



To our Veterinary Partners:

During this difficult time, we'll be keeping our website updated continuously with our mitigation protocols. Most recently, we've adopted telemedicine to best serve the needs of our shared patients.

Please visit our website at metro-vet.com, and the link to our Covid-19 page will be accessible at the top of the screen (and from any page within our site).

Should you have questions or concerns please feel free to reach out to me at any time. Wishing you, your family, and your teams health and safety.

John W. DeBiasio, DVM, DACVIM Medical Director | Internal Medicine Specialist

ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY (ARVC)

Megan Poad, VMD, DACVIM (Cardiology)

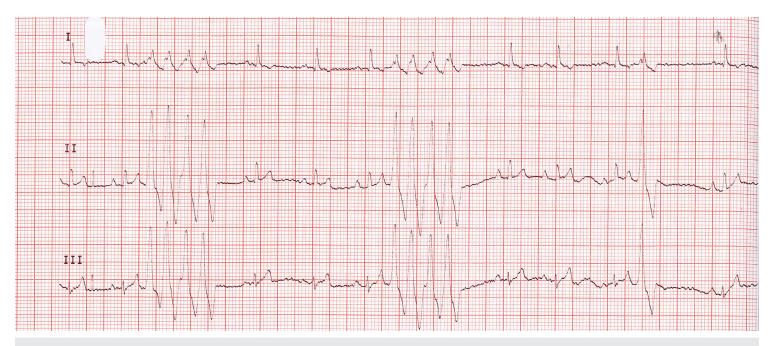
Arrhythmogenic right ventricular cardiomyopathy (ARVC) is an adult-onset, inherited myocardial disease that is seen commonly in Boxers but can occur in other breeds, including Bulldogs. It is caused by fibrous fatty infiltration of the myocardium of the right ventricle and primarily affects the electrical system of the heart. ARVC causes ventricular arrhythmias, which can result in syncope and/or sudden death. In a small percentage of cases, ARVC can result in development of left ventricular

systolic dysfunction, ventricular dilation, and congestive heart failure.

A similar type of heart disease exists in humans, in which there are various mutations in multiple genes that can lead to development of ARVC. So far, one genetic mutation in the gene striatin has been found to be associated with development of ARVC in Boxers. Striatin is a desmosomal protein located in the intercalated disc of cardiac myocytes.

Desmosomes play an important role in cell-to-cell adhesion and structural integrity of heart muscle, and it has been hypothesized that abnormalities in desmosome function may lead to cardiac myocyte death, inflammation, and fibrosis.

Most commonly, dogs are diagnosed with ARVC between 5 and 7 years of age, however some dogs are diagnosed at a young age (1-3 years old). The most common presenting complaint is syncope, which often is associated



Electrocardiogram (leads I, II, and III) from a Boxer presenting for syncope. Upright ventricular premature complexes (left bundle branch block morphology) occurring as non-sustained runs of ventricular tachycardia (with R-on-T) and one single VPC.

with a period of exercise or excitement, however they do not have to be. Some affected dogs are completely asymptomatic. Dogs with myocardial systolic dysfunction may present with signs of exercise intolerance or lethargy. Dogs may present in left or right congestive heart failure, or both.

DIAGNOSIS

Diagnosis of ARVC is based on the presence of a combination of findings rather than on one specific diagnostic test. The presence of a ventricular tachyarrhythmia in an adult Boxer (and/or Bulldog) without another cause for the arrhythmia supports presence of the disease. In dogs with this clinical presentation, a family history of ARVC, a positive test for the mutation in the striatin gene, and a history of syncope are strongly supportive. Although, it is important to remember that many dogs with ARVC are asymptomatic.

Physical examination findings may include occasional premature beats, a heart murmur, or signs of left or right heart failure (including dyspnea, ascites, and/or jugular venous distention). In some dogs with ARVC, no physical exam abnormalities are detected. An electrocardiogram performed over a few minutes is commonly normal in affected dogs. Ventricular premature

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complexes (VPCs) may be present as singles, pairs, triplets, or runs of paroxysmal ventricular tachycardia. The VPCs are most commonly of right ventricular origin, represented by a left bundle branch morphology (appearing as upright QRS complexes on a lead II electrocardiogram).

Holter monitoring is an important test for diagnosis, screening, and management of ARVC. A 24-hour Holter monitor is recommended for any patient for which there is a clinical suspicion of ARVC. Holter monitor placement typically is considered for any dog with an arrhythmia (either on auscultation or recorded on ECG), clinical signs of syncope or exercise intolerance, a family history of ARVC, or presence of the genetic mutation in an adult boxer (even if the ECG findings are normal). Although strict criteria for diagnosis of ARVC based on Holter monitor results have not been established, the observation of >100 VPCs, or ventricular

couplets, triplets, or runs of ventricular tachycardia in the absence of another cause for the ventricular arrhythmia, strongly supports a diagnosis of ARVC. Since ARVC is an adult-onset disease, and ventricular ectopy may increase with age, clinical screening typically involves annual Holter monitoring.

Thoracic radiograph findings are usually normal. Although, in a small number of dogs with ventricular dilation and systolic dysfunction, cardiomegaly and evidence of congestive heart failure (left and/or right-sided) might be seen. Echocardiography is performed to assess for myocardial systolic dysfunction and left ventricular +/- left atrial chamber dilation, which occurs in a small number of dogs with ARVC. Many affected dogs have normal echocardiographic examinations. Cardiac biomarkers, including cardiac troponin I and BNP. have not been shown to be useful independent screening tests for ARVC. Postmortem findings can be helpful in

diagnosing ARVC in an adult boxer or bulldog that has died suddenly.

Histopathology should identify segmental or diffuse, fatty or fibrofatty replacement of the myocardium. The right ventricular free wall is most commonly affected; however, the interventricular septum and left ventricular free wall can also be involved.

Genetic screening to identify boxers with the deletion mutation in the striatin gene is available through the North Carolina State University Veterinary Genetics laboratory. This test is recommended for boxers intended for breeding. The test also can be used to identify dogs at risk of developing ARVC.

TREATMENT

Treatment of ARVC typically involves administration of antiarrhythmic medication(s) to try to reduce frequency and complexity of ventricular tachyarrhythmias and to decrease frequency of syncopal episodes. Absence of clinical signs does not mean that there is no risk for sudden cardiac death. Strict criteria for initiation of antiarrhythmics in asymptomatic dogs with ARVC have not been established. If an arrhythmia is detected incidentally, Holter monitor placement is recommended to evaluate the frequency and complexity of the arrhythmia. This information is used to help determine if therapy is indicated. Treatment typically is recommended in dogs with frequent VPCs, runs of ventricular tachycardia, and other evidence of increased complexity of the arrhythmia (i.e. ventricular couplets and triplets, R-on-T). Dogs with syncope and ventricular tachyarrhythmias generally are started on antiarrhythmic therapy.

The most common oral antiarrhythmic medications used to treat ARVC in dogs are sotalol and mexiletine. Other anti-arrhythmic medications might be required. Owners should be made aware that antiarrhythmics have the potential for proarrhythmic effects and that there is no evidence that treatment decreases the risk of sudden death. However, antiarrhythmic treatment has been shown to decrease frequency of VPCs and number of syncopal episodes.



Ideally, a Holter monitor would be placed before initiation of treatment and repeated 2-3 weeks after starting antiarrhythmic medication to evaluate the efficacy of treatment and assess for any potential proarrhythmic effect. A pretreatment Holter monitor recording might not be possible in some cases, especially if syncopal episodes are frequent or ventricular tachycardia is observed. If the posttreatment Holter monitor shows inadequate control of the arrhythmia, antiarrhythmic therapy can be adjusted followed by Holter monitor placement in another 2-3 weeks. Oral supplementation of fish oilsmight also decrease the number of VPCs and can be considered as another possible treatment in combination with antiarrhythmic medication.

For affected dogs with echocardiographic evidence of ventricular dilation and systolic dysfunction, specific treatments for dilated cardiomyopathy (pimobendan, ACE inhibitors, diuretics) might be recommended.

PROGNOSIS

Unfortunately, dogs with ARVC are always at risk for sudden cardiac death. Many affected dogs live for years on anti-arrhythmic medication without clinical signs. A small percentage of dogs may develop ventricular dilation, systolic dysfunction, and congestive heart failure.

SPECIALIZED SERVICES

BEHAVIOR

Jacqueline Wilhelmy, MS, VMD, DACVB, CCBC-KA

CARDIOLOGY

Marc Kraus, DVM, DACVIM (Cardiology) Michael Miller, MS, VMD, ABVP Megan Poad VMD, DACVIM (Cardiology) Risa Roland, DVM, DACVIM (Cardiology)

DENTISTRY

Corinne Durand, DVM

DERMATOLOGY

Katherine Backel, DVM DACVD Karen B. Farver, DVM, DACVD

EMERGENCY AND CRITICAL CARE

James Buckman, PhD, VMD
Allison Buysse, VMD
Jason Chamberlin, VMD
Kathleen Crossman, DVM
Cierra French, DVM
Robert Gaunt, VMD
Jill Kalman, VMD
Jennifer McGough, VMD
Rachel Morgan, DVM
(Practice limited to Emergency & Critical Care)
Katie Slade, VMD
Marisa Suvannavejh, VMD
Katrina Tumielewicz, DVM
(Practice limited to Emergency & Critical Care)

INTERNAL MEDICINE

John V. DeBiasio, DVM, DACVIM James F. Dougherty, MS, VMD Tabitha A. Hutton, DVM, MTR, DACVIM (SAIM) Leslie A. Kuczynski, VMD, DACVIM

NEUROLOGY

Lisa Lipitz, VMD, DACVIM (Neurology Daniella Vansteenkiste, BVetMed

ONCOLOGY

Lillie Davis, DVM, DACVIM (Oncology) Suzanne Rau, DVM, DACVIM (Oncology)

OPHTHALMOLOGY

Amanda Corr, VMD, DACVO Chloe Spertus, DVM

RADIOLOGY

Robert McLear, VMD, DACVR Lisa Suslak, VMD, DACVR

SURGERY

Kendra Hearon, VMD, DACVS-SA ACVS Fellow, Surgical Oncology A. Jon Nannos, DVM Jacqui Niles, BVETMED, SAS, DACVS Catherine Popovitch, DVM, DACVS, DECVS Timothy M. Schwab, VMD, DACVS-SA Rebecca Wolf, VMD, DACVS-SA

POSTPONED TO A FUTURE DATE TBD:

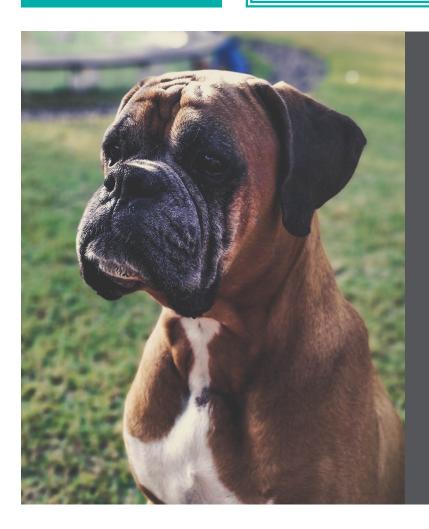
11th Annual MVA5K All April CE events All May CE events

Stay tuned for reschedule dates

ABOUT MEGAN POAD, VMD, DACVIM (CARDIOLOGY)

Dr. Megan Poad grew up in Bel Air, Maryland. She earned her Bachelor of Science degree in Animal Science from the University of Delaware and her Master of Science degree from Cornell University, where she worked primarily with dairy cows.

Megan graduated summa cum laude from the University of Pennsylvania School of Veterinary Medicine in 2015. She stayed at the Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania for a one-year rotating internship in small animal medicine and surgery and a three-year residency in Cardiology, which she completed during the summer of 2019 before joining the MVA team. During her residency, Megan received the Student Chapter of the American Veterinary Medical Association Class of 2019 Resident Teaching Award. She became board certified in cardiology by the American College of Veterinary Internal Medicine in 2019.



PET LOSS SUPPORT GROUP

At MVA we understand the depth of loss one experiences when a beloved four-legged family member has passed. For that reason, we provide a Pet Loss Support Group to help grieving owners in need. Our group is designed to provide grieving pet parents with a safe, confidential environment to share their feelings with others who have experienced pet loss.

The group is operated by professionals who have experience with pet loss. A board certified psychiatrist consults with us regarding the implementation of the group, however, our group leaders are not mental healthcare professionals. Clients experiencing difficulty coping are urged to seek help from a mental healthcare professional. We can provide you with the names of health care professionals if needed.

Our Pet Loss Support Group meets on a varying schedule. Until further notice our support group will be held virtually.

For dates please call the hospital at 610.666.1050 or visit metro-vet.com/petloss



















