siddharth Sudunagunta** Willows Veterinary Centre Solihull	**Claire Denny** Manor Vets Halesowen **Vicky Ironside** Park Vet Hospital Sheffield treasurer@vet-cardio.co.uk

As in previous editions, we'll be using Mentimeter to collect questions following each talk. Please scan the QR code or click the link below to access the webpage and submit your question.

QR Code:

Website link: https://www.menti.com/al33p212trtz

Welcome to the 50th Anniversary meeting of the Veterinary Cardiovascular Society.

50 years ago the world was a simpler place without internet, mobile phones, or cashless payment. There were almost no echocardiography machines and furosemide had only been available for ten years. Yet Peter Darke and colleagues had the foresight to found the Veterinary Cardiovascular Society.

Whilst the world has changed the society remains more important than ever. As a speciality cardiology is flourishing and benefitting from the technological and scientific advancements of the last 50 years. Yet fundamentally cardiology remains a hands-on clinical skill requiring excellent human communication.

The VCS is an organisation that exists to promote animal welfare through the study and development of veterinary cardiology in all species. We provide an opportunity anyone with an interest in cardiology to meet socially; to communicate ideas; mentor; network and collaborate. Members can freely express their views in an open environment that encourages debate and appreciates the value of both experience and scientific endeavour. Against the back drop of unimaginable technological advancements, environmental challenges and shifts in social norms an intellectually stimulating meeting with friends remains valuable and relevant.

As in previous years, considerable work been required to develop the behind-the-scenes management of VCS. Specifically we can now start to track members – and membership growing! The web site now offers increasing interaction with access to recordings of previous meetings and exclusive material available from BSAVA.

We are very pleased to host a full day stream of cardiology for nurses at this Autumn meeting. This reflects the reality that there is a growing cardiology workload and an ever-greater need for a team approach. The changes in our constitution now also allow all members, including nurse members, to vote at our AGM.

We have continued to support member training with a variety of grants and bursaries and are keen to continue the development of the poster presentations as a stepping stone to scientific presentation. With the end of the Spring BSAVA meeting there is now a need for cardiology researchers to be able to present abstracts in the UK. We aim to continue bi-annual VCS meetings and we are exploring the potential to host cardiology abstracts at the Spring VCS meeting.

This year the Cardiac Screening Advisory Group (CSAG) also successfully launched a breed screening accreditation exam. Whilst breed screening remains a focus for a minority of members without increased breed screening capacity it may be harder to help progress more formal breed screening schemes with the aim of improving breed welfare.

There are a variety of ways in which the society could develop further to support members interests. As well as supporting existing projects these might include exploring additional ways to help members stay abreast of recent developments or to assist research from practice.

As a member of the VCS this is your society – for you to help shape the future of.

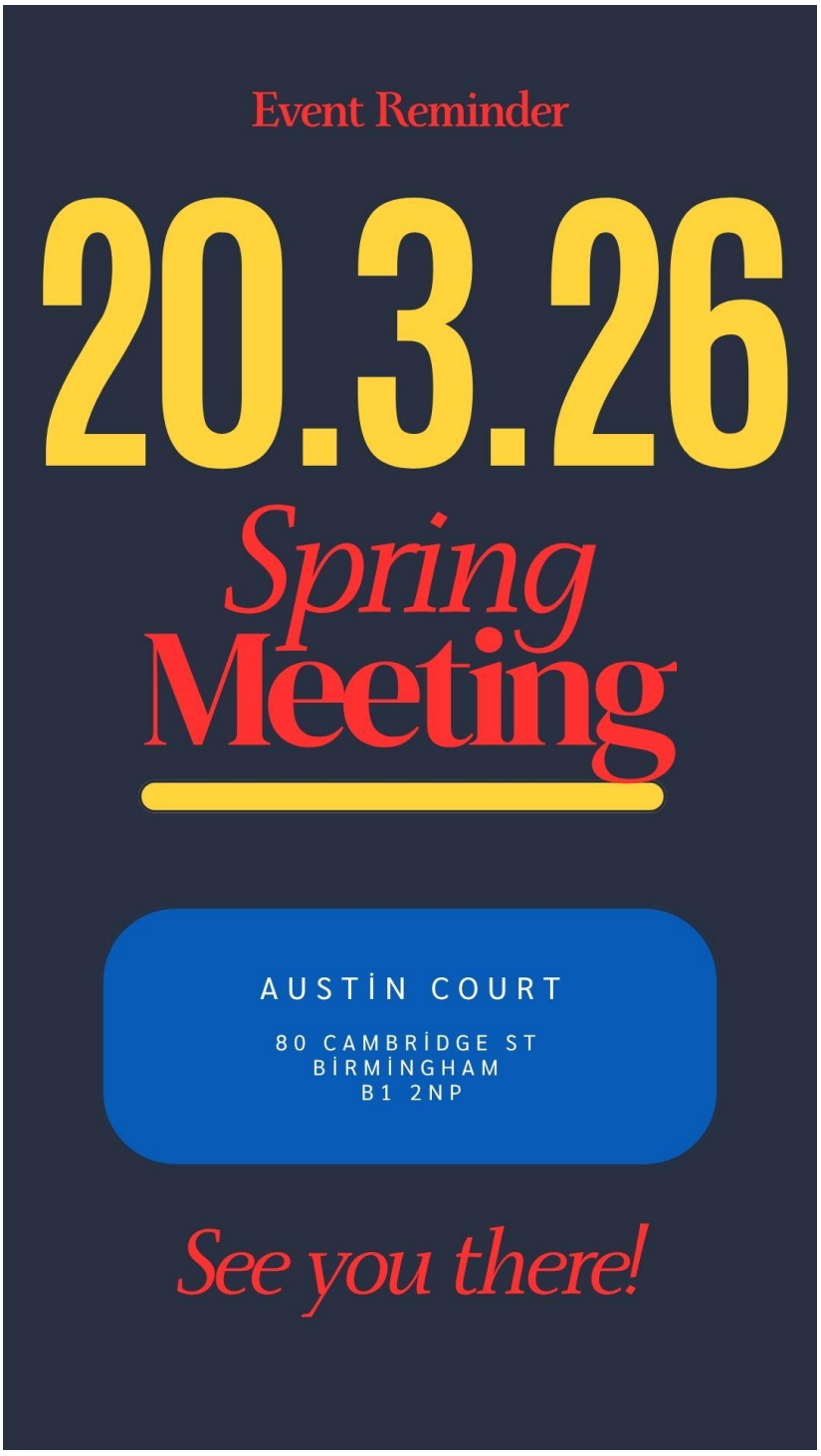
I would like to thank the committee: Vicky Ironside; Claire Denny; Julie Hamilton-Elliott; Sid Sudunagunta, and Mattia Basili for the enormous amount of work they have put into running VCS this year and in organising an amazing 50th Anniversary meeting.

I would also like to extend our thanks to Liza Ebeck and Lauren Osborne for programming the veterinary nurse stream; the Cardiac Screening Advisory Group and members developing clinical audit, and for the BEVA liaisons, John Keen and Katya Potter.

Lastly I would to extend our gratitude to Cindy Laing, our event manager, Katrina Culshaw our book keeper and our generous sponsors, all of whom make this fantastic event possible.

Have a great meeting!

Gavin McAulay, VCS Chair


Event Reminder
20.3.26
Spring
Meeting
AUSTIN COURT
80 CAMBRIDGE ST
BIRMINGHAM
B1 2NP
See you there!

VETERINARY CARDIOVASCULAR SOCIETY – a brief history

Dr Peter Darke

Dr Peter Darke published a letter in the Veterinary Record in September 1975, inviting anyone interested in forming a 'special interest' group for veterinarians interested in cardiology, to attend a meeting to be held at the Royal Veterinary College, Camden Town on 2nd October 1975. At this time, Peter was the Lecturer in Small Animal Medicine at Langford, University of Bristol. Peter had completed a PhD in equine cardiology in 1968, and had spent several years in small animal practice, before being appointed at Langford in 1973. By the mid-1970s, there was no formal recognition of specialists by the RCVS , but the Royal College had established diplomas in ophthalmology (DVO) and radiology (DVR). However, although some cardiology was taught in each of the 6 UK veterinary schools at that time, there seemed to be only one veterinary cardiologist who could be considered a specialist, as equine cardiology was his main teaching and research commitment: Dr J R Holmes, at Langford. Sadly he showed little interest in our proposed development of a special interest group: he joined later, when we were fairly well established, and he then was elected as one of the early VCS chairmen.

Early members were a mixture of people that were known to be teaching cardiology in the various veterinary schools; had published a little in the field; were working in the pharmaceutical industry; or as 'GPs' were known to be practising cardiology. These included Dr Brian Alps (Syntex pharmaceuticals) – appointed as first chairman; Dr Peter Darke (Bristol University) – appointed as first Secretary/Treasurer; Mike Litttlewort (Cambridge University); Dr Brendan Glazier (Dublin); Dr E W Fisher (Glasgow University); Dr Harry Ritchie (Liverpool University); and Chris Hillidge (RVC – he helped to organise the venues for our early meetings), along with various folk in general practice, such as: Ron Lowe (Yorks) – an early Honorary Secretary, but he moved on to the field of oncology; Dermod Malley (Essex), who developed a greater interest in 'exotic' species; John Sheridan (Sussex?); and Rob Thomas (Canterbury). Other vets joined the group relatively soon, including: Prof Rod Else (Bristol then Edinburgh); Sue Matic (Cambridge); Dr Simon Swift (Liverpool); Dr Paul Wotton (Bristol, then RVC); Dr Serena Brownlie (RVC); and Dr Malcolm Cobb (RVC). Quite early in the VCS era, some continental vet cardiogists joined the VCS, notable from Sweden, France, Belgium, Holland and, Portugal, in the years before the European group (ESVC) developed. From the mid-1980s it became normal practice for postgraduate students and residents to join the VCS as part of their training.

At our inaugural meeting, the late Dr Alps insisted on 'vascular' being part of the title of our organisation, although it could be said that he had a vested interest, as the development of vasoactive drugs was his field of study! The structure and function of the VCS was based on the well-established model of the British Veterinary Radiology Association (founded in the 1960s and now part of the European Association of Veterinary Diagnostic Imaging), and their pattern of inviting human medical specialists to speak at their meetings was adopted. It is reassuring that this is still the case.

For some years we held only one meeting a year, usually at the RVC, as an accessible central English location free-of-charge, and attendance at the annual meeting was sometimes a little thin (the minimum number seems to have been about a dozen). But as early as February 1977, the meeting was held at Leahurst, the field station for Liverpool University. The VCS started to grow fast in the mid-1980s, as the RCVS developed the Certificate and the Diploma in Veterinary Cardiology, when candidates were required to show active involvement in cardiology. In the mid-1980s, we started to hold two meetings a year, with a one-day meeting at the time of the BSAVA conference in London, and this led BSAVA to incorporate meetings of various specialist groups on the day before BSAVA Congress itself opened – we were the pioneers in this concept! Sometimes the European Society of Veterinary Cardiology (founded in 1981) have joined us in organising meetings (e.g. in 1985).

In good hands, the VCS has continued to grow, and to produce interesting and varied programmes for the scientific part of the gatherings. The tradition of a good spread of papers from medical specialists presenting a narrow feature of human cardiology through young residents or practitioners presenting individual cases or case series, through to experts in the field of veterinary cardiology presenting overviews of their research of clinical experiences is a healthy part of our meetings. We have contributions from our continental colleagues which are still well accepted. Furthermore, we have always tried to include members and speakers working with species other than small animals, as it is important that our society should cover a broad field. The fact that there are now at least four [or is it 5 now??] professors of Veterinary Cardiology in the UK is also to some extent a credit to the strength of our group. The fact that there seem to be no veterinary cardiologists in the UK that shun the VCS is also a credit to the management of the VCS, in that no-one seems to distance themselves from our organisation. And the sound finances of the VCS are a credit to the (honorary) committee members. May the VCS flourish in the next 50 years, whatever the modern world may bring!

Peter Darke, 2022

VETERINARY CARDIOVASCULAR SOCIETY

A meeting is to be held at the Veterinary School, Park Row, Bristol 8 (by the main University Tower in the City) on Wednesday, September 29th, 1976.

PROGRAMME

10.00 - 10.30 a.m.	Coffee
10.30 - 11.15 a.m.	"Vectorcardiography in the Horse" Speaker: Dr. J.R. Holmes, M.V.Sc., M.R.C.V.S.
11.15 - 12.00 noon	"Physiological Basis for the Interpretation of the ECG". Speaker: Mr. M.W. O'Callaghan, B.V.Sc.,M.R.C.V.S.
12.00 - 12.45 p.m.	"Some Considerations of Cardiovascular Surgery" Speaker: Mr. D.G.Clayton Jones, BVet.Med.,MRCVS.
12.45 - 2.15 p.m.	L U N C H
2.15 - 3.00 p.m.	Acquired C.V. disease in the Cat ~~"Thromboembolic Disease in Cats".~~ Speaker: Mr. T.J.Gruffydd-Jones,B.Vet.Med.,MRCVS.
3.00 - 3.45 p.m.	Case Reports and Discussion: Those attending the meeting are invited to bring material for discussion.
3.45 - 4.15 p.m.	ANNUAL GENERAL MEETING
4.15 p.m.	T E A

The cost of the meeting (including coffee, lunch and tea) will be £1 for members £2 for non-members.

Will you please return the attached slip to me not later than 18th September, 1976.

P.G.G.Darke,
Hon.Sec. V.C.S.,
Langford House,
Langford, Bristol. BS18 7DU

STD. (0934)
Tel: Churchill 852581


VETERINARY
CARDIOVASCULAR SOCIETY
Veterinary Cardiovascular Society
Golden Fiftieth Anniversary Meeting
in association with the British Equine Veterinary Association.
Thursday 6th November (BEVA),
Friday 7th & Saturday 8th November 2025 (VCS)
Chesford Grange Hotel, Warwickshire

NOVEMBER MEETING: 7th November 2025

0830 - 0900	**Registration and Coffee**
0900	**Introduction**
0900 –0945	**The Development of Echocardiography in Veterinary Medicine** ***John Bonagura*** *North Carolina State University*
0950 – 1035	**Multiple Catheter Recording, 3D Mapping and Radiofrequency Ablation In Horses: A New Era in the Arrhythmia World** ***Gunther Van Loon*** *University of Ghent*
1040 – 1110	**Coffee break and sponsors exhibition**
1110 - 1125	**ACVIM update** ***Eve Lo*** *Royal Veterinary College*
1130 – 1220	**Peer Review in the Publication Process – How to Undertake Peer Review of Scientific Manuscripts** ***Adrian Boswood*** Royal Veterinary College
1225 - 1325	**Clinical Audit in the Veterinary Cardiovascular Society** ***Mike Martin, John Keen, Geoff Culshaw, Tobias Wagner***
1330 - 1435	**Lunch break and sponsors exhibition**
1435 – 1520	**3D Assessment of Right Heart Remodelling in Patients with Atrial Fibrillation** ***Dr Luigi Badano*** Center for Integrated Cardiovascular Diagnosis, Istituto Auxologico Italiano, IRCCS, Milan, Italy
1525 - 1555	**Case Studies in Echocardiography** ***John Bonagura*** *North Carolina State University*
1600 - 1625	**Coffee break and sponsors exhibition**
1625 - 1725	**VCS Annual General Meeting (AGM)**
1730 - 1830	**Cheese and wine with sponsors**
1900 - 1930	**Pre-dinner drinks**
1930	**Dinner**

NOVEMBER MEETING: 8th November 2025

0830 - 0900	**Registration and Coffee**
0900	**Introduction**
0900 –0930	**The Evolution of Veterinary Cardiology** ***Malcolm Cobb*** *University of Nottingham*
0935 – 1020	**ECVIM update** ***Francesca Edgerton,*** *University of Edinburgh* ***Anna Jakubczak,*** *Country Vets* ***Laura Korenchy,*** *ChesterGates Veterinary Specialists*
1025 – 1055	**Coffee break and sponsors exhibition**
1055 – 1125	**Management of Acute CHF in Cats: Quick Improvement Means Soonest Home** ***Rebecca Stepien*** *University of Wisconsin*
1130 - 1200	**Bugs in the System: Transient Myocardial Thickening** ***Phil Tricklebank*** *Hallam Veterinary Centre/Pride Veterinary Referrals*
1205 - 1235	**Feline Anaesthesia** ***Tristan Merlin*** *Eastcott Veterinary Referrals*
1240 - 1345	**Lunch break and sponsors exhibition**
1345 – 1400	**ARVC in the Cat: A Challenge** ***Ana Maria Pentel*** *Vet Specialists Scotland*
1405 – 1435	**DCM in Deerhounds: Some Heart-Stopping Pathology** ***Natasha Wayne-Wynne and Emily Dutton*** *Cheshire Cardiology*
1440 - 1510	**Hearts of Champions: Canine Athletes and the Whippet Addendum** ***Rebecca Stepien*** *University of Wisconsin*
1515 - 1545	**Coffee break and sponsors exhibition**
1545 - 1645	**Panel discussion: What is "gold standard" veterinary cardiology? What does the future look like?** ***Chair: Malcolm Cobb*** ***Rebecca Stepien*** ***Emily Dutton*** ***Chris Linney*** ***Lesley Young***
1700	**CLOSE**

The Veterinary Cardiovascular Society gratefully acknowledges the support of all our sponsors.

GOLD SPONSORS

Ceva Animal Health Ltd,
Telephone: +44 01628 334 056
Website: https://www.ceva.co.uk

Boehringer Ingelheim Animal Health
Tel: +44 (0) 1344 746 959
E-mail: vetenquiries@boehringer-ingelheim.com
Website: https://www.boehringer-ingelheim.com/uk/animal-health

Probo Medical
Tel: +44 333 004 7657
E-mail: sales@probomedical.co.uk
Website:
https://www.probomedical.co.uk/

SILVER SPONSORS

HBLB
Tel: +44 020 7333 0043
E-mail: enquiries@hblb.org.uk
Website: https://www.hblb.org.uk

HeartVets
Tel: +44 (0) 800 999 4333
E-mail: diagnostics@heartvets.co.uk
Website: https://heartvets.co.uk

BRONZE SPONSORS

Imotek

Tel: +44 (0) 1487 843 193

E-mail: sales@imotek.com

Website: www.imotek.com

IMV IMAGING

Tel: +44 (0) 1506 460023

E-mail: Hello@imv-imaging.com

Website: www.imv-imaging.co.uk

Infiniti Medical

Tel: +44 (0) 20-8533-9922

Website: https://infinitimedical.com

Email: europe@infinitimedical.com

QMS LTD

Tel: + 44 01981 433117

Email: info@qms-med.com

Website: www.qms-vet.com

LinkedIn profile:

https://www.linkedin.com/company/qms-veterinary/

BRONZE SPONSORS

SPACELABS HEALTHCARE LTD. (UK)

Website: https://spacelabshealthcare.com

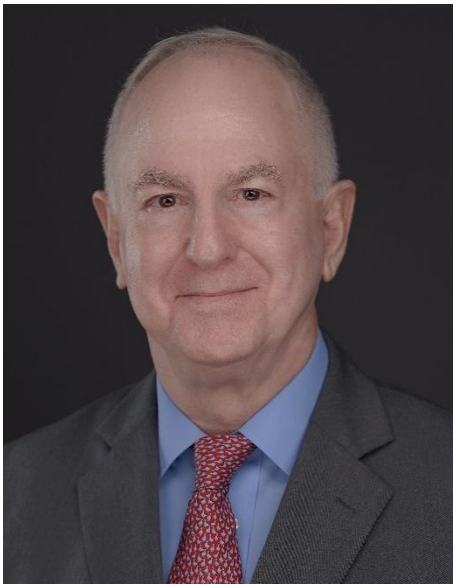
Tel: + 44 (0) 1992 507700

Timeless Veterinary Systems
Website: https://timelessveterinary.com
Email: sales@timelessveterinary.com
Tel: +1 (855) 247-2327

VetPlus
Email: customer.services@vetplus.co.uk
Tel: 01253 797400

Biographies

John Bonagura DVM, MS, FASE, DACVIM (Cardiology, Internal Medicine)



John Bonagura is a graduate of the Ohio State University. He completed a rotating internship at the Animal Medical Center and residencies and specialty board certifications in Cardiology and Internal Medicine at Ohio State. Dr. Bonagura was Head of Cardiology & Interventional Medicine at Ohio State for much of his career and is currently Professor Emeritus. John was visiting research fellow at Edinburgh University, Scotland, and he later held the Gilbreath-McLorn Endowed Professor of Cardiology at the University of Missouri before rejoining the faculty at OSU in 2001. Since 2019, he has been affiliated with North Carolina State University College of Veterinary Medicine and is presently Adjunct Professor of Cardiology at Colorado State University CVM. John is the long-time Editor of Kirk's Current Veterinary Therapy, a co-author of the Colour Atlas of Veterinary Cardiology and Cardiovascular Diseases in Companion Animals, and he has co-authored over 300 scientific publications and book chapters. He has served as President of the ACVIM specialty of Cardiology and as an editorial board member for the *Journal of Veterinary Cardiology*, *American Society of Echocardiography CASE*, and *Journal of Veterinary Internal Medicine*. Dr. Bonagura is recipient of multiple teaching awards including the Ohio State University campus Award for Distinguished Teaching. Other honors include the Bourgelat award (from BSAVA), Kirk Lifetime Achievement Award (from ACVIM), Doctor Honoris Causa (from the Autonomous University of Barcelona), Faculty Achievement Award (AAVC), and distinguished Alumnus awards from The Ohio State University College of Veterinary Medicine and the Animal Medical Center.

Gunther Van Loon, DVM, PhD, DECEIM, Ass DECVDI

Gunther van Loon graduated from Ghent University, Belgium, in 1992 and has worked at Ghent University, Equine Internal Medicine, ever since. In 2004 he became ECEIM Diplomate and in 2011 Associate Member of ECVDI. In 2001 he finished his PhD on “Atrial pacing and experimental atrial fibrillation in equines”. He is author or co-author of about 220 A1 publications and 26 book chapters, and gave about 260 presentations at international congresses. In 2015 he received the British Equine Veterinary Association (BEVA) award for ‘Clinical Research’ (Liverpool, UK) and in addition the prestigious Merial Applied Equine Research Award for outstanding research regarding ‘Advances in Equine Cardiology’, awarded by the World Equine Veterinary Association (WEVA) (Guadalajara, Mexico). Gunther van Loon is head of the Equine Internal Medicine department at Ghent University and head of the Equine Cardioteam Ghent. He is Past-President of the Belgian Equine Practitioners Society (BEPS). His major interests are in the field of equine cardiology and vascular diseases with specific expertise in cardiac ultrasound (including 4D echo, intracardiac echocardiography), cardiac biomarkers, arrhythmias, electrophysiological studies, cardiac pacing and pacemaker implantation, 3D electro-anatomical mapping and radiofrequency ablation. Gunther has established the Equine Cardioteam Ghent which performs high level cardiac research and advanced treatment of clinical cases in a fully equipped lab.

Eve Lo, BVM&S MRCVS

Eve graduated from the University of Edinburgh. After this, she worked in general practice prior to undertaking a Clinical Fellow in Veterinary Cardiology position at the Royal Veterinary College (RVC). She then continued on at the RVC as a cardiology resident, where she is currently in her final year.

Prof. Adrian Boswood MA, VetMB, MRCVS, DVC, DipECVIM-CA (Cardiology)

Adrian graduated from Cambridge University Veterinary School in 1989. After graduation he spent a year in mixed practice. In 1990 he joined the Royal Veterinary College, University of London as an intern and he has worked there ever since. He is currently Professor of Veterinary Cardiology and holds the position of Vice Principal for Learning, Teaching and Assessment.

He obtained the RCVS Diploma in Veterinary Cardiology in 1996 and the ECVIM Cardiology Diploma in 2001. He is past President of the European College of Veterinary Internal Medicine and has chaired the Quality and Standards committee of the European Board of Veterinary Specialisation.
His main research interests lie in the diagnosis, progression and treatment of acquired canine cardiovascular diseases. He has a particular interest in myxomatous mitral valve disease and has been involved in a number of influential clinical trials.

Mike Martin MVB DVC MRCVS Veterinary Cardiologist & Interventional Coach

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, until 2020 when he retired from clinical practice. He has been both Honorary Secretary and Chairman of the Veterinary Cardiovascular Society (UK), and examiner for the Diploma in Veterinary Cardiology for the Royal College of Veterinary Surgeons.
Since 1992 to 2015, he ran his own private referral practice (The Veterinary Cardiorespiratory Centre, Kenilworth). In October 2015 he moved his Cardiology Service into Willows Referral Centre, Solihull, UK. Since the beginning of 2018 he is again self-employed, providing consultancy work, training in cardiac interventions at specialist centres around Europe, as well as lecturing at CPD events around the world.
He is honorary veterinary cardiology adviser to Twycross Zoo and the Ape Heart Project since 2014 performing echocardiographic examinations, ECG and general research guidance on the great apes as well as other zoo animals. He has published over 40 scientific peer reviewed papers. He is author of two 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 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.

John Keen BVetMed PhD Cert EM (Int Med) Dip ECEIM FRCVS

Following graduation from the Royal Veterinary College, London in 1996, John spent 4 years in mixed and then equine practice before being appointed the RCVS Clarke and Sparrow Resident in Equine Studies at the 'Dick' Vet in 2000. He has remained there ever since and currently holds a personal Chair in Equine Cardiovascular Medicine. Although the 'day job' is general internal medicine he has special clinical focus on equine cardiovascular disease and, with two other colleagues, runs a peripatetic transvenous electrocardioversion service ('The Dicky Tickers'). Research interests focus on the relationship between cardiac structure and function, particular in the Thoroughbred (athletic) heart.

Geoff Culshaw BVMS PhD CertVC DVC SFHEA FRCVS

Geoff graduated from Glasgow vet school in 1994. After 11 years in practice, he joined the R(D)SVS where he heads the cardiology service. He is an RCVS Diplomate in veterinary cardiology and an RCVS recognised specialist.

In 2018, Geoff completed a PhD on endothelin-1 and renal salt handling at the Queen's Medical Research Institute, and his post doctoral research focused on renal sodium and glucose transport in diabetes. Geoff is

also a clinical research associate of the Roslin Institute. His research interests include cardiorenal interactions and the role of senescence in cardiovascular and renal disease.

Tobias Wagner Dip ECVIM-CA (Cardiology) Dr.med.vet. 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. Tobi then moved to the Royal Veterinary College, London, completing his cardiology residency and attaining his ECVIM-CA(Cardiology) Diploma in 2010. Tobi then joined Southern Counties Veterinary Specialists where he has been working as an European Specialist in Veterinary Cardiology for the last five years. After a brief spell back in Germany, Tobi returned to work full-time as a Specialist in Veterinary Cardiology and continue developing the very successful interventional cardiology service at SCVS. Tobi's main interest is feline cardiology and interventional cardiology.

Luigi Badano MD, PhD, FESC, FACC Honorary Fellow ASE, EACVI and BSE

Prof. Badano currently serves both as Full Professor of cardiovascular medicine and coordinator of the Ph.D. course on Public Health at the University of Milano Bicocca (Milan, Italy), and as director of the integrated cardiovascular diagnosis unit at the Istituto Auxologico Italiano, IRCCS, Milan, Italy.

Prof. Badano's clinical interests include native and prosthetic valvular heart disease, right ventricular and atrial function, with research interests in three-dimensional and deformation imaging echocardiography, and cardiac mechanics.
Prof. Badano is a member of the Italian Society of Cardiology, European Society of Cardiology, European Association of Cardiovascular Imaging, American College of Cardiology, and the American Society of Echocardiography (ASE). He is also a fellow of the European Society of Cardiology and the American College of Cardiology. Prof. Badano is a regular invited speaker at major international cardiology meetings as well as at annual ESC, ACC, ASE and EuroEcho scientific meetings
He has 387 peer-reviewed publications (current Scopus h-index= 85), authored 9 books about echocardiography and cardiovascular imaging. Prof Badano has been listed among the highly cited researcher from 2018 to 2021 by Clarivate- Web of Science to have produced multiple highly cited papers that rank in the top 1% by citations for field and year. Prof. Badano served as President of the European Association of Cardiovascular Imaging from 2010 to 2012. He is an honorary member of the Hungarian, Romanian, and Korean Societies of Cardiology. He is also honorary Fellow of the American Society of Echocardiography, the British Society of Echocardiography, and the European Association of Cardiovascular Imaging. In 2013, he was awarded the silver medal of the European Society of Cardiology for his clinical and research activity and his commitment as President of the European Association of Cardiovascular Imaging.

Malcolm Cobb MA, Vet MB, DVC, PhD, MBA, FHEA, MRCVS

Malcolm graduated from Cambridge in 1984 and spent 4 years in mixed practice in East Anglia. After a residency in internal medicine at RVC, he obtained his DVC in 1993, and a PhD in canine dilated cardiomyopathy in 1996. In 1997 he joined LEO Animal Health as Technical Manager, obtained an MBA in 2002 and was appointed Country Manager of LEO for the UK and Ireland. In September 2005 he joined the new Veterinary School at Nottingham as Deputy Head of School where he has contributed to the establishment of the School and the development of the curriculum. Malcolm has completed a number of vet school accreditation visits on behalf of the RCVS within the UK and abroad.

Francesca Edgerton BSc MRCVS

Francesca graduated from the Royal Veterinary College in 2020 before spending 2 years in general practice in Liverpool. Following this she completed a rotating internship at the University of Edinburgh where she is currently a final year Cardiology resident. Her research interests include Holter monitoring and interventional surgery.

Anna Jakubczak, DVM MRCVS

Anna graduated from the Warsaw University of Life Sciences in Poland in 2014. She spent four years working in an emergency and referral hospital in Poland before moving to the UK. After three years in general practice, she completed a 13-month rotating internship at Lumbry Park Veterinary Specialist in 2023. She then worked as a cardiology intern in an independent referral hospital in Cheshire. Anna is currently working towards her certificate in cardiology (CertAVP VC) and intends to undertake another cardiology internship and residency in the near future.

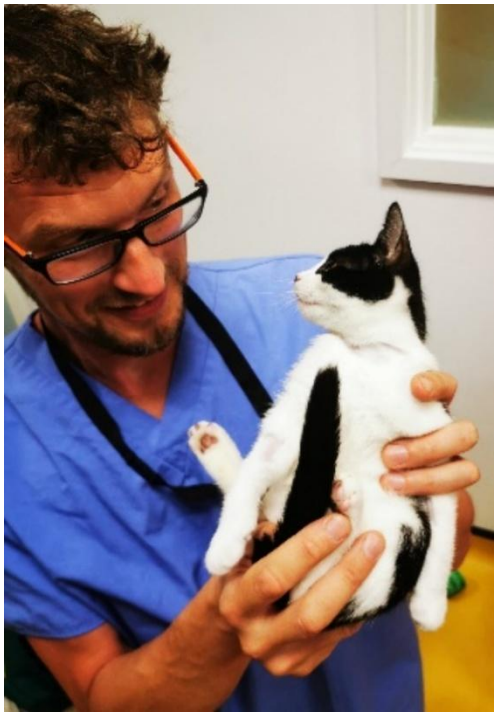
Laura Korenchy, DrMedVet Cert AVP(VC) MRCVS

Laura graduated in 2015 from Szent Istvan University in Budapest with a degree in Veterinary Medicine. She worked for a year in Hungary before moving to North Wales, where she gained experience in a busy primary care hospital. In 2019, Laura joined James Specialist Cardiology, achieving advanced practitioner status. She has been working as a peripatetic cardiology clinician in North Wales and Cheshire. Currently, she is completing an internship at ChesterGates Veterinary Specialists, aiming for a residency in veterinary cardiology. Her main interest are feline heart disease, including feline friendly practices and stress free investigations.

Rebecca Stepien DVM MS DACVIM (Cardiology)

Dr. Rebecca L. Stepien is an Emerita Clinical Professor at the University of Wisconsin School of Veterinary Medicine, where she was a Clinical Professor Cardiology for more than 30 years. She is a past president of the Specialty of Cardiology in the American College of Veterinary Internal Medicine and is the associate editor for veterinary medicine at CASE Journal (Cardiovascular Imaging Case Reports) and a founding member of the Cardiac Education Group (cardiaceducationgroup.org). Dr. Stepien is a frequent speaker at national and international conferences. Her research interests include myxomatous valve disease in whippets, the effect of athletic training on the canine heart, diagnostic echocardiography and systemic hypertension.

Phil Tricklebank



Phil Tricklebank is an RCVS Certificate Holder cardiologist, general practitioner and Clinical Director at Hallam Vets in Sheffield. He also sees cases at Pride Referrals in Derby and does some peripatetic work. He has been somewhat involved in the past in research into risk indictors in cats with preclinical hypertrophic cardiomyopathy. It feels like most of his working day is spent herding cats. He has two grown-up children, two cats and a dog. He has too many bikes but enjoys riding them all with a preference for mountain biking; he possesses no lycra and refuses to wear it.

Tristan Merlin

Tristan qualified in France (ONIRIS, Nantes) in 2013 before moving over to the UK in 2015, where he completed a residency in Anaesthesia and Analgesia at the Royal Veterinary College. Following this, he started the anaesthesia department at Eastcott Referrals in Swindon, and led the team there until 2021. He became a Diplomate of the European College of Veterinary Anaesthesia and Analgesia that same year.

Ana Maria Pentel

Ana Maria graduated from the Technical University of Lisbon, Portugal, in 2010. After graduation, Ana Maria returned to the UK where she worked in general practice while studying for her Certificate in Veterinary Cardiology. This was completed in 2023. Ana Maria joined the cardiology team at Veterinary Specialists Scotland in 2025 and she enjoys all aspects of cardiology.

Natasha Wayne-Wynne

Tasha graduated from the Royal Veterinary College in 2020. She worked in a first opinion clinic in London before completing a rotating internship at Willows Referral Hospital, followed by a cardiology internship at Cheshire Cardiology. Tasha is currently a first year cardiology resident at Langford Vets. Her main interests are interventional cardiology and management of cardiac emergencies.

Emily Dutton BVM&S DVC MRCVS

Emily graduated from the University of Edinburgh and was awarded the Diploma in Veterinary Cardiology in 2014, shortly followed by RCVS Specialist Status in Veterinary Cardiology. Emily was appointed Secretary of the Veterinary Cardiovascular Society in 2015 and has been heavily involved in CPD training for vets. She has contributed to international clinical trials and has written a number of articles for professional and peer-reviewed journals including establishing echocardiographic reference intervals in deerhounds. Her interests include echocardiography, interventional cardiology, congenital heart disease and cardiomyopathy in sphynx cats. Despite this, she is currently actively researching ambulatory ECG recordings, cardiac biomarkers and the genetics of dilated cardiomyopathy in deerhounds. Emily is Director of Cheshire Cardiology, offering a specialist cardiology referral service for veterinary practices throughout the North West.

A Brief History of Veterinary Echocardiography

John D Bonagura, DVM, DACVIM, FASE

Veterinary echocardiography is an indispensable diagnostic study. It has transformed the recognition, staging and management of cardiac diseases in both small and large animals. The evolution of echocardiography in veterinary practice has paralleled that of human investigation and has been fueled by the continuing priorities for obtaining detailed and non-invasive cardiac assessment. The author first performed clinical M-mode studies in 1975 and has been privileged for the past 50 years to witness the tremendous developments in echocardiography in medicine in general and also within our profession. Notably, multiple members of the Veterinary Cardiovascular Society have been instrumental to this advancement.

This presentation focuses on a number of topics, that include highlighting some of the milestones in veterinary echocardiography and – in the hope of stimulating discussion – offering a personal perspective about areas of cardiac ultrasound that might require some reconsideration or future refinement. Meeting participants interested in obtaining a PDF version of the presenter's slides can scan the QR code that will be displayed during the meeting or email the author at jdbonagu@ncsu.edu or john.bonagura@colostate.edu

Earliest Studies

The roots of echocardiography can be traced to the discovery of piezoelectricity in the late 1800's by the Curies. The technology was developed for military use in the 20th century and subsequently constituted as a clinical tool in the 1950s in Sweden by the medical cardiologist Inge Edler (who imaged calves in his research) and by the physicist Hellmuth Hertz. Dr. Satomura in Japan and Dr. Kalmanson in France pioneered applications of the Doppler echocardiography in humans based on a principle originally applied to observations involving light (and later to audible sound). Detailed clinical applications of spectral Doppler in humans were subsequently reported by the Norwegian team headed by the physician Liv Hatle and the engineer Bjørn Angelsen (who the presenter had the privilege of meeting in Glasgow at the 1988 meeting of the British Medical Ultrasound Society, where a number of veterinarians presented their work).

Initially, A-mode (Amplitude-mode) studies were obtained (probably of greatest application to midline shifts in the brain). But the A-mode turned "on-end" converted amplitude to brightness (B-mode) and when swept across (actual) moving paper formed the Motion or M-mode echocardiogram. Dr. Harvey Feigenbaum, the first President of the American Society of Echocardiography, was prominent in the advancement of this modality, which was soon adapted by veterinarians, who also attended some of his courses in Indianapolis. This led to an explosion of clinical studies in adults, children and eventually veterinary patients. Some of the earliest veterinary papers were published by Dr. Frank Pipers from Ohio State, where (as best the author can determine) the first veterinary M-mode course in the US was also held (with instructors that included Drs. Pipers, Mark Kittleson, and Bob Hamlin). This meeting attracted prominent cardiologists – such as Dr. George Eyster – as well as researchers from Colorado State University who were early adaptors of this technology (with June Boon, a veterinary technician eventually publishing the first detailed textbook on the subject). In 1980, Frank and I delivered two "state of the art" echocardiography lectures at the American College of Veterinary Internal Medicine Forum in 1980, summarizing an increasing body of work by produced by investigators around the world.

The first sector scanners were introduced in veterinary practice in the 1980s and the number of cardiologists and radiologists adapted "general" B-mode platforms and later cardiac-specific imaging systems to veterinary patients, small and large. There are too many individuals to cite in this abstract but one – Dr. Bill Thomas at UC Davis – was a leader in advancing 2D imaging in our profession and headed the first (and to date, only) published guidelines for 2D imaging in dogs and cats (more on this subject later in the lecture). The 2D study offered a more comprehensive anatomical assessment, enabling us to visualize the spatial relationships of cardiac structures and identify abnormalities more accurately. Ultrasound venders came and went (often swallowed by larger companies who adapted their technologies). The technical advances led to the migration

from (often gigantic) mechanical sector scanners to phased array transducer systems. Overall, the veterinary focus was applying echocardiography to morphologic and functional diagnoses and to quantitation of cardiac size and function – the latter issue being one that still confounds!

Doppler modalities were added to M-mode and 2D imaging systems, and since most large referral centers used human hospital grade equipment, these newer modalities were adopted for veterinary use. As I will attempt to demonstrate during this presentation, some of the earliest reported Veterinary Doppler studies were performed in dogs, horses, and cats in the late 1980s and early 1990s by multiple members of the Veterinary Cardiovascular Society. (The author experienced a wonderful sabbatical year at Edinburgh [and the UK in general] in 1989-1990 and had the chance to witness this evolution first-hand). Doppler imaging augmented the anatomical studies of M-mode and 2D imaging and vastly improved our understanding of congenital heart defects beyond that of 2D imaging augmented by saline contrast. Further applications that included forms of tissue Doppler imaging have also been widely adopted in specialty practices, although the day-to-day value of these modalities for clinical decision-making requires better definition.

Further advancements in technology are well recognized by members of this Society and include transesophageal imaging, 3D/4D imaging, speckle tracking, the use of human contrast agents for delineating structure, and new forms of blood flow mapping. The value of TEE for guidance of interventional procedures is clearly evident from publications, case reports and personal experience. Contrast agents have been used intermittently for decades but should perhaps become more routine, especially as newer studies demonstrate their advantages to feline or coronary imaging. In terms of the “black box” technologies, there are scores of veterinary publications and research studies devoted to these topics, however their application and advantages in our daily workflow require more study. Some examples of these applications as well as challenges involved with their applications will be offered.

The role of artificial intelligence and machine learning in echocardiography holds immense promise but also pitfalls. AI algorithms could potentially automate measurements, improve diagnostic accuracy, and even predict disease progression, ultimately leading to more efficient and effective cardiac care for animals. How these will be validated and standardized for the wide range and physiology of veterinary species is uncertain.

Unresolved Issues and Future Investigations

There is still much to do. Despite the tremendous progress seen in veterinary echocardiography, we can all list some of the many challenges and gaps in our knowledge. Here is a short and quite incomplete one:

1) Technology and instrumentation optimized for our patients, think: 3D imaging, standardization of vendors for speckle tracking, processing Doppler signals used for hemodynamic quantitation, and use of contrast agents.

2) Standardization: We have limited guidelines and the upcoming ASE guidelines will highlight some of the different approaches that many might not be willing to change or adapt. Somehow our specialty needs to develop a consensus on standardizing image acquisition and interpretation and be willing to change as new data are presented.

3) Reproducibility, in the most general context: Standardization should help but perhaps we need clinical studies that involve more agnostic “core” echo laboratories to better evaluate accuracy and precision and enforce quality control.

4) Diagnostic Criteria, think: Feline HCM, canine subaortic stenosis, and preclinical canine dilated cardiomyopathy – can these diagnostic “standards” be clearly linked to outcomes or to underlying genetic or pathophysiologic stages?)

5) Quantitation: Aside from the measurements that defines feline HCM and canine DCM, we also stage, treat and prognosticate a number of disorders based on the size or function that we quantify with M-mode, 2D, Doppler, and speckle tracking echocardiography. Therapy of preclinical mitral valve disease is perhaps the most prominent example. Which exponents should be used and when? Can we further refine our approaches to establishing reference ranges?

6) Training of operators: We all should practice within the confines of our training and experience, but how can we better define and validate our expertise to optimize patient care and conserve client resources?

7) How many variables do we need? Consider this simple question: is the diagnosis of congestive heart failure a clinical/pulmonary imaging assessment, supported by echo, or is it an echocardiographic diagnosis used to explain clinical signs? Do we need echocardiographic variables that delineate stages B2 from C or should a competent clinician be able to do that with a basic echo that supports the cardiac diagnosis? Should a "new" echo variable be validated by an "old" one or should it be unique and validated by a different gold standard? Can we move beyond "p-values" and "correlations" in echo studies to focus on clinically meaningful variables or cut-offs that change the way we prognosticate or practice? (I could go on… :)

In summary, the history of veterinary echocardiography is a testament to both our application of comparative medicine or "one health" and the drive for innovation in veterinary medicine. From rudimentary beginnings to the sophisticated, multi-modal imaging available today, echocardiography has fundamentally changed our understanding and management of heart disease in animals. I am honored to be able to offer a personal perspective about this subject that has been so integral to my career as a cardiologist, and believe the future of veterinary echocardiography will bring further value and excitement.

NEW

vetmedin®

ORAL SOLUTION

MORE LIFE TO LOVE. ONE DROP AT A TIME.

Ideal for smaller breeds where precise dosing matters most or for dogs who need a different approach to tablets and capsules.

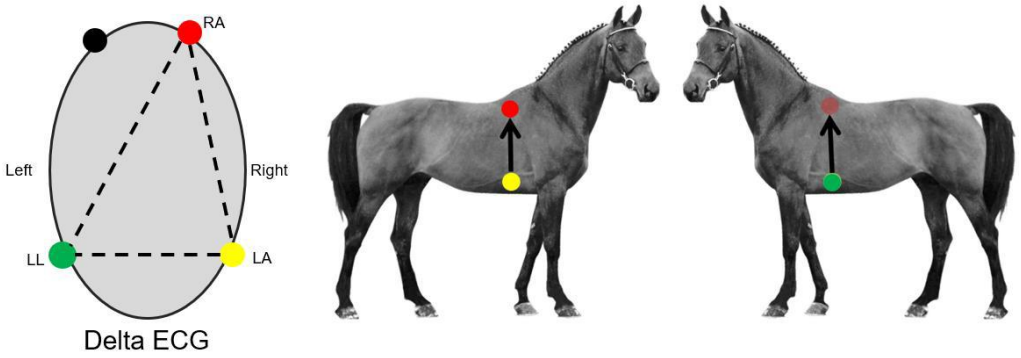
Scan to learn more

Boehringer Ingelheim

Multiple Catheter Recording, 3D Mapping and Radiofrequency Ablation In Horses: A New Era in the Arrhythmia World

Gunther Van Loon

The cardiac depolarization is a complex process which involves precisely timed events from different structures. Arrhythmias change this depolarization process which affects cardiac function and can be associated with impaired athletic ability or even horse and rider safety issues. Surface electrocardiography (ECG) is the method of choice for diagnosing arrhythmias. In human and small animal medicine, the development of electrocardiography (ECG), about a century ago, has led to a continuously improving insight into normal and abnormal rhythms, allowing better diagnosis and treatment of a huge variety of arrhythmias. In equine medicine, techniques for ECG recording and electrode placement were copied from human medicine with somewhat disappointing results: only rate and rhythm could be obtained from the ECG. Due to these shortcomings, there has been limited improvement in the diagnosis and treatment of arrhythmias in horses over the last 50 years. ECGs were not recorded in a standardized fashion, making further progress difficult. Recently, introduction of better equipment and new ECG recording techniques with standardization of electrode placement, has led to a revival of the equine ECG. It has become apparent that arrhythmias in horses are more important than previously thought and an important cause of sudden death in racehorses. A standardized recording technique, such as the 'Delta ECG recording technique', is crucial for further advancing our knowledge on equine arrhythmias. Only standardized ECG recording allows for better pattern recognition of the morphology and phase of the different waves on the different leads. The advantage of this Delta ECG is that it can be further expanded to a 12-lead ECG, which allows to create an orthogonal lead system, apply vectorcardiography and better localize the region of origin of an arrhythmia.

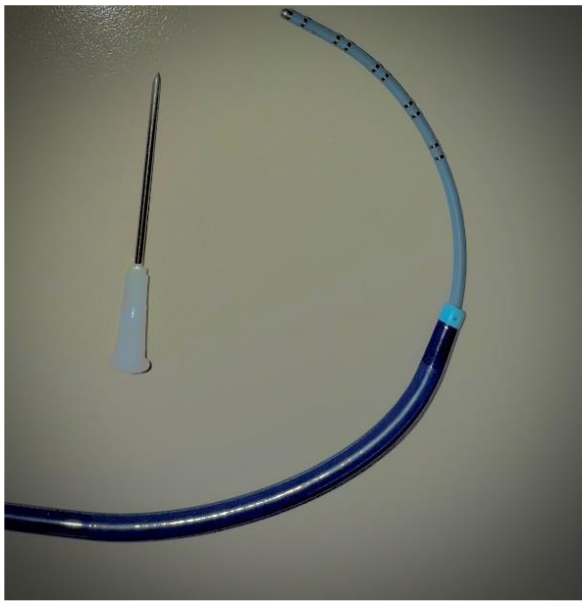


Arrhythmias often originate from abnormal tissue showing altered electrophysiological properties, such as impaired conduction and increased impulse generation. Although medical treatment might be effective to terminate certain arrhythmias, recurrence is relatively common because tissue properties have not changed. Ablation is a treatment technique that does alter the tissue properties by heating (or freezing). This results in non-conductive fibrosis with inactivation of a firing focus or abnormal conduction pathway, or interruption of a reentry circuit. This technique not only terminates the arrhythmia, it also prevents recurrence, which is a major advantage.

The first step towards treatment of an arrhythmia (e.g. premature depolarizations, reentry arrhythmia, accessory pathway, …) is to determine the type of arrhythmia on an ECG. The standardized Delta 3-lead electrode configuration for surface ECG recording was recently introduced and already provides an indication of a right-sided versus left-sided origin of the arrhythmia (unpublished data). Recording a 12-lead Delta ECG and vectorcardiography provides further detail on the area of origin of the abnormal region (left-right and cranial-caudal). Further detail is, however, needed when deciding to treat an arrhythmia by radiofrequency ablation. In human and small animal medicine, further characterization of the arrhythmia can be done with a

catheter-based technique in order to 'map' the arrhythmia. Multiple catheter recording from within the heart, as well as 3D electro-anatomical mapping (3D EAM) are established methods. For multiple catheter recording, typically 4 catheters, each with multiple electrodes, are positioned at strategic locations in the heart. Intracardiac electrograms are recorded and the timing of all signals are compared to each other and to that from a reference electrode. At the same time, electrical stimulation of the myocardium ('pacing') from a specific catheter location might be performed to record the resulting electrical activation pattern of the heart. During such and electrophysiological study, the technique of intracardiac electrogram recording and pacing allows to determine the depolarization pattern of the heart, which is 'an electrical map', to define the earliest activation which could, for example, be an ectopic focus and further allows to unravel the mechanism of an arrhythmia. This '4 wire technique' requires very precise visualization of each catheter in relation to specific cardiac anatomical structures using fluoroscopy, but also incorporates 3D images from CT or MRI.
A totally new imaging technique for catheter guidance in human medicine, uses a magnetic field (or impedance tracking), created over the heart, and catheters with a magnetic sensor. This technique allows 3D guidance of catheters with very high precision for electrical mapping of the heart. It creates a 3D anatomical image of the heart, including all electrical information, which is called a 3D electro-anatomical map (3D EAM). The 3D EAM can show the timing of all potentials and thus create an activation map, or can show the voltage of all potentials, a voltage map. Such a map visualizes the arrhythmia with high anatomical precision and also determines the mechanism (focal, reentry, accessory pathway, …). With this information, the ablation catheter, also with magnetic sensor, can be inserted and manoeuvred to this precise location of the arrhythmia. Subsequently, radiofrequency ablation of that region can be performed to 'destroy' the arrhythmic zone by thermal heating. Specific radiofrequency settings need to be used in order to create a sufficiently deep lesion and avoid damage to cardiac or extra-cardiac structures. In order to inactivate a focal origin several ablation points need to be delivered at that focus. To interrupt a reentry mechanism, the 'point-by-point' ablation technique is used to create an uninterrupted line by closely spaced ablation points to fully interrupt the reentry circuit. Creating such an uninterrupted line very precise manoeuvring the catheter as the size of an ablation lesion is only about 6-9 mm in diameter.
Performing such catheter-based procedures in horses is associated with many challenges.
The size and anatomy of the horse are associated with specific challenges. Total anaesthesia time for such a large animal is limited and horses are more sensitive to complications associated with anaesthesia and recovery, such as fracture, myopathy and myelopathy. A femoral catheter insertion is not possible: the angle between femoral vein and artery with vena cava and aorta hamper catheter insertion or performing a transseptal puncture. Most importantly, the distance from femoral access to the heart is too large which means that commercial catheters, with a typical length of about 110 cm, cannot be used. A transhepatic approach for transseptal puncture in horses was developed but could be associated with complications related to the hepatic vein puncture. Catheterization via the jugular vein or carotid artery is the best option but is associated with a rather tortuous route towards the heart.
The large equine heart means that examination of such a large cardiac compartment (especially atrium) is more complex, more extensive. Mapping of the heart and localizing an arrhythmogenic area therefore takes much more time. Ablation of an area or point-by-point ablation to create a line of block usually requires much more ablation points. In addition, achieving a transmural lesion might not be possible when ablating in thick myocardium: deeply located arrhythmias in thick myocardium might not be reached and creating an uninterrupted ablation line might fail. Moreover, heart rate is very low in horses in anaesthesia (around 30-35 bpm) which means that mapping during a physiological rhythm is very slow. The large heart, with a large internal diameter of the different compartments, makes it difficult to manoeuvre the catheter through the heart and find a stable catheter position, as the catheter more easily 'falls down'. Ideally, catheters should be more stiff, ridged, to facilitate a stable position in the heart. Touching the opposite wall with the catheter tip is often difficult because the curvature of steerable catheters is too small. Therefore, a steerable sheath is usually needed in order to reach every area in the heart and obtain enough contact force with the wall when performing ablation. Finally, the large equine heart is too big to fit in the area of 'view' of certain mapping

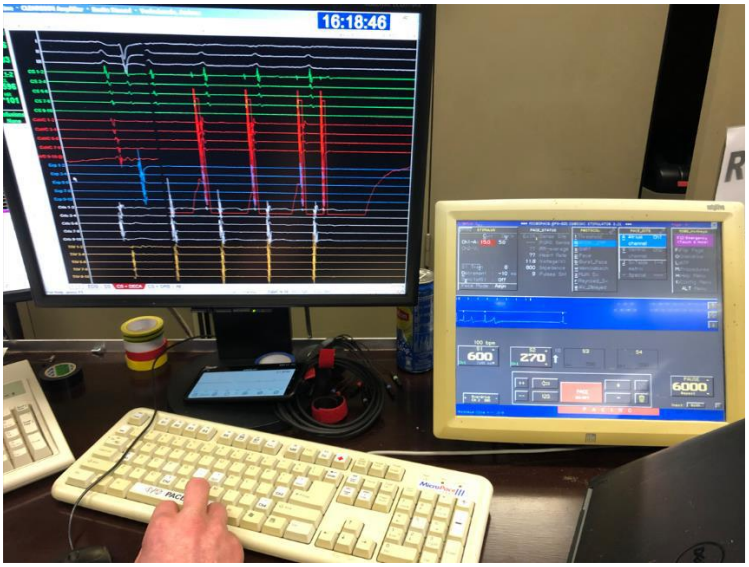
systems, whereby an entire heart might not fit into view. Very careful positioning of the horse on the magnet is needed to get the area of interest into view.



The use of a deflectable sheath (dark blue) facilitates the manoeuvrability of the deflectable decapolar diagnostic catheter (light blue).

Imaging of the heart and catheters is another important limitation in horses. The MRI and CT of the adult horse heart are very difficult, usually impossible, as the horses thorax does not fit in the gantry. But even fluoroscopy is difficult. A fluoroscopic procedure would be associated with massive radiation doses putting the clinicians at risk. Fluoroscopy allows visualizing a catheter in latero-lateral projections, but not in dorsoventral projections, and small anatomical landmarks are barely visible. Therefore, fluoroscopy would only allow rough guidance of catheters towards the heart but detailed positioning could still be challenging. The fluoroscopic procedures would probably require the aid of echocardiography for more precise catheter positioning. However, performing cardiac ultrasound while intermittently using fluoroscopy would require a complex setup whereby it is difficult to avoid the clinician standing very near to the radiation field.

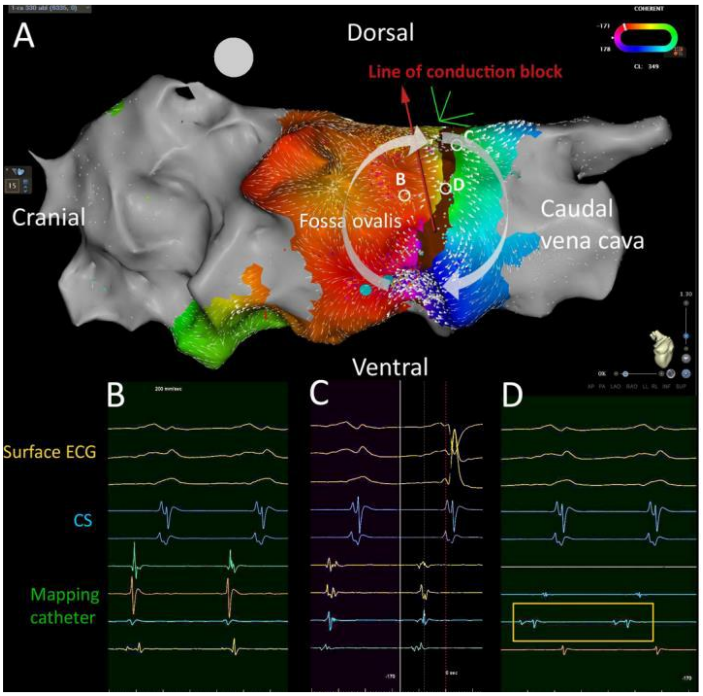
Recently, a technique for placing multiple catheters at specific locations in the heart, solely based on echocardiographic-guidance, has been developed. With this technique, arrhythmias can be better located within the heart and as such, unravel complex arrhythmias. This technique can be applied in the standing horse when mapping the right heart, but requires anaesthesia for the left heart. For the left heart, a retrograde access via the carotid artery, aorta and left ventricle is possible. When performing ablation in the left atrium, this approach is associated with substantial catheter movement because of ventricular contractions. Therefore, a transseptal puncture technique, performed via jugular vein and right atrium, puncturing through the oval fossa, has been developed in horses. Intracardiac echocardiography (ICE) proved to be essential to perform the puncture. Specific challenges of transseptal puncture in the horse include the very thick atrial septum, the fact that a needle cannot be used and the tortuous access to the left atrium. The latter makes manoeuvring the catheters in the left atrium much more complicated.
Echocardiographic guidance of catheters is possible but the technique remains difficult to apply in case catheter ablation would be attempted, because guiding the 3D-steering of a catheter is difficult to visualize on ultrasound in some regions, even when 3D ultrasound is used. Catheter ablation requires the catheter to be positioned exactly at the arrhythmic origin of an arrhythmia, requiring a highly detailed imaging technique.
In horses, 3D EAM of the right heart but also of the left heart has been performed to investigate normal depolarization in healthy horses. Access to the left heart was achieved by transseptal puncture, which means creating a small opening in the interatrial septum for catheter insertion into the left heart. This small opening spontaneously closes after the procedure. 3D mapping and RF ablation have been successfully used in horses to treat different supraventricular arrhythmias and accessory pathways. So far, only limited experience in a research setting is available with ablation in the ventricles. Indeed, delivering RF energy to the ventricular myocardium and especially the Purkinje system might briefly induce arrhythmias and thus includes a risk for development of ventricular fibrillation, which is always fatal in horses. Clinical cases with ventricular arrhythmias have not been treated with ablation yet. Currently, research is performed to develop a ventricular defibrillator for horses as this would allow to investigate the applicability of ventricular ablation.



An electrophysiology (EP) system during a standing EP study in a horse.

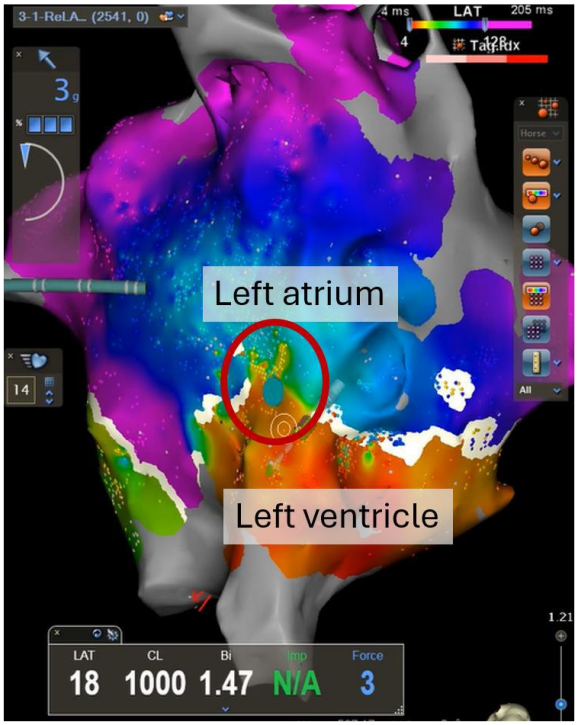
"Typical atrial tachycardia" (AT) in horses is a very rapid depolarization of the atria, usually around 180/min. It can be treated with quinidine sulphate or transvenous electrical cardioversion, similarly as atrial fibrillation (AF), but might be more difficult to convert and shows a higher recurrence rate compared to AF. In horses, AT is caused by a reentry mechanism in the myocardial sleeves of the caudal vena cava, just caudal to the fossa ovalis. This area shows a small region of conduction block and one or two areas with slow conduction, the necessary ingredients to create a perfect substrate for macro-reentry. Creating an ablation line at across the reentry circuit, interrupting the circling pathway, has been shown to be extremely efficient to terminate AT but also to avoid recurrence of this arrhythmia. AT is also known to be able to deteriorate into the more rapid and chaotic AF. When a horse is presented with AF, one cannot be sure whether or not AT was the initial cause. If AF originated from AT, ablation of the caudal vena cava might help prevent AT and thus AF.

RF ablation has also been used to treat 3 horses with a high burden of atrial premature depolarizations. These premature depolarizations originated from the right atrial free wall in all three horses. Ablation was performed in two horses, and succeeded in one horse. In the other horse, the myocardium showed a pronounced, relatively thick pectinate muscle near the arrhythmogenic area, which probably prevented the RF energy to reach sufficient depth into the myocardium to reach the focus. During ablation of the atrial free wall, one



Left lateral view on a coherent map of the right atrium. Coherent mapping is a module of the CARTO 3 system, integrating local activation time with conduction velocity vectors, thereby helping to identify areas of conduction slowing or block. Bold vectors indicate slow conduction and areas without conduction velocity vectors (zones of conduction block) appear as brown on the map. A clockwise reentry circuit in the caudomedial aspect of the right atrium rotates around a line of conduction block (brown line). A dorsal and ventral zone of slow conduction can be identified and are represented by the bold vectors. **B:** Surface ECG lead I, II, III during atrial tachycardia on top (yellow), coronary sinus electrograms (2 blue traces) and electrograms from the mapping catheter located at the area dorsal of the line of conduction block (last four traces). Electrograms from the mapping catheter show fractionated signals, which represent slow conduction. **C:** Surface ECG lead I, II, III on top (yellow), coronary sinus electrograms (2 blue traces) and electrograms from the mapping catheter at the line of conduction block (last four traces). Intracardiac recordings from the mapping catheter represent double potentials (highlighted by the yellow frame), which corresponds to the line of conduction block.

should be cautious in the region of the phrenic nerve, although the risk for damage to the nerve seems very small due to the thickness of the myocardium and pericardium.



Activation map of the left atrium and left ventricle shows a small 'breakthrough' from ventricle to atrium, through the annulus fibrosus (white line), indicating the accessory pathway location and the target for ablation.

RF ablation allowed to permanently restore normal sinus rhythm in two horses with an accessory pathway. One horse showed an accessory pathway with anterograde conduction, causing ventricular pre-excitation (conduction from atrium to ventricle). The accessory pathway was located in the right free wall. The other horse showed an accessory pathway with retrograde conduction (from ventricle to atrium) that caused tachycardia (orthodromic atrioventricular reentry tachycardia) but also bradycardia. This time the accessory pathway was located in the left heart, requiring transseptal puncture. The pathways were successfully treated by RF ablation. In human and small animal patients, ablation of left-sided accessory pathways is occasionally attempted from within the coronary sinus or great cardiac vein. However, because of the relatively large distance between the vein and the myocardium, and the fat surrounding it (unpublished data), this approach seems less likely to be effective. In humans and small animals, ablating the accessory pathway from the ventricular side can be more successful. In our horses with accessory pathways, ablation was performed from the atrial side to avoid the potential risk associated with ventricular ablation.

In human medicine, the most common indication for ablation is the treatment of atrial fibrillation. The goal of this procedure is to electrically isolate the myocardial sleeves of the pulmonary veins from the left atrium, as these myocardial sleeves are thought to be strongly related to atrial fibrillation pathophysiology as trigger and/or substrate. A technique for electrically isolating pulmonary veins in horses has been developed, which requires a transseptal puncture to access the left atrium. The technique has also been applied in horses with one or more AF recurrences after quinidine or transvenous electrical cardioversion treatment, but remains challenging. Catheter manoeuvrability in the left atrium is very complex. In addition, horses show very large pulmonary veins with substantial anatomical variation. Therefore, the procedure is currently still time-consuming and does not yet allow to isolate all pulmonary veins in one procedure. So far, only 7 horses with recurring AF were treated, of which 2 remained in sinus rhythm. It is difficult to determine the benefit of the ablation in this small number of cases. The treatment is supposed to decrease the amount of potential arrhythmogenic tissue.
However, in a horse with AF the exact contribution of the pulmonary veins to AF is unclear, and it remains unknown which pulmonary veins should be isolated to maximize the potential effect. Further optimization of the pulmonary vein ablation technique is needed. Also, further research is needed to determine the effect of caudal vena cava ablation in horses with AF, as this might be beneficial in those horses that present AT as the initial arrhythmia before it deteriorates into AF. Further studies into different ablation energy sources (pulsed field ablation, cryo-ablation) and different ablation catheter types to achieve deeper lesions and shorten procedure times, should be performed.

In conclusion, important advances are currently being made in equine cardiology, to obtain better quality standardized ECG recording, 12-lead recording and vectorcardiographic information. The multiple catheter recording is helpful for better localisation of the origin of an arrhythmia and can be applied in the standing horse. The 3D EAM is the ultimate tool to better characterize and understand normal cardiac depolarization and to obtain precise information regarding the location and mechanism of arrhythmias, allowing treatment by RF ablation in selected cases. These techniques allow a totally new, catheter-based approach for diagnosis and treatment of arrhythmias in horses.

ACVIM update

Eve Lo

Doxorubicin Cardiotoxicity: Cardiologist vs. Oncologist Perspective

Jackie M. Wypij, DVM, MS, DACVIM (Oncology) (she/her/hers)
Danielle Smith Laughlin, DVM, DACVIM (Cardiology) (she/her/hers)

Presentation Description / Summary:
The anthracycline drug doxorubicin has historically been one of the top chemotherapy choices for successful treatment of common canine cancers such as lymphoma & hemangiosarcoma. However, its effects on the heart can lead to severe cardiac toxicity including dilated cardiomyopathy and arrhythmias. This is often complicated by the effects of cancer on the heart itself. In addition, many canine breeds prone to cancer are unfortunately also at risk for heart disease. This creates both a unique challenge and opportunity in veterinary medicine that crosses the specialties of cardiology and oncology. The first part of this interactive lecture will provide a critical review of current literature of anthracycline-induced cardiotoxicity. The second part of the lecture will include a tandem case-based approach to optimizing successful treatment and patient care. Focus will be on cardiologist's perspective regarding underlying heart disease that could be worsened with doxorubicin treatment, early detection of doxorubicin cardiotoxicity, and cardiac medical management as well as oncologist's perspectives on modifying chemo protocols and alternative chemo options to balance effectiveness and patient safety.
Learning Objectives:
Describe types of underlying heart disease that could potentially be worsened with doxorubicin treatment.
Discuss cardiac medical management with clients.
List potential modifications and alternatives to standard doxorubicin chemo protocols.

Multimodality Imaging for Mitral Interventions

I-Jung Bernard Chi, DVM, MS, DACVIM (Cardiology)
North Grafton, MA, USA

THE MITRAL APPARATUS
The mitral valve (MV) is an intricate structure with complex biomechanical properties. Each component of the mitral apparatus can be affected by various acquired or congenital disease of the MV. The anatomy of the aortic (AML) and parietal mitral leaflets (PML) was first described by Carpentier. The AML and PML each have three subsegments (or scallops) which are coded numerically as the lateral (A1 and P1), middle (A2 and P2), and medial (A3 and P3) segments. Any of the scallops may experience myxomatous degeneration resulting in primary mitral regurgitation (MR) in degenerative mitral valve disease (DMVD). In congenital MV malformations such as mitral stenosis (MS), the normal architecture of the MV leaflets can be severely distorted that no subsegments can be identified. The subvalvular component of the MV consists of the chordae tendineae (CT) and papillary muscles. In cases with ruptured or anomalous CT, excessive (flailing) or restrictive (tethering) motion of the MV leaflets can also disrupt the closure mechanism of the MV. The mitral annulus (MA) is a critical component of the MV that consists of the aortomitral continuity, medial/lateral fibrous trigones, and the conjunction of the left atrial (LA) and left ventricular (LV) myocardium. It is known to be a functional structure in dogs and does not have an organic fibrous body. The temporal variations in mitral annular shape and size constitute mitral annular dynamics (MAD) which may be altered secondary to the underlying disease process.

Understanding Transient Myocardial Thickening in Cats

Jose Novo Matos, DVM, PhD, MSc, DECVIM (Cardiology), AFHEA
University of Cambridge, Cambridge, England, UK

INTRODUCTION

Transient myocardial thickening (TMT) is a transient form of myocardial disease in cats that closely resembles hypertrophic cardiomyopathy (HCM). Affected cats typically present with left-sided congestive heart failure (CHF), although, less commonly, TMT may also result in arterial thromboembolism (ATE).
This condition is characterized by left ventricular wall thickening on echocardiography (HCM phenotype) at initial presentation, and there are currently no definitive criteria to distinguish it from HCM at this stage. TMT is relatively uncommon; busy referral centers in Europe report 1–4 cases every two years,1 and in a large survey of American and European veterinary cardiologists, 182 out of 213 respondents indicated they had seen 10 or fewer TMT cases. Despite its rarity, recognizing TMT is crucial, as its prognosis is significantly better than that of cats with HCM stage C (CHF and/or ATE). Identifying TMT correctly can influence clinical decision-making, encouraging treatment in cases previously thought to have a poor prognosis and potentially preventing premature euthanasia.

Clinical Audit in the Veterinary Cardiovascular Society

Committee members:
Mike Martin, Geoff Culshaw, John Keen, Tobi Wagner, Luca Ferasin, Kieran Borgeat, Fabio Sarcinella, Katie Glyde.

Background to Clinical Audit

Clinical audit remains in its infancy in veterinary medicine, but is an emerging aspect of clinical governance (https://vetaudit.rcvsk.org/). Clinical papers in veterinary journals are dominated by research that documents new knowledge, often following randomisation of differing cohorts, with the aim of progressing and evolving novel methods or techniques. By contrast, clinical audit aims to measure standards of care or service set by that research and to monitor established techniques or processes to ensure they are working as expected. Research typically requires ethical approval whereas clinical audit does not [1].

Clinical audit has been in place for surgical and interventional cardiac procedures in human medicine for over 30 years. Since 1988, the British Cardiovascular Interventional Society (BCIS) has been collating outcome data for consultant cardiologists who perform percutaneous coronary intervention (PCI) in the UK [2]. Their aim was to create a registry of all PCI procedures to assess quality of care, drive improvements and provide a benchmark. The first publication of their findings was in 1990 [3]. In 2011 management of the BCIS registry was moved to the National Institute of Cardiovascular Outcomes Research (https://www.nicor.org.uk/about-nicor/) [4]. The results and reports from this audit (https://www.bcis.org.uk/public-information/public-reports/), as well as individual outcomes data, are viewable in the public domain. Outcome clinical audit monitors the success and complication rates of an established technique or procedure. If audit is used appropriately and efficiently, it can be an effective tool for improvement. Or, put more simply, audit helps to find out whether or not a method or process is attaining an established standard, with the potential to drive further improvements in outcomes.

As interventional procedures become more commonplace in veterinary cardiology, audit becomes an important tool to assess current clinical standards, or as a means of benchmarking for new centres and individuals embarking on interventional procedures.

Practical application of Clinical Audit

1. Understanding what audit is (versus research)

Source: chrome www.rqia.org.uk/RQIA/files/fe/fe8b6683-a3ea-428b-9e19-b1d1e05bcac1.pdf

2. How much data to record

There is a compromise in how much data to be collected. As ‘researchers’ we tend to collect excessive data, but this would make an audit onerous to complete, acquiring follow-up data is difficult, so there needs to be a focus on the bare essentials. The data collated needs to highlight two important questions: how successful is a procedure and what are the major complications.

3. Developing definitions of success and complications

Procedural Success to Discharge

This is a binary question yes (1) or no (0), hence the wording of the definition needs to suit a binary answer. The criteria need to be based on accepted ‘research’ and established expert opinion, but equally needs to be achievable without being excessively stringent. Additionally, ambiguous adjectives, such as optimal, appropriate, or acceptable, for example, need to be avoided. So, for example the definition of procedural success for PDA closure is: Successful procedure and closure of the duct with no more than trivial flow at discharge.

Complications

The main aim here is to record significant or major complications that necessitate an intervention, as opposed to minor complications (ie. insignificant consequences or requiring minimal intervention). Again, this is a binary question yes (1) or no (0). For example, in PDAs this might include: any complication resulting in a decision to abort the procedure, any complication that led to death in the peri-interventional period or in the following few days, dislodgement/embolisation of the device, ventricular fibrillation (VF) needing defibrillation, significant lameness, device infection or thrombus formation, rupture or tearing of a vessel resulting in

haemorrhage or the access vessel not being useable. But it might not include ventricular arrhythmias that have no clinical consequence, or require limited medication (eg. lidocaine).

Medium Term Outcome

The longer-term success of any intervention is key to understanding the success of that procedure, however obtaining follow-up once a patient has left the clinic, has always been a challenge. Thus, the timeline within which to obtain this data needs to be not excessively long. How this timeline may differ between procedures. For example a short time period of a few months might be sufficient to corelate with long term success in PDA occlusion, but for BVP on PS, as re-stenosis is a recognised problem, that would ideally require a much longer follow-up time period. So for example, the definition for PDAs would be: maintained successful closure, device in place and dog not in congestive heart failure and not needing diuresis, in the 3-12 month period, and without an unexplained sudden death, continuing heart failure or severe pulmonary hypertension. Again, this is a binary question yes (1) or no (0).

4. Other data

As 'researchers' it is easy to want to collect lots of other data. But the more that is asked for, the more onerous the clinical audit becomes and the inclination for participation is reduced, especially in the early years of establishing audit and recruiting and involving other centres. Any additional data needs to address the primary objectives: does this change how we define 'success' or record the 'complications'. There is certainly always room to add additional comments, for example to explain a complication that occurred.

References

[1] Wade DT. Ethics, audit, and research: all shades of grey. BMJ. 2005;330(7489):468.

[2] Hubner PJB. Cardiac interventional procedures in the United Kingdom during 1988. Br Heart J. 1990;64(1):36.

[3] Ludman PF. British Cardiovascular Intervention Society Registry for audit and quality assessment of percutaneous coronary interventions in the United Kingdom. Heart. 2011;97(16):1293–7.

[4] Rashid M, Ludman PF, Mamas MA. British Cardiovascular Intervention Society registry framework: a quality improvement initiative on behalf of the National Institute of Cardiovascular Outcomes Research (NICOR). Eur Heart J Quality of care & clinical outcomes. 2019; 5(4):292–7.

Additional References / Sources

Martin M et al (2022) Outcome clinical audit: Analyses of interventional closure of patent ductus arteriosus in dogs. JVC (2022) 43, 27-40.


HORSERACE BETTING
HBLB
LEVY BOARD


HeartVets

Echocardiographic Case Studies

John Bonagura

This presentation offers the audience a chance to consider short case vignettes that are focused on veterinary echocardiography. To keep the session moving the cases will be incomplete but should provide sufficient imaging to advance an assessment. Since the cases are "unknowns" the following notes are simply general considerations for echocardiographic interpretation and are based on a table that has been updated over the years (although it is still incomplete). Meeting participants interested in obtaining a PDF version of the presenter's slides can scan the QR code that will be displayed during the meeting or email the author at jdbonagu@ncsu.edu or john.bonagura@colostate.edu

The following table represents one approach for evaluating an echocardiogram.

Approach to Echocardiographic Interpretation
Examination Conditions – Information to report Indication(s): Clinical problem(s) or reason(s) for echocardiography ECG: Normal, arrhythmia or conduction disturbance Sedation if administered Drugs: Cardioactive drugs the patient was receiving Technical aspects: Overall quality of the echocardiographic study › Comprehensive transthoracic echocardiogram (TTE) or limited/focused exam › Artifacts of ultrasound imaging › Complete or incomplete image acquisitions › Patient factors (panting, uncooperative, etc.)
Congestive heart failure (CHF) is suspected – Findings supporting the diagnosis Serous cavity effusions › Pleural effusion › Ascites › Pericardial effusion (with structural heart disease) B-lines: Number of positive lung fields on thoracic ultrasound (supporting left-sided CHF) Structural heart disease: Underlying cause of CHF Left or right atrial dilation: Present in most cases except in peracute CHF or post-diuretic therapy Caudal vena cava & hepatic veins: Increased size or reduced collapsibility (supporting right-sided CHF) *Doppler exam*: Spectral/Tissue Doppler findings supporting increased left or right atrial pressures
Pericardial effusion or cardiac-related neoplasia are identified – Key Considerations *Appearance / echogenicity* of any identified effusion › Mixed echoic fluid: Rule out (r/o) active/recent hemorrhage or inflammatory effusion › Organizing thrombus: r/o cardiac rupture or recent tumor-related hemorrhage
Volume of pericardial effusion (estimated) › Scant: Systolic separation of the ventricular walls from the pericardium › Small to moderate: Without echocardiographic or clinical signs of cardiac tamponade › Small to moderate effusions with signs of hemodynamic compromise (tamponade) Acute hemorrhage from a cardiac or heart base tumor Pre-existing cardiomegaly limiting pericardial stretch (e.g., rupture of the left atrium) › Moderate to large volume effusion Frequently associated with signs of acute or chronic cardiac tamponade Large effusion relative to overall cardiac size (subjective) Exuberant cardiac motion ("swinging") within the effusion
Echocardiographic findings of *acute or chronic cardiac tamponade*? › *Clinical signs*: Hypotension, elevated systemic venous pressures, right-sided CHF › Ascites or pleural effusion › Diastolic collapse of the right ventricle (RV)

- Prolonged inversion of the lateral wall of the right atrium (RA)

- Pseudohypertrophy of the ventricular chamber walls
- Dilation/reduced collapsibility of the caudal or cranial vena cava
- Distended hepatic veins

Pericardial effusion associated with s*tructural heart disease* and atrial dilation?
- Dilation of the left atrium (in cats): suggests pericardial effusion secondary to CHF
- Dilation of the right atrium or right heart disease: potentially an effusion from right heart failure
- Advanced mitral valve disease: consider left atrial tear

Is a mass lesion identified – cardiac, heart base, or within the pericardial space?
- Right atrial mass in a dog: r/o hemangiosarcoma > other tumors
- Heart base mass contiguous with aortic root: r/o chemodectoma, ectopic thyroid carcinoma, neuroendocrine tumor
- Heart base or intracardiac mass: r/o obstruction of pulmonary venous return
- Heart base mass: r/o obstruction of a pulmonary artery branch (usually right)
- Effusion with *myocardial thickening involving the right ventricular wall*: r/o lymphosarcoma
- *Intrapericardial mass*: r/o tumor from cardiac muscle, mesothelioma, granuloma, thrombus
- Cystic structure within pericardial space: r/o intrapericardial cyst
- Intrapericardial omental fat, liver, or bowel: r/o peritoneopericardial diaphragmatic hernia

Is an *Intraluminal mass lesion* identified?
- r/o tumor, thrombus, or granuloma
- Organized left atrial appendage mass with spontaneous echo contrast (cat): likely thrombosis
- Right atrial mass obstructing systemic venous return or causing acquired tricuspid stenosis: likely a neoplasm (extensive differential diagnosis including hemangiosarcoma, neuroendocrine tumors, myxosarcoma/myxoma, rhabdomyosarcoma).
- Mass on the mitral valve (rare): r/o myxosarcoma/myxoma
- Mass on the tricuspid valve: r/o tumor or thrombus (especially post-catheterization or pacing)

Are there signs of *effusive-constrictive pericardial disease*?
- Thickened parietal pericardium
- Atrial dilation and signs of right-sided CHF (see above)
- Restricted diastolic motion of the RV wall (also on M-mode)
- Diastolic septal bounce (also on M-mode)
- Loculated pericardial effusion (also r/o mesothelioma or prior surgery)
- *Doppler*: spectral and tissue Doppler features suggestive of constriction

<u>Atria – Key Considerations</u>

Four cardiac chambers are identified
- Venoatrial and atrioventricular connections
- Mitral and tricuspid valve morphology and motion
- Relative sizes of the four cardiac chambers on RPLAx and left apical 4-chamber views
- Size of the left atrium relative to aorta at the aortic valvular level on RPSAx view

Atrial chamber size: Measurements – decreased, normal, or increased
- *Decreased right and left atrial size*: r/o hypovolemia or cardiac compression
- *Decreased left atrial size*: r/o right to left shunt or pulmonary arterial hypertension
- *Increased right or left atrial size* – multiple rule outs

Shunts: Right atrial enlargement from ASD, AVSD, anomalous pulmonary venous drainage, or systemic arteriovenous fistula

Shunts: Left atrial dilation from PDA, VSD, AVSD, AVSD with atrial septal deviation (double outlet right atrium), systemic-to-pulmonary shunts/fistulas

Atrioventricular valve disease: valvular or supravalvular stenosis, valvular regurgitation

Cardiomyopathies: dilated, hypertrophic, restrictive, and others types

Heart failure: diastolic and systolic heart failure

Atrial myopathies (e.g., silent atrium)

Arrhythmia related: chronic bradycardia or tachycardia

Circulatory volume overloads (iatrogenic) and volume retentive states (RA > LA)

High output states: anemia (RA > LA), thyrotoxicosis (multifactorial)
- Divided atrial chamber: cor triatriatum dexter, cor triatriatum sinister, AVSD with double outlet RA

Interatrial septum (IAS) – defects
- Atrial septal defects: secundum, primum/AVSD, sinus venosus defects

- Absent IAS (common atrium)
- *Doppler & saline contrast* echocardiography: right-to-left or left-to-right shunting

Interatrial septum – appearance
- Leftward deviation of IAS with AVSD and supra-mitral stenosis: "double-outlet right atrium"
- Leftward bowing of IAS from increased RA pressures (r/o pulmonary hypertension & cor pulmonale)
- Rightward deviation of IAS from increased LA pressures (often due to mitral regurgitation)

Atrial function
- M-mode/2D indices measuring LA fractional shortening and changes in LA area or LA volume
- *Doppler*: transmitral and pulmonary venous flow patterns; tissue Doppler imaging
- Atrial strain
- See also: "Congestive heart failure" and "Ventricles"

Pulmonary veins
- Three normal venous confluences entering the LA of dogs and cats: right cranial/middle lung lobes; left & right caudal and accessory lung lobes; left cranial lung lobes
- Diameter of right cranial/middle pulmonary veins > adjacent right pulmonary artery:
  r/o increased LA pressure or chronic mitral regurgitation
- Increased diameter of pulmonary veins with dilation of adjacent pulmonary artery:
  r/o left-to-right shunting or left heart failure with pulmonary hypertension
- Diameter of right cranial/middle pulmonary veins < adjacent right pulmonary artery:
  r/o pulmonary arterial hypertension
- Abnormal pulmonary venous entry into RA: r/o sinus venosus atrial septal defect

<u>Ventricles – Key Considerations</u>

Four cardiac chambers are identified
- Atrioventricular and ventricular to arterial connections
- Relative sizes of the four chambers on RPLAx and left apical 4-chamber views
- Shape and positions of the right ventricle (RV) and left ventricle (LV)

Ventricular wall thickness – Measurements: decreased , normal, or increased?
- Decreased from segmental or generalized wall thinning

Ischemia/infarction (presumed) in feline cardiomyopathies
Absolute or relative (to chamber size) wall thinning in dilated cardiomyopathy phenotypes
Fibrofatty replacement (e.g., arrhythmogenic cardiomyopathy)
Tachycardia-induced cardiomyopathy (variable)
- Increased from focal, segmental or generalized wall thickening

Pseudohypertrophy of ventricle (volume depletion, cardiac tamponade)
Concentric hypertrophy from pressure overload (aortic stenosis, systemic hypertension, pulmonary stenosis, Type I pulmonary arterial hypertension)
Mixed hypertrophy (wall thickening with chamber dilation): chronic pressure overload with myocardial failure from aortic stenosis or systemic hypertension, combined aortic stenosis with aortic regurgitation (AR), acquired pulmonary hypertension, combined pulmonary stenosis with pulmonary regurgitation, semilunar valve stenosis with concurrent atrioventricular valve regurgitation
Proximal chamber hypertrophy of the RV: double-chamber right ventricle
Hypertrophic cardiomyopathy: focal, segmental or generalized wall thickening
Hyperthyroid heart disease
Myocardial infiltration: neoplasia, myocarditis (note: amyloidosis is rare in dogs/cats)
Noncompaction of the myocardium
Idiopathic basal septal thickening (discrete upper septal thickening)

Ventricular chamber size – Measurements: decreased , normal, or increased for body weight/breed
- *Decreased*: see decreased atrial size

See causes of decreased atrial size
Hypoplastic ventricle (often from atrioventricular valve stenosis)
interventricular septal shifting from contralateral chamber enlargement (ventricular interdependence)
- *Increased* (dilation or eccentric hypertrophy of ventricle; also see mixed hypertrophy, above):

Shunts – ASD, systemic arterio-venous fistula: RV enlargement
Shunts – PDA, VSD, systemic to pulmonary shunts/fistulas: LV enlargement
Atrioventricular valve regurgitation: mitral (LV), tricuspid (RV)

Semilunar valve regurgitation: aortic (LV), pulmonic (RV)
Cardiomyopathies: dilated, end-stage hypertrophic, arrhythmogenic RV, non-specific, doxorubicin cardiotoxicity
Myocarditis leading to dilated cardiomyopathy phenotype
Arrhythmias: tachycardia-induced cardiomyopathy; chronic bradycardia
High-output states (e.g. moderate-severe anemia), thyrotoxicosis
Interventricular septum (IVS)
- *Defects in the IVS*

Ventricular septal defect defects: perimembranous, subarterial, inlet (AVSD), muscular
Related lesions: Ventricular septal aneurysm, aortic leaflet prolapse into VSD (with AR)
Doppler: demonstration of shunt; velocity relates to size of defect and RV & PA pressures
- *Ventricular septal thickness*

Discrete upper septal thickening or hypertrophy (DUST, DISH) in older cats
IVS hypertrophy – isolated or related to generalized LV concentric hypertrophy
- *Ventricular septal curvature*

Large radius of septal curvature – "flattened septum" due to right ventricular disease
- *Abnormal IVS motion*

Paradoxical (rightward systolic) septal motion: right ventricular volume overload
Flat (systolic) septal motion: right ventricular pressure overload
Hyperdynamic systolic septal excursion: left ventricular volume overload
Hypokinetic septal motion: myocardial thinning or ischemia/infarction (cats > dogs)
Dyssynchrony with LV posterior wall: multiple associations including arrhythmias and conduction disorders (dogs > cats)
Systolic Ventricular Function
- Subjective assessment of ventricular wall motion across visualized segments

Mitral annular motion, LV radial shortening (short-axis), length-area changes (long-axis)
Tricuspid annular motion (TAPSE), RV length-area changes (optimized RV inflow view)
- Quantitative left ventricular systolic function: see Table 6 and Supplemental Table A5
- Quantitative right ventricular systolic function: see Table 6 and Supplemental Table A5

Ventricular diastolic function and estimates of "filling" pressures"– Grades (by Doppler)
- Subjective interpretation of patterns mitral valve & tissue Doppler velocity patterns:

Normal | Relaxation abnormality | Pseudonormal | Restrictive (± reversible)
- Quantitative estimates of diastolic function and veno-atrial pressures using Doppler imaging

Great vessels
Identification of aorta and pulmonary artery
- Both great vessels are present with normal connections to their respective ventricles
- Congenital defects involving great arteries

Stenosis, hypoplasia, absence of the right or left pulmonary artery
Pulmonary atresia, truncus arteriosus, double-outlet right ventricle
Hypoplastic aorta / left heart (rare in dogs and cats)
Shunting at the level of the great vessels
- Patent ductus arteriosus
- Aortopulmonary window
- Coronary to pulmonary artery fistula
- Anomalous pulmonary origin of a coronary artery
- Systemic-to-pulmonary shunts including fistulas and major aortopulmonary collateral arteries
- *Doppler*: identification of (typically) high-velocity, continuous (left-to-right) blood flow
- *Doppler*: identification of bidirectional or right-to-left shunting (Eisenmenger's pathophysiology)

Coronary arteries
- Ostia usually identified from RPSAx image at the aortic valve/left atrial, the RPLAx optimized for the LVOT (left coronary artery), and the left-cranial RV inflow-outflow tract image planes
- Dilated or enlarged coronary artery: potentially anomalous or secondary to ventricular hypertrophy
- Absent coronary artery ostium: potentially single-origin left or right coronary artery

Abnormalities of the aorta or the main pulmonary artery (pulmonary trunk)
- *Aortic dilation*: post-stenotic dilation, primary aortopathy, systemic hypertension, connective tissue disease (breed-related), dissection of the aorta

- *Abnormal septal-to-aortic angle* (association with subaortic stenosis and mature cats with discrete upper septal hypertrophy or thickening)
- *Pulmonary trunk dilation*: post-stenotic dilation, left-to-right shunt, pulmonary hypertension, idiopathic dilation of the pulmonary trunk
- *Intraluminal abnormalities*:

Parasitic infection (D. immitis in the pulmonary arteries)
Thrombus (more common in PA)
Intraluminal mass (aorta or PA)
Extraluminal compression (usually PA or right PA)
Intimal aortic dissection (from systemic hypertension or connective tissue disease)
Intimal pulmonary artery dissection (spontaneous with PDA or iatrogenic from catheterization)
Systemic hypertension

- Remodeling of the LV, aortic dilation, high-velocity mitral or aortic regurgitation (Doppler)

Pulmonary hypertension

- Remodeling of the RV, pulmonary artery dilation, high-velocity tricuspid or pulmonary regurgitation

IMOTEK

imv imaging

INFINITI®

30 Years of VCS and counting.

Malcolm Cobb

My invitation to speak this year was bittersweet, it went something like this:
"Would you like to speak at the 50th anniversary meeting in November?"
"It would be an honour thank you, I have some material that is new and should interest the members."
"Actually, we thought you might talk about what life was like in the old days? You know, before echocardiography and stuff."
So the chronology of my 30 years in the Society will go something like this:

- Cardiology without echocardiography
- Cardiological "firsts", or maybe close seconds, mentors and heroes
  - Tangling with anaesthesia
- Publication in The Lancet
- Comments on prognostication
- Research into cardiomyopathy – some new data on wolfhounds
- Research into cardiomyopathy – antibodies and proteins – mentors and heroes
- Skeletal muscle for cardiac assistance
  - I've done this surgery, can you keep the dog alive for me
- Early experiences with cardiopulmonary by-pass
  - I've done this surgery, can you keep the dog alive for me
- Cardiological road trips
- Time as the VCS secretary – the 25 year celebration!
- Starting a new Vet School – heroes and mentors
- Starting a new Vet School – curriculum and clinical teaching
  - The gold standard discussion
- Undergraduate projects – data on compliance with treatment in the management of cardiac failure
- Undergraduate projects – safety of drugs used to treat cardiac failure
- The community that is VCS and its care for the elderly – 50 years and counting

Francesca Edgerton

Does increasing left atrial enlargement or congestive heart failure in dogs with mitral valve disease increase the likelihood of coughing?

Mark Rishniw 1, 2, Michele Borgarelli 3, Giulio Menciotti 3, Luca Ferasin 4
1 Cornell University , Ithaca, United States
2 Veterinary Information Network , Davis, United States
3 VA-MD College of Veterinary Medicine , Blacksburg, United States
4 Specialist Veterinary Cardiology Consultancy Specialist Veterinary Cardiology Consultancy, Alton, United Kingdom

Most clinicians consider coughing a feature of congestive heart failure (CHF) in dogs with myxomatous mitral valve disease (MMVD), because coughing often resolves or improves with treatment; however, loop diuretics act as bronchodilators and antitussives, reducing coughing from all causes in most dogs. Therefore, we examined data collected in the DELAY study and a second, earlier study, in a cross-sectional design to determine if increasing left atrial size with subclinical disease, or the onset of CHF, resulted in changes in coughing probability in dogs with MMVD.
We included every examination at which coughing status was recorded in the DELAY dataset as a separate datapoint; the second study included only a single datapoint. First, we calculated the proportions of dogs recorded as coughing within each of 4 levels of left atrial (LA) size: normal, mildly, moderately and severely enlarged without regards to CHF status for each study separately. We then separated out dogs presenting with CHF in both studies and examined the proportions of coughing dogs with CHF separately from those with subclinical disease. Next, we excluded dogs with only one visit recorded in the DELAY study. For the remaining dogs, we retained only the earliest instance of coughing recorded for that dog – effectively a “new” cough. All subsequent visits were coded as “not coughing”, regardless of the coughing status for that visit. We examined the proportions of dogs developing a new cough at each level of LA enlargement, and separately for dogs that developed CHF. Finally, we compared ages of dogs within each level of LA size.
We included 88 (and 66) small-breed older dogs, undergoing a total of 298 (and 276) examinations, respectively, from the DELAY study, and 206 dogs from the earlier study. Ages between different levels of LA size did not differ (p=0.55). Dogs with severe LA enlargement had a higher probability of coughing (P=0.0004). Dogs that developed CHF had a higher probability of coughing (P=0.005). However, when examined as “new cough”, we observed a difference between groups (p=0.013) on the omnibus test, but not on post-hoc pairwise comparisons. When dogs that developed CHF were examined as a subgroup, the proportion of dogs developing a new cough differed (P=0.049) on the omnibus test, but not on post-hoc pairwise comparisons. Marked left atrial enlargement in dogs with MMVD increases the risk of developing a cough, which might be further increased with the development of CHF.

Feasibility of a new minimally invasive implantable cardiac monitor for remote heart rhythm monitoring in dogs and a cat

Anne Watering 1, 2, Caroline Melis 2, Niek Beijerink 2
1 University of Utrecht Dept of Clinical Sciences, Fac of Veterinary Medicine , Utrecht, Netherlands
2 Veterinaire Specialisten , Vught, Netherlands

Long-term cardiac rhythm monitoring is essential for both diagnosing arrhythmias and evaluating treatment efficacy through repeated rhythm assessments. Remote monitoring enables continuous rhythm surveillance without the need for in-clinic visits. The aim of this prospective case series was therefore to assess the feasibility of a novel insertable cardiac monitor (ICM), implanted without general anesthesia, and whether it provides usable data for clinical evaluation.
The ICM system provides a cutting tool and a single-piece insertion device for implantation of the miniature monitor. The procedure was performed under local anesthesia. An incision was created at the left parasternal region, at the level of the heart base, through which the device was inserted. Data were transmitted daily

through a wireless connection to a cloud-based data management system. Additionally, owners could use a synced mobile app to record symptoms.
A total of 7 implantation procedures were successfully performed for diagnostic purposes in 6 dogs and 1 cat with client informed consent. Primary indications were recurrent unexplained weakness, transient loss of conscience (TLOC) episodes, as well as monitoring of known arrhythmias. No perioperative or postoperative antibiotics were administered to any of the animals. The procedure went uneventful and was completed in under 6 minute in all animals. Initial programming was adjusted post-implantation by changing the threshold settings for arrythmia detection. Bradycardia was set at 30 bpm for ≥30 seconds, tachycardia at 220 bpm for ≥60 seconds in all animals. An event was automatically generated and transmitted whenever the detected rhythm exceeded the programmed detection thresholds.
In all 7 patients, the ICM provided decisive information guiding further management. Recorded events were sinus arrhythmia, supraventricular tachycardia, isolated premature ventricular beats, and both monomorphic and polymorphic ventricular tachycardias. Manual activation during an episode of TLOC revealed abrupt bradycardia, a brief escape rhythm, transient tachycardia, and spontaneous recovery. Limitations of the ICM included misclassification due to human threshold settings, where sinus tachycardia above 220 bpm and physiologic pauses longer than 4.5 seconds were incorrectly identified as pathological. The low tachycardia threshold resulted in excessive data storage, while inaccurate atrial fibrillation (AF) detection from unrecognized P waves reduced the reliability of AF burden estimates. Occasional undersensing was also noted. These findings demonstrate that the ICM enabled anesthesia-free implantation and remote monitoring in dogs and a cat, providing clinically relevant rhythm data such as tachograms, mean ventricular rates, and multi-day histograms that supported pattern recognition and informed treatment decisions.

Quality of life assessment in dogs with atrial fibrillation: the optimal rate control in dogs with atrial fibrillation 4 study

Alexander Brown 1, Christopher Linney 1, Brigite Pedro 2, Antonia Mavropoulou 3, Joao Neves 2, Joanna Dukes- McEwan 4, Anna Gelzer 5
1 Paragon Veterinary Referrals , Wakefield, United Kingdom
2 Virtual Veterinary Specialists Ltd , Middlesex, United Kingdom
3 Plakentia Veterinary Clinic , Athens, Greece
4 Small Animal Teaching Hospital, Department of Small Animal Clinical Science , University of Liverpool, Leahurst Campus, United Kingdom
5 Department of Clinical Studies and Advanced Medicine , University of Pennsylvania, Philadelphia, United States

In humans with atrial fibrillation (AF), heart rate control has been shown to improve quality of life (QoL) as well as longevity. Recent studies in veterinary medicine have shown that mean 24-hour heart rate <125 bpm (rate control, RC) improves survival outcomes in dogs with AF. No studies in veterinary medicine have yet examined the impact of RC on QoL in dogs with AF.
The aim of this study was to evaluate the effect of RC on patient and owner QoL for dogs with AF.
60 client-owned dogs with a diagnosis of AF were recruited across three veterinary cardiology centres. Holterderived 24-hour mean heart rate (meanHR) and echocardiographic and biomarker variables were analysed prospectively and grouped into RC and non-RC (NRC). Owner-reported questionnaires adapted for canine AF were used to evaluate patient and owner QoL at four timepoints: admission (baseline), RC visit (VisitRC), 6 months following RC visit (Visit6m), and 12 months following RC visit (Visit12m).
Patient QoL scores at VisitRC and Visit6m were significantly improved from baseline (P<0.001 and P=0.011 respectively). Patient QoL scores at Visit12m were also improved from baseline, although this did not reach statistical significance (P=0.057). At VisitRC, no significant difference in patient or owner QoL scores was observed between RC and NRC groups. Owner QoL scores were not significantly different between visits (baseline and VisitRC P=0.401, baseline and Visit6m P=0.556, baseline and Visit12m P=0.502, VisitRC and Visit6m P=0.510). The diagnosis of lone AF was associated with significantly higher patient and owner QoL at baseline (P=0.02 and P=0.08 respectively) and VisitRC (P=0.024 and P=0.044 respectively) compared to other cardiac diseases.
Stabilisation of dogs with medical therapy significantly improves QoL in dogs with AF. Despite improved survival with optimised rate control, this study did not identify any significant improvement in QoL scores in dogs with optimised rate control.

Anna Jakubczak

A modified mitral insufficiency echocardiographic score differentiates severities of subclinical mitral valve disease

Mark Rishniw 1, 2, Michele Borgarelli 3, Giulio Menciotti 3
1 Cornell University , Ithaca, United States
2 Veterinary Information Network , Davis, United States
3 VA-MD College of Veterinary Medicine , Blacksburg, United States

The ACVIM classification of subclinical mitral valve disease (MMVD) fails to differentiate levels of subclinical disease severity, limiting its utility. The Mitral INsufficiency Echocardiographic (MINE), attempted to differentiate mitral valve disease into several categories of severity, but included dogs with congestive heart failure, and the first two levels failed to provide clinically useful information. A revised MINE score (MINE2) reduced the number of variables, but still provided classifications that might be less-than-ideal for classifying subclinical MMVD. Therefore, we created a modified MINE score: the MINE ReVised Analysis (MINERVA) score to see if it provided more useful information about outcomes for subclinical MMVD, based on left atrial size, left ventricular size and mitral early inflow velocity.
Data from two previous studies (DELAY and an earlier retrospective study) were analyzed, based on the initial ACVIM staging and using the MINERVA scheme. MINERVA modifies the MINE score by changing the cut-offs for each category, to provide a more even distribution of integers per category, and removes the “end-stage” category (“mild” = 3-6, “moderate” = 7-9, “severe” = 10-12). We tested the validity of our scheme using the arguments that almost all dogs considered Stage B1 should be classified as “mild”, and most dogs with CHF should be classified as “severe” or “moderate”.
We included 268 (177 retrospective and 90 DELAY study) small-breed older dogs, undergoing a total of 314 examinations; at 141 visits, dogs were classified as ACVIM Stage B1, and ACVIM Stage B2 at 173 visits. Using the MINERVA score, dogs were classified as “mild” (n=206), “moderate” (n=81) and “severe” (n=27). 136/141 B1 dogs were classified as “mild” and 5 as “moderate”, B2 dogs were classified as “mild” (n=70), “moderate (n=76) and “severe” (n=27). 19/29 dogs with CHF scored ≥9 on the MINERVA score. All-cause median survival (or time to CHF) in dogs decreased with increasing scores (“mild”:1764 days, “moderate”:743 days, “severe”:287 days, $p<0.001$). For B2 dogs, median survivals were similar (only “mild” had a shorter survival at 1156 days). B1 dogs classified as “mild” had slightly longer survivals than B2 dogs classified as “mild”. When evaluated by MINERVA score, dogs scoring between 3 and 6 did not differ in median survivals, which then decreased incrementally with increasing scores.
The MINERVA classification scheme appears to improve on the MINE score, using the same echocardiographic variables as MINE2, but with biologically and clinically more useful outcome estimates.

Prognostic utility of mitral regurgitant fraction and doppler estimation of mitral regurgitation severity in dogs with myxomatous mitral valve

Gavin McAulay 1, 2, Mark Rishniw 3
1 Brighton Veterinary Referrals, , Brighton, BN1 8QR, , United Kingdom
2 North Downs Specialist Referrals, , Bletchingley RH1 4QP , United Kingdom
3 Cornell University, 930 Campus Road, , Itheca, New York 14853 , United States

Dogs with myxomatous mitral valve disease (MMVD) are typically staged based on cardiac chamber remodelling. The severity of mitral regurgitation precedes cardiac remodelling therefore integration of quantified mitral regurgitation might assist prognostication. The aim of this study was to report the effectiveness of mitral regurgitant fraction (MR%) and the Doppler derived estimate of mitral regurgitant severity, left ventricular early inflow outflow index (LVEIO), in predicting cardiac death.
This was a single centre retrospective observational study of dogs diagnosed with MMVD between May 2020 and May 2023 with follow up to December 2024. Mitral regurgitant fraction was calculated by subtracting forward stroke volume from total stroke volume. Left ventricular volumes were calculated using Simpsons method of discs from a right parasternal long axis view. The velocity-time integral (VTI) of aortic flow was measured from the left apical 5-chamber view and aortic outflow diameter measured between the maximally open aortic leaflets at mid systole in a right parasternal long axis view. LVEIO was calculated using the ratio of

early mitral inflow velocity to aortic velocity time integral. MR% and LVEIO were grouped into four bands of severity based on previous work. Outcome was performed by review of clinical histories, questionnaire and telephone interview with referring clinics. Cardiac death was defined as death without preceding signs, following tachypnoea or euthanasia attributed to heart disease. Dogs were censored on the date of last assessment, mitral valve repair or non-cardiac death. Survival was analysed using Kaplan-Meier curves and log rank tests. ROC analysis and Youdens index were used to determine the best cut-off to predict cardiac death. Median follow up time of 275 dogs was 603 days. Cardiac death was reported in 92 dogs (33.5%) with 183 (66.5%) dogs censored including 59 dogs (21.5%) with non-cardiac death. Median survival was different between severity bands: MR%: =>85% 105 days (95%CI 31-196); =>75%<85%, 381 days (95%CI 179-711); =>50%<75%, 1365 days (95%CI 992-undefined), <50% undefined. p=0.0001. Median survival LVEIO was; >25, 89 days (95%CI 51-196); =>17.5<25, 381 days (95%CI 136-638); =>10<17.5, 1365 days (95%CI 957-undefined); <10 undefined; p=0.0001. Both tests showed excellent predictive capability with best cut offs for cardiac death of MR% >67.5% (AUC 0.8487, 95%CI 0.7982-0.8992, p<0.0001); LVEIO >11.85 (AUC 0.8367, 95%CI 0.7828-0.8906; p<0.0001).
Mitral regurgitation quantified by MR% or LVEIO is predictive of cardiac death and may complement standard echocardiographic measurements.

SPACELABS HEALTHCARE

TIMELESS™ VETERINARY SYSTEMS

VetPlus

A Global Leader in Veterinary Nutraceuticals

QMS QUALITY MEDICAL SERVICES VETERINARY

Laura Korenchy

Contrast-enhanced echocardiographic assessment of left ventricular wall measurements in cats

Jose Novo Matos 1, Isabella Harding 2, Catheryn Partington 3
1University of Cambridge Veterinary Medicine, Cambridge, United Kingdom
2University of Cambridge University of Cambridge , Cambridge, United Kingdom
3University of Cambridge , Cambridge, United Kingdom

This study compared standard echocardiography (S-ECHO) and contrast-enhanced echocardiography (C-ECHO) for measuring left ventricular wall thickness (LVWT) in cats, which is crucial for diagnosing hypertrophic cardiomyopathy (HCM). Forty-three cats were examined, and results showed that S-ECHO consistently overestimated LVWT compared to C-ECHO. C-ECHO produced smaller wall thickness and larger cavity dimensions, with significantly higher measurement reliability and lower variability between observers. Agreement between the two methods for diagnosing HCM was only moderate. Overall, C-ECHO provided more accurate and consistent measurements by better distinguishing the compact myocardium from intracavitary structures, suggesting it could improve diagnostic accuracy in feline HCM assessment.

BEATING CANCER AT THE HEART: RADIOTHERAPY INNOVATIONS IN VETERINARY CARDIAC ONCOLOGY

James Elliott, BVM&S CertSAM DipECVIM-CA (Oncology & Radiation Oncology) DACVR (Radiation Oncology) MRCVS Southfields Veterinary Specialists (SFVS), United Kingdom

Recent advances in veterinary oncology have improved the treatment of canine heart-base tumours, especially using radiotherapy. Techniques such as conventional fractionated radiotherapy and stereotactic body radiation therapy (SBRT) have shown good tumour control, minimal side effects, and excellent quality of life. SBRT, a highly focused method delivering high radiation doses in just a few sessions, has proven effective and precise while minimizing damage to nearby organs like the heart and lungs. However, potential risks such as arrhythmias and cardiac toxicity remain concerns due to the high dose per session. Ongoing studies at SFVS are investigating tumour motion, treatment precision, and the safety of modified SBRT protocols to reduce toxicity. Supportive therapies, including ACE inhibitors, may further limit cardiac and lung side effects, and future research aims to refine these treatments and assess long-term outcomes.

TARGETING TUMOURS WITH PRECISION: FOCUSSED RADIATION, CHEMOTHERAPY AND DRUG THERAPIES FOR CARDIAC ONCOLOGY IN PETS

James Elliott, BVM&S CertSAM DipECVIM-CA (Oncology & Radiation Oncology) DACVR (Radiation Oncology) MRCVS Southfields Veterinary Specialists (SFVS), United Kingdom

Managing cardiac tumors in veterinary patients remains challenging due to their complex location and risk of severe complications. However, chemotherapy and targeted drug therapies are increasingly improving outcomes. Toceranib phosphate (Palladia), a tyrosine kinase inhibitor, has shown encouraging results in dogs with heart-base tumors, providing stable disease and median survival times of 16–27 months, with mostly manageable gastrointestinal side effects. Doxorubicin-based chemotherapy can also extend survival in right atrial tumors, while combinations of radiotherapy, vinblastine, and propranolol show emerging promise. Overall, integrating systemic therapies like toceranib and doxorubicin with localized treatments such as radiotherapy offers a promising, multidisciplinary approach to improving prognosis in veterinary cardiac oncology.

Management of Acute CHF in Cats: Quick Improvement Means Soonest Home

Rebecca Stepien

Congestive heart failure (CHF) is a common complication of feline cardiac disease. In a large study of cats with preclinical hypertrophic cardiomyopathy (HCM) and hypertrophic obstructive cardiomyopathy (HOCM), 30% of cats with HCM or HOCM eventually experienced CHF, arterial thromboembolism, or both.[1] Cats often manifest CHF as pulmonary edema and pleural effusion (PLE) but ascites and physical or biochemical evidence of decreased cardiac output may also be observed.[2,3] Systolic or diastolic dysfunction may predominate in various feline cardiomyopathy (CM) phenotypes, but it is increasingly acknowledged that most CMs likely have elements of both.[4,5] Optimal therapy of acute CHF in cardiomyopathic cats remains unknown, but centesis of cavitary effusions, oxygen supplementation and loop diuretic therapy are frequently recommended.[6,7] Several retrospective reports regarding various therapies in cats and dogs[2,8,9] have been published.

Identification of CHF as cause of respiratory distress

Careful physical examination of cats in respiratory distress (RD) is challenging and involves gathering key information efficiently without unduly distressing the patient. A few key findings on the physical that identify CHF in a cat with RD include detection of a gallop heart sound, rectal temperature < 37. 5 C., tachycardia and increased respiratory rate. Taken as single findings, the gallop and hypothermia are more reliable than the tachycardia or increased respiratory rate, but in conjunction with each other, and with other clinical findings suggestive of heart disease (distended jugular veins, arrhythmia, heart murmur), CHF can be strongly suspected based on physical findings.[10]

Cage-side assessment of NT-proBNP continues to be a helpful adjunct in the differentiation of cardiac and respiratory etiologies of RD. In the case of a dyspneic cat, a normal finding on a cageside NTproBNP test is a strong indicator that heart disease is not the cause of the RD, but other methods of assessment are better positive indicators.[10,11] Thoracic point-of-care ultrasound (POCUS) has quickly become a mainstay of assessment of fragile atients in respiratory distress, delivering information about the thoracic cavity (e.g. pleural and pericardial effusions, pneumothorax), the status of the lung parenchyma (e.g. dry or wet) and the heart, particularly the size of the left atrium. In cats with RD, findings of left atrial enlargement in conjunction with evidence of pulmonary infiltrates or thoracic effusions are highly suggestive of CHF, and critical pleural effusions can be removed using POCUS guidance. Immediate triage based on physical findings and thoracic POCUS will identify the most critical issues (hypoxia, arrhythmias) that might need to be addressed immediately.

Assessment of the patient

Once CHF is strongly suspected based on triage and acute supportive therapy (usually oxygen, centesis if needed and parenteral furosemide) has been delivered, evaluation of the patient's condition can continue. Obtaining additional information on the patient will be important, but only if the patient is alive; stabilizing the fragile patient always takes priority over additional evaluative testing. Use of anxiolytic agents that have minimal cardiovascular effects (e.g. butorphanol) is especially helpful in feline patients to lessen anxiety in the early stages of therapy. Once the patient has improved rate and effort of breathing, additional testing, including radiographs and laboratory evaluation, can commence. While lung POCUS is efficient at identifying effusions and pulmonary infiltrates acutely, thoracic radiographs still have a place in evaluation of acute cardiac patients because the extent of the problem is immediately visible and improvements due to therapy can be followed over time, and because it provides the opportunity to identify unsuspected pathologies that may be present. Most cats with cardiomyopathy that develop acute congestive heart failure are middle-aged to older, and within that age group, significant co-morbidities like anemia, thyrotoxicosis and chronic renal disease (CRD) are common.

Echocardiographic examination to diagnose the exact heart disease and any complications (e.g.

pericardial effusion) is particularly important for cats without a cardiac history but should be performed in all cats with CHF as soon as they are stable enough to tolerate it. Assessments of left atrial size, function and contents (e.g. possible thrombi) as well as LV size and function can be compared to available previous findings; in repeat echocardiographic patients, changes in these findings can guide adjustments in therapy.

Therapy

Oxygen supplementation, centesis of life-threatening effusions, administration of loop diuretics (IV bolus or CRI) and anxiolytic therapy remains the basic therapy that keeps most feline acute CHF patients alive. Parenteral furosemide therapy for acute CHF signs is a longstanding and familiar regimen. Oral torsemide, another loop diuretic, has been assessed for acute and chronic CHF therapy, but is currently not approved for use in cats. Adjunctive therapies that might be considered later in the course of chronic therapy (e.g. ACE-inhibitors and spironolactone) are not frequently used in the acute setting due to the need for (possibly stressful) oral administration, the longer timeline required for beneficial effects and concerns about possible negative effects on blood pressure and renal perfusion when RAAS inhibitors are used in conjunction with (possibly high doses of) loop diuretics. Dobutamine can be used to increase systolic function and cardiac output and has been particularly recommended for cats with hypothermia with or without bradycardia at presentation. Dobutamine has dose-related side effects in some cats, particularly neurologic side effects like depression or seizures, and arrhythmias. In general, these side effects resolve when the drug dose is decreased, or the drug is discontinued. Pimobendan remains controversial in the setting of acute CHF due to cardiomyopathy in cats. Use of pimobendan in these circumstances is off-label, but pimobendan promotes both systolic and diastolic function as well as increasing left atrial contractile function. This makes pimobendan an interesting potential therapy in feline cardiomyopathies, which often involve limitations of these functions. Use of pimobendan has caused concern in the case of dynamic left ventricular outflow tract obstructions (LVOTO), which may worsen due to the vasodilating and positive inotropic effects of the drug. Retrospective studies of chronic administration of pimobendan to treat CHF due to a variety of cardiomyopathies has revealed few apparent side effects, and one retrospective study of cats with HCM and CHF focused on those with dynamic LVOTO and found few adverse effects.[12] One case-matched study found marked survival benefit of chronic therapy with pimobendan in cardiomyopathic cats13 but other studies have found a more modest survival benefit, if any, when PB is used to treat chronic CHF.[14,15] Pimobendan has been discussed as therapy for cats with systolic dysfunction[7,15-17] and in some cases, for cats with severe CHF due to HCM.[7,17]

Expected outcomes and limitations

Many cats show significant respiratory improvement within the first 24 hours of therapy. Improvement can be assessed by counting respiratory rate and/or assessment of radiographs or pulmonary ultrasound. Aggressive and life-saving loop diuretic dosing carries with it the concern for temporary or permanent renal dysfunction and electrolyte concentration changes that are associated with varying degrees of concern. Renal values of BUN and creatinine may be abnormal at admission for CHF due to pre-existing renal disease or renal hypoperfusion due to decreased cardiac output, coupled with potential dehydration due to inappetence associated with illness. These values typically rise with diuresis, associated with temporary acute kidney injury ± dehydration. Hypokalemia and hypochloremia are common features of feline CHF after diuresis and may be accompanied in some cases by hyponatremia. The changes in sodium and chloride concentrations have been attributed to free water retention due to increased RAAS activity secondary to diuretic therapy. Similarly, hypokalemia may be due to potassium loss mediated by RAAS activity, but loop diuretics also directly cause potassium wasting. In either situation, hypokalemia may be exacerbated by inadequate potassium intake due to inappetence. Despite these biochemical derangements and concerns, studies of acute CHF therapy in cats thus far have indicated that in the short term, increases in creatinine concentration, hypochloremia and hypokalemia may not affect short term survival. These conclusions

are limited, however, but the difficulties in assessing in-hospital survival in patients that may be euthanized for a variety of reasons, not all of which are medical, at any time.

Prognosticating for clients

Overall survival to discharge for cats with acute CHF is approximately 80%, and most cats who survive to discharge are released within 2-3 days. Azotemia and electrolyte disturbances associated with aggressive therapy for CHF are generally not limiting factors for survival and should not be assessed too pessimistically. Many cats may do better at home than in the hospital and recover their appetites and activity levels in a non-medical environment if they have improved enough to oxygenate themselves without help and can be released.

References:

1. Fox PR, Keene BW, Lamb K et al. International collaborative study to assess cardiovascular risk and evaluate long-term health in cats with preclinical hypertrophic cardiomyopathy and apparently healthy cats: the REVEAL study. J Vet Intern Med 2018; 32(3):930-43.
2. Goutal CM, Keir I, Kenney S, et al. Evaluation of acute congestive heart failure in dogs and cats: 145 cases (2007–2008). J Vet Emerg Crit Care 2010;20(3):330–7.
3. Poissonnier C, Ghazal S, Passavin P, et al. Tolerance of torasemide in cats with congestive heart failure: a retrospective study on 21 cases (2016–2019). BMC Vet Res 2020; 16:339-349.
4. Wess G, Sarkar R, Hartmann K. Assessment of left ventricular systolic function by strain imaging echocardiography in various stages of feline hypertrophic cardiomyopathy. J Vet Intern Med 2010;24(6):1375-82.
5. Chetboul V, Blot S, Sampedrano CC, et al. Tissue Doppler imaging for detection of radial and longitudinal myocardial dysfunction in a family of cats affected by dystrophin-deficient hypertrophic muscular dystrophy. J Vet Intern Med 2006;20:640-647.
6. Ferasin L, DeFrancesco T. Management of acute heart failure in cats. J Vet Cardiol 2015;17:S173-S189.
7. Côté E. Feline congestive heart failure. Current diagnosis and management. Vet Clin Small Anim 2017;47:1055-1064.
8. Ohad DG, Segev Y, Kelmer E, et al. Constant rate infusion vs. intermittent bolus administration of IV furosemide in 100 pets with acute left-sided congestive heart failure: a retrospective study. Vet J 2018;238:70-75.
9. Roche-Catholy M, Van Cappellen I, Locquet L, et al. Clinical relevance of serum electrolytes in dogs and cats with acute heart failure: A retrospective study. J Vet Intern Med 2021;35:1652-1662.
10. Dickson D, Little CJL, Harris J et al. Rapid assessment with physical examination in dyspnoeic cats: the RAPID CAT study. J Small Anim Pract 2018;59:75–84.
11. Shimoda T, Osuga T, Nakamura K. Diagnosis of congestive heart failure by combining echocardiography and blood biomarkers in cats with cardiomyopathy. J Vet Cardiol 2025;60:70-78.
12. Ward JL, Kussin EZ, Tropf MA, et al. Retrospective evaluation of the safety and tolerability of pimobendan in cats with obstructive vs. nonobstructive cardiomyopathy. J Vet Intern Med 2020;34:2211-2222.
13. Reina-Doreste Y, Stern JA, Keene BW, et al. Case-control study of the effects of pimobendan on survival time in cats with hypertrophic cardiomyopathy and congestive heart failure. J Am Vet Med Assoc 2014;245(5):534-9.
14. MacGregor JM, Rush JE, Laste NJ, et al. Use of pimobendan in 170 cats (2006–2010). J Vet Cardiol 2011;13(4):251-60.
15. Schober KE, Rush JE, Fuentes VL, et al. Effects of pimobendan in cats with hypertrophic cardiomyopathy and recent congestive heart failure: Results of a prospective, double-blind, randomized, nonpivotal, exploratory field study. J Vet Intern Med 2021;35:789–800.
16. Hambrook LE, Bennett PF. Effect of pimobendan on the clinical outcome and survival of cats with nontaurine

responsive dilated cardiomyopathy. J Feline Med Surg 2012;14: 233–239.
17. Gordon SG, Saunders AB, Roland RM, et al. Effect of oral administration of pimobendan in cats with heart failure. J Am Vet Med Assoc 2012;241(1):89-94.
18. Fuentes VL, Abbott J, Chetboul V, et al. ACVIM consensus statement guidelines for the classification, diagnosis, and management of cardiomyopathies in cats. J Vet Intern Med 2020;34(3):1062-77.

Bugs in the System: Transient Myocardial Thickening

Phil Tricklebank

In humans, transient myocardial hypertrophy has been reported associated with myocarditis since the 1980s[1]. Transient myocardial thickening (TMT) has been described in cats since 2018[2,3], and has been defined as "a clinical condition in cats characterized by reversible left ventricular wall thickening"[4]. Affected cats often present with congestive heart failure and/or arterial thromboembolism (ATE), and at initial evaluation, TMT mimics hypertrophic cardiomyopathy (HCM). TMT is an uncommon observation predominantly affecting young cats, with antecedent events (general anaesthesia or surgery, a fright or trauma, systemic illness) frequently reported. Infectious diseases do not appear to be a common underlying cause. Many aspects of TMT remain unclear, including its aetiology, pathophysiology, early diagnosis, and optimal treatment. A 2025 survey of veterinary cardiologists suggested that recurrence of TMT has been observed, whereby once again there is resolution of the HCM phenotype on echo and an absence of clinical signs[4].

I am cautiously avoiding calling TMT a "diagnosis", as it may be that there are multiple aetiologies that produce a similar reversible clinical picture. For example it has been reported that cats with confirmed Covid-19 infection were documented to go from normal LV wall thickness, to hypertrophy, and back to normal during the disease process[5]. Other proposed aetiologies are a catecholamine surge effect similar to Takutsubo (stress) cardiomyopathy reported in people; oedema or cellular infiltration; or a myocarditis with or without an infectious cause[4].

I present a case of a young cat in acute heart failure with an HCM phenotype on initial presentation. The HCM phenotype resolved completely, confirmed on follow up echo. The case is somewhat unusual in that the patient is my own pet cat and therefore provides some insight into the sudden onset of the condition, and the challenges I faced as both vet and owner in treating the condition in a primarily first opinion setting outside of the care of a dedicated intensive care facility. It gave me first-hand experience and an understanding of the expectations we sometimes place on our owners to medicate pets daily, especially when they start feeling much better and become difficult to pill!

References

1. Hauser AM, Gordon S, Cieszkowski J, et al. Severe tran sient left ventricular "hypertrophy" occurring during acute myocarditis. Chest 1983;83:275–277.

2. Novo Matos J, Pereira N, Glaus T, Wilkie L, Borgeat K, Loureiro J, Silva J, Law V, Kranjc A, Connolly DJ, Luis Fuentes V. Transient Myocardial Thickening in Cats Associated with Heart Failure. J Vet Intern Med. 2018

3. Joseph JL, Oxford EM, Santilli RA. Transient myocardial thickening in a Bartonella henselae-positive cat. J Vet Cardiol. 2018 Jun;20(3):198-203.

4. Novo Matos J, Rishniw M. Understanding transient myocardial thickening in cats: a global survey of veterinary cardiologists. ECVIM abstract Sept 2025 & personal communication.

5. Ferasin L, Fritz M, Ferasin H, Becquart P, Corbet S, Ar Gouilh M, Legros V, Leroy EM. Infection with SARS-CoV-2 variant B.1.1.7 detected in a group of dogs and cats with suspected myocarditis. Vet Rec. 2021 Nov;189(9) & personal communication

CRI furosemide infusion dose for cats
0.33-0.66mg/kg/hr for up to 6-12 hours (from Ware W, Bonagura J. Cardiovascular Disease in Companion Animals: Dog, Cat and Horse *2nd Edition*. CRC Press 2021)
CRI infusion calculator
https://www.vin.com/Members/Calculators/jsCalcs/criCalc_iOS.html

Feline Anaesthesia

Tristan Merlin

Anaesthetic Management of Cardiac Feline Patients

This presentation outlines a structured approach to anaesthesia in feline patients with cardiac disease, integrating species-specific particularities, pharmacological considerations, and clinical case management. We review the goals of anaesthesia in cats, emphasizing the importance of minimizing stress-induced sympathetic stimulation. Balanced anaesthesia is advocated to reduce the cardiovascular impact of individual agents, with an emphasis on suggested uses of alpha-2 agonists. The second section focuses on hypertrophic cardiomyopathy (HCM), the most prevalent cardiac condition in cats. Anaesthetic planning for HCM requires strict heart rate control to preserve diastolic filling, careful fluid management to avoid volume overload, and vigilant blood pressure monitoring.

The final section presents two complex cases:

- A 17-week-old kitten with patent ductus arteriosus (PDA) and subaortic stenosis undergoing open thoracotomy for PDA ligation.
- A feline patient exhibiting ST segment elevation following balloon valvuloplasty.

This presentation underscores the necessity of individualized anaesthetic protocols, informed by cardiac pathophysiology, drug pharmacodynamics, and vigilant perioperative monitoring to optimize outcomes in feline cardiology patients.

Anaesthetic Management of Interventional Cardiac Procedures in Small Animal Patients

This presentation outlines anaesthetic strategies for three high-risk interventional cardiac procedures in small animal patients including patent ductus arteriosus (PDA) closure, balloon valvuloplasty, and pacemaker implantation. Each procedure presents distinct physiological and pharmacological challenges requiring species-specific adaptations.

PDA Closure is commonly performed in paediatric patients and may involve surgical ligation or transcatheter occlusion. Intraoperative goals include maintaining preload, avoiding bradycardia, and ensuring multimodal analgesia. Postoperative care focuses on monitoring for haemorrhage, pneumothorax, and residual shunting.

Balloon Valvuloplasty, typically indicated for pulmonic stenosis, requires precise haemodynamic control. Anaesthetic protocols often include premedication with dexmedetomidine or opioids, followed by induction with midazolam and propofol, and maintenance with sevoflurane. Continuous ECG and invasive blood pressure monitoring are critical due to the risk of arrhythmias.

Pacemaker Placement is more frequently performed in dogs but is also indicated in cats with bradyarrhythmias. Anaesthetic management prioritizes minimizing vagal stimulation and avoiding agents that depress sinoatrial or atrioventricular nodal conduction. Etomidate or alfaxalone may be considered for induction in unstable patients. Emergency protocols for asystole or lead dislodgement should be in place. Postoperative analgesia and sedation must be carefully balanced to prevent sympathetic stimulation or pacing interference.

This presentation emphasizes the importance of individualized anaesthetic planning, advanced monitoring, and interdisciplinary coordination to optimize outcomes in feline and canine cardiac interventions.

ARVC in the cat: a challenge

Ana Maria Pentel

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is an uncommon cardiac disease in feline patients, where the myocardium is gradually replaced with fibro-adipose tissue. It shares characteristics with the human disease of the same name (Fox et al, 2000).

There are few publications regarding ARVC in the feline patient, with the first report found being from the year 2000 that included 20 cats in the USA (Fox et al, 2000), followed by another in 2005 which included 2 patients in Europe (Harvey et al, 2005). There have been some other single case reports since (Backschat et al, 2016; Ciaramella et al, 2009; Nam et al, 2016).

Calan and Thea were two cats that presented to VSS with similar signs at different times, in April 2024 and May 2025, respectively.

Calan was a 6-year-old male neutered Scottish fold cat, that was referred due to pleural effusion. He was in severe respiratory distress when presented at VSS and was stabilised prior to further investigations.

Thea was an 11-year-old female neutered Ragdoll cross, that had been diagnosed at the referring vets with congestive heart failure and started on treatment with furosemide. She was very bright on presentation but had mild dyspnoea and tachypnoea. She was tachycardic and had an irregularly irregular heart rhythm, with poor pulses.

Both patients had pleural effusion, very dilated right atrium and right ventricle with aneurismal areas on echocardiography. There was concurrent left atrial enlargement. The left ventricular walls had normal thickness and left ventricular systolic function was normal in Thea and reduced in Calan, whilst right ventricular systolic function was reduced in both patients. All the heart valves appeared normal and there was no evidence of pulmonary hypertension.

Arrhythmias were present in both patients, however these differed. Calan had a supraventricular tachycardia, whilst Thea had a ventricular arrhythmia with frequent single ventricular premature complexes and paroxysms of ventricular tachycardia.

Both cats had a short survival as has been reported in the literature (Fox et al, 2000).

The diagnosis of ARVC in cats can be challenging. It shares some echocardiographic features of other feline cardiac diseases, such as tricuspid valve dysplasia and restrictive cardiomyopathy. Cardiac MRI is now the gold standard in human medicine for diagnosis of ARVC (Corrado et al, 2024) however echocardiography remains the standard test in our feline patients due to cost and availability of cardiac MRI.

References:

Backschat et al (2016) Arrhythmogenic right ventricular cardiomyopathy in a feline: case report. *Brazilian Journal of Veterinary and Animal Sciences*, 68(5), pp. 1112-1116

Ciaramella, P. et al (2009) Arrhythmogenic right ventricular cardiomyopathy associated with severe left ventricular involvement in a cat. *Journal of Veterinary Cardiology*, 11(1), pp. 41-45

Corrado, D. et al. (2024) Proposed diagnostic criteria for arrhythmogenic cardiomyopathy: European Task Force consensus report. *International Journal of Cardiology,* 395, 131447.

Fox, P. et al (2000) Spontaneously occurring arrhythmogenic right ventricular cardiomyopathy in the cat – a new animal model similar to the human disease. *Circulation*, 102 (15), pp. 1863-1870

Harvey, A. et al (2005) Arrhythmogenic right ventricular cardiomyopathy in two cats. *Journal of Small Animal Practice*, 46 (3), pp. 151-156

Nam, A. et al (2016) Arrhythmogenic right ventricular cardiomyopathy on a cat with hepatic lipidosis. *Journal of Veterinary Clinics*, 33(3), pp. 160-164

DCM in Deerhounds: Some Heart-Stopping Pathology

Natasha Wayne-Wynne and Emily Dutton

Dilated cardiomyopathy (DCM) is a genetic and acquired disease that commonly affects large breed dogs. The prevalence of DCM in deerhounds has been reported at 21.6%, with a 13.6% incidence of ventricular arrhythmias in affected dogs. We were interested in exploring the associated histopathology changes with DCM in this breed.

Two histological patterns have previously been associated with DCM; fibro-fatty infiltration of the myocardium and attenuated wavy changes. The aim of our study was to confirm and describe the histopathological findings on post-mortem of deerhounds that died as a result of DCM diagnosed using either echocardiography, Holter analysis, or both.

Data was collected prospectively at breed screening events from 2014 until 2025, including a population already described. Owner consent was obtained and samples were sent for post-mortem analysis by the pathology team at the Small Animal Teaching Hospital at Liverpool University. The dogs were categorised as normal, equivocal or affected for DCM based on echocardiographic findings. A continuous 24-hour ambulatory ECG (Holter) analysis was offered to all patients enrolled

Of 175 deerhounds enrolled, consent was received for 7 hearts from those deerhounds that died to be sent for post-mortem. One was excluded due to the presence of non-cardiac systemic disease that may have contributed to the patient's death. All dogs died or were euthanised due to their cardiac disease except for one, who was euthanised due to hindlimb weakness. This bitch was dam to a dog which died suddenly due to suspected ventricular arrhythmias.

Our report aims to describes histology and Holter monitor findings in deerhound hearts with echocardiographic or ECG changes due to suspected DCM. We identified a clinically relevant cardiomyopathy which closely mimics arrhythmic cardiomyopathy in other dog breeds. Limitations included the absence of post-mortem findings in control (i.e. non-DCM) deerhounds and non-standardisation of treatment protocols. We hope that the findings will improve our understanding of this disease in Deerhounds, and contribute to a platform for continued research in the future.

Hearts of Champions: Canine Athletes and the Whippet Addendum

Rebecca Stepien

Exercise-Induced Cardiac Remodeling

It has long been recognized in the scientific literature that distinct cardiovascular adaptations occur in human beings and other mammals in response to strenuous exercise. For hundreds to, in some cases, thousands of years, domesticated dogs have been subject to breeding and training pressure as their human companions used them for work and for sport.

The so-called "athlete's heart" has been used as a descriptor of the cardiac remodeling and changes in function that occur in response to strenuous training. More recently, the term "exercise-induced cardiac remodeling" (EICR) has come into use to describe these changes.[1] Typical morphologic changes noted across many types of athletes include balanced biventricular dilation, mild-to-moderate increases in left ventricular wall thickness, and biatrial dilation, but there is variety in the degree of change seen in these parameters that relates to the type of physiologic stress applied to the heart by different types of exercise. Classically, the changes have been described as attributable to isometric stress and isotonic stress, and the preponderance of either of these in a given sport results in "classic" morphologic changes.[2] More recently, these physiologic stress components have been categorized as a continuum of "static" and "dynamic" stressors,[3] emphasizing that most trained athletes have a combination of findings. The term "exercise-induce cardiac remodeling" has come to cover a range of changes that are not binary (i.e. either present or not), and EICR is considered complex variable involving intensity, frequency and duration as well as type of training. Less easily measured and highly variable factors will also "load" the heart and include the stress of competition during the event (sympathetic nervous system activation), environmental factors, electrolyte changes that may occur during events, and other factors.[4] Despite the recognized changes in morphology that occur with athletic training, functional measures at rest do not typically change much, or may even decrease slightly, and resting heart rate decreases. It's important to note that these static or decreased functional measures are at rest; when trained athletes are mid-performance, their cardiac output, underpinned by increased stroke volume due to remodeling, undergoes multifold increases as a result of the augmented contractile reserves of the ventricles. Understanding EICR and its echocardiographic appearance is an important part of pre-participation screening in human athletes where cardiac remodeling associated with peak physical conditioning might be mistaken for disease (HCM, DCM, ARVC) and the athlete might not be allowed to continue competing. In dogs, precompetition screening is not currently an issue, but changes associated with EICR might be confused with DCM or other forms of cardiomyopathy, "normal" remodeling in response to training be misjudged as pathologic in a dog presented for evaluation of "poor performance" and in breeding dogs, EICR changes might complicate pre-breeding screening, especially where subtle forms of diseases like aortic stenosis are of concern. Lastly, dogs and people with EICR share a common concern: just because one is an athlete doesn't mean that subclinical cardiac disease is not present, and familiarity with EICR changes is needed to recognize when EICR is unlikely to be the cause of certain findings.

The echocardiographic appearance of the hearts of different breeds of dogs differs[5-12] and the characteristic changes seen in individual canine athletes are likely to the result of both genetic and training pressure.[13] Even in non-purebred athletes like Alaskan sled dogs, purpose breeding for athletic performance results in dogs with the physical capability to respond to strenuous training. The athletic profile of the training and of the competitive events likewise provides specific stimulus for remodeling.

Alaskan sled dogs are trained to perform endurance running while in harness. The load that a team (often 12-16 dogs) pulls varies based on the sled, the musher and the load on the sled. Dogs run the Iditarod (~1100 miles, 1770 km) over ~10 days in February, but organized training (daily runs up to 160 km) begins in September, so dogs undergo organized training for 4-5 months per year. Changes in cardiac morphology occur as a result of training in sled dogs performing this type of endurance running, including an increase in LV internal diameter in diastole and systole, increased IVS thickness in diastole, increased LA diameter and a 24% increase in LV mass. Measures of systolic function (fractional shortening, stroke volume, ejection fraction) in sled dogs do not change with training and tend to remain at the lower of end of the range generally accepted for healthy dogs of any breed

(mean FS after training 24%, mean EF 47%),[14] and resting heart rate decreases. Measures used to identify concentric remodeling (LVIDd: LVWd and IVSd: LVWd) in conjunction with increases in LV diastolic diameter indicate that, although there was evidence of dynamic loading based on increased LVIDd, the wall thickening changes were disproportionate and suggest that a combined static and dynamic load was the stimulus for the EICR noted. The combination of increased blood volume associated with endurance training and the pressure load during running (sled dogs' systolic blood pressure while running is as high as 300 mmHg and is maintained at that level throughout the course of a run[15]) forms the basis of this "mixed load". In addition, there a static load that each dog bears while pulling a loaded sled and musher, but unlike two-legged runners like humans, four-legged runners are by definition using their upper (cranial) body as well as their lower (hind) limbs, making their sport more akin to cross-country skiing or rowing than straight endurance running.3
Whippets and other sighthounds have echocardiographic measurements that exceed expected ranges based on body weight.16 Previous studies have indicated that the "normal" reference range for different dog breeds varies by weight, but also by body conformation such that breed-specific normal echocardiographic reference ranges may require more than "just" weight-normalized values.[6-12,16,17]
In breeds with a long history of athletic performance, it's conceivable that reference range development included healthy dogs with morphology reflecting their purpose breeding as well as their degree of athletic training at the time of examination. A recent study attempted to discern higher degrees of EICR in competitive whippets from "normal healthy" whippet cardiac morphology.[17]
Whippets participate in a variety of competitive sports, including short track racing, lure coursing and agility events. Although many of these events call for sprinting short distances, there are also isometric challenges (jumping, climbing). Because of the four-legged nature of all dog sports, neither pure isometric or isotonic-type changes are anticipated, and these dogs' hearts may also be expected to lie on a continuum between dynamic and static sports.
In human athletes, expected LV changes of EICR include some combination of increases in LV wall thickness, increases in LV cavity dimension, normal LVEF and normal or supranormal diastolic function in endurance athletes (D. Phelan, personal communication). The conditioned whippets in our recent study showed increased LVIDd and LVIDs in conditioned dogs compared to unconditioned dogs when normalized to body mass, as well as an ~24% increase in calculated LV mass/kg. As in people, conditioned dogs FS and EF did not differ based on training, but differences in E wave velocity and E/A ratio supported more rapid early diastolic filling of the LV. This "pro-diastolic" functional change (in diastolic performance) is of interest in human athletes. It has been said that athletes with EICR have "large and compliant" hearts (Levine B, personal communication). The finding that "lifelong competitive athletes seem largely spared from the typical declines in LV diastolic function that accompany advancing age"[18] makes the case for continued exercise throughout life in a species (human beings) in which both advanced age and heart failure with preserved ejection fraction (HFpEF) is not uncommon. In whippets, values associated with early diastolic filling were higher in conditioned dogs (vs. unconditioned), supporting the pro-diastolic findings in athletic dogs. Other echocardiographic findings associated with athletic training that have not been systematically studied in dogs but occur in human athletes include RV enlargement (associated with endurance but not power sports, roughly proportional to LV enlargement) and aortic enlargement (generally mild and measures stay within reference ranges).

<u>The Whippet Addendum</u>
Several common findings in athletic whippets (trivial mitral regurgitation, larger LV diameters and lower systolic functional parameters compared to other breeds of similar size, aortic systolic velocities greater than some other breeds) may introduce uncertainty when whippets are screened for breeding purposes, examined because of a newly detected heart murmur or examined for poor performance. Mitral regurgitation is common in the whippet breed, with approximately 50% of dogs affected to some degree by ~7 years of age.[19] Twenty-one percent of dogs of breeding age (2-5 years) had echocardiographically-identifiable MR;[19] these years commonly coincide with peak competitive years in common whippet events (median [range] age of conditioned dogs 2.5 [1-5.8] years[17]), suggesting that peak competitive performance might temporally coincide with the presence of some degree of pathology in some dogs. In addition, many whippets in peak athletic condition may have "trivial" mitral regurgitation as a consequence of increased LV size in the absence of identifiable markers of myxomatous mitral valve disease (MMVD) like valvular thickening or valve leaflet prolapse. These dogs, while within the range of normal variation with trivial, non-eccentric MR jets

in the absence of valvular abnormalities, may go on to develop MMVD as a consequence of genetic background.[20] The coincidence of predisposition to MMVD and athletic competition in whippets bear a resemblance to concerns in human athletes regarding pre-existing cardiac disease and clearance for athletic training, although actual pre-competition screening is rare in dogs. The larger size of the whippet ventricle compared to other dogs of similar size, in conjunction with systolic parameters commonly appearing at the lower end of the reference range for other breeds may lead to concerns about dilated cardiomyopathy, and higher aortic systolic velocities and increase LW wall thickness in some young athletic whippets may lead to exclusion from the breeding pool due to concerns about aortic or subaortic stenosis. More studies regarding the natural history of MV regurgitation due to MMVD in this breed are ongoing and may provide additional information regarding specific echocardiographic risk factors for development of MMVD. In addition, characterization of MMVD diagnosed in competitive whippets may shed light on which findings are most concerning as athletic whippets continue to compete having been diagnosed with MMVD.

References

1. Weiner RB, Baggish AL. Exercise-induced cardiac remodeling. Prog Cardiovasc Dis 2012;54:380-386.
2. Morganroth J, Maron B, Henry WL, et. al. Comparative left ventricular dimensions in trained athletes. Ann Intern Med 1975;82:521-524.
3. Martinez MW, Kim JH, Shah AB, et. al. Exercise-induced cardiovascular adaptations and approach to exercise and cardiovascular disease. JACC State-of-the-Art Review. J Am Coll Cardiol 2021;78(14):1453-1470.
4. Mitchell JH, Haskell W, Snell P, et al. Task Force 8: Classification of Sports. J Am Coll Cardiol 2005;45(8):1364-1367.
5. Dickson D, Bode E, Linney C, et. al. Differences in left ventricular enlargement secondary to chronic volume loading between english springer spaniels and two similar sporting breeds. J Vet Cardiol 2023;48:37-45.
6. Seckerdieck M, Holler P, Smets P, et.al. Simpson's method of discs in salukis and whippets: Echocardiographic reference intervals for end-diastolic and end-systolic left ventricular volumes. J Vet Cardiol 2015;17:271-281.
7. Giraut S, Häggström J, Koskinen LLE, et. al. Breed-specific reference ranges for standard echocardiographic measurements in salukis. J Small Anim Pract 2019;60:374–378.
8. Bavegems V, Duchateau L, Sys SU, et. al. echocardiographic reference values in whippets. Vet Radiol Ultrasound 2007;48:230–238.
9. Vollmar AC. Echocardiographic measurements in the Irish wolfhound: reference values for the breed. J Am Anim Hosp Assoc 1999;35:271–277.
10. Dutton E, Cripps P, Helps SAF, et. al. Echocardiographic reference intervals in healthy UK deerhounds and prevalence of preclinical dilated cardiomyopathy: a prospective, longitudinal study. J Vet Cardiol 2022;40:142-155.
11. Della Torre PK, Kirby AC, Church DB, et. al. Echocardiographic measurements in greyhounds, whippets and Italian greyhounds-dogs with a similar conformation but different size. Austr Vet J 2000;78:49–55.
12. Page A, Edmunds G, Atwell RB. Echocardiographic values in the greyhound. Austr Vet J 1993;70:361–364.
13. Shave R, Howatson G, Dickson D, et. al. Exercise-induced cardiac remodeling: lessons from humans, horses, and dogs. Vet Sci 2017; 4(9). doi:10.3390/vetsci4010009.
14. Stepien RL, Hinchcliff KW, Constable PD, et. al. Effect of endurance training on cardiac morphology in Alaskan sled dogs. J Appl Physiol 1998;85:1368–1375.
15. Van Citters RL, Franklin DL. Cardiovascular performance of Alaska sled dogs during exercise. Circ. Res. 1969;24:33–42.
16. Esser LC, Borkovec M, Bauer A, et. al. Left ventricular M-mode prediction intervals in 7651 dogs: Population-wide and selected breed-specific values. J Vet Intern Med 2020;34:2242–2252.
17. Stepien RL, Kellihan HB, Visser LC et.al. Echocardiographic values for normal conditioned and unconditioned North American whippets. J Vet Intern Medicine 2023;37: 844–855.
18. Baggish AL. Exercise-induced cardiac remodeling: Competitive athletes are just the tip of the iceberg. Circ Cardiovasc Imaging. 2016;9:e005321.
19. Stepien RL, Kellihan HB, Fuentes VL. Prevalence and diagnostic characteristics of non-clinical mitral regurgitation murmurs in North American Whippets. J Vet Cardiol 2017;19:317–324.
20. Stern JA, Hsue W, Song K-H et. al. severity of mitral valve degeneration is associated with chromosome 15 loci in whippet dogs. PLoS ONE 2015;10:e0141234.