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ARRHYTHMIAS IN DOGS WITH CARDIOMYOPATHY

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The American heart association defined cardiomyopathy as a *"group of diseases of the myocardium associated with mechanical and/or electrical dysfunction that usually (but not invariably) exhibit inappropriate ventricular hypertrophy or dilatation and are due to a variety of causes that frequently are genetic. Cardiomyopathies either are confined to the heart or are part of generalized systemic disorders, often leading to cardiovascular death or progressive heart failure–related disability." (Circ 2006)*

This definition is important because it would include other conditions that fall into the definition of cardiomyopathy, including electrical dysfunction (ion channelopathies and conduction system disorder). Additionally, arrhythmia can facilitate the development of arrhythmia-induced cardiomyopathy. In some patients, it is challenging to determine whether the arrhythmia caused the remodeling or if an underlying cardiomyopathy led to arrhythmias.

Arrhythmia-induced cardiomyopathy

Arrhythmias can occur due to different underlying reasons: congenital conduction system abnormalities (i.e., accessory pathways leading to orthodromic-atrioventricular-reciprocating tachycardia), ventricular or atrial tachycardias associated with structural heart disease, as well as atrioventricular or atrial conduction disturbances with no obvious structural or congenital substrate to explain their origin.

Until recently, only fast arrhythmias were considered capable of causing myocardial remodeling. One of the typical examples was the observation that a DCM phenotype could be reproduced in the experimental settings within few weeks from the implant of a pacemaker programmed at a relatively fast heart rate. However, arrhythmias can also induce structural changes in patients with frequent atrial or ventricular arrhythmias, bundle branch blocks and even when chronic, pathological bradycardia is present. This led to the updated definition of arrhythmia-induced cardiomyopathy (AIC) rather than tachycardia-induced cardiomyopathy. However, please note that in people this term does not include cardiomyopathies due to conduction abnormalities/ dissynchrony, such as chronic RV pacing, left bundle branch block and pre-excitation.

Arrhythmia-induced cardiomyopathy in people: current information

Arrhythmia-induced cardiomyopathy is defined as a potential reversible form of cardiomyopathy, which leads to left ventricular dysfunction and heart failure secondary to atrial or ventricular tachyarrhythmias or frequent ventricular ectopy. 2 categories of AIC are recognised:

1. arrhythmia induced: the arrhythmia is the sole reason for ventricular dysfunction
2. arrhythmia mediated: the arrhythmia exacerbates ventricular dysfunction and/or worsens HF in a patient with concomitant heart disease.

The latter is more frequently identified in dogs.

Arrhythmias in dogs and comparison with people

There are a few conditions in dogs that can result in AIC, but most of the clinical scenarios are arrhythmia-mediated, rather than arrhythmia-induced. This may explain why, in most situations, arrhythmia management will not be associated with reverse remodeling despite clinical and partial echocardiographic improvement.

Tachycardia- induced cardiomyopathy (supraventricular or ventricular origin)

Reversible LV dysfunction solely due to increase in ventricular rates, regardless of tachycardia origin. In dogs, the following incessant or sustained arrhythmias could potentially be associated with AIC:

- focal atrial tachycardia
  In a retrospective study, mean heart rate in dogs with FAT was 164-270 bpm, and a DCM phenotype was reported in 3/16 dogs in the same case series. FAT arising from the right atrium were predominant (63%, mainly from crista terminalis and triangle of Koch) followed by pulmonary veins (37%). Persistent and paroxysmal AF were triggered by FATs in 7 dogs (the majority with at least one focus at the pulmonary veins).
  Different treatments are available for FAT management. No data is available about reverse remodeling following successful FAT management in dogs with a DCM phenotype.
- orthodromic AV reciprocating tachycardia associated with accessory pathways (OAVRT)
  DCM phenotype in 46.1% of dogs with complete or partial resolution of AIC after successful radiofrequency catheter ablation. 38% of dogs presented with congestive heart failure. Labrador breed and male sex overrepresented.
  OAVRT identified in the vast majority of dogs, and 48.3% of the dogs in a study exhibited ventricular pre-excitation sometime during sinus rhythm (53% dogs showed manifest, 47% dogs had intermittent pre-excitation).
- isorhythmic atrioventricular dissociation with focal junctional tachycardia
  a DCM phenotype was reported in 6/11 dogs in a case series. Labrador Retriever breed overrepresented.
  Type I IAVD was characterised by P waves (sinus node) and rhythmic fluctuation of the PR, with P waves marching in and out of the QRS
  Type II fixed, short PR and P-QRS coupling
  No optimal treatment for these cases. AIC could potentially arise due to a fixed, higher than normal HR (mainly higher than HR at rest in a normal dog); median heart rate reported was 140 bpm.
- ventricular tachycardia
  There are no specific case series defining the incidence of AIC in dogs. Ventricular tachycardia can be associated with structural heart disease as well as less commonly be associated with severe systemic condition as a result of myocardial damage, toxin release or hypoxia.

Atrial fibrillation

In people, atrial fibrillation is a common cause of AIC. So far ventricular rate during AF does not seem to predict reversibility of the AIC, and this has changed the focus to AF duration and/or irregularity rather than ventricular rate. As a matter of fact, a DCM phenotype can arise despite appropriate rate control (and this can also be observed in dog's breeds prone to lone AF).

In people, atrial fibrillation- induced cardiomyopathy is characterised by LV systolic dysfunction in patients with paroxysmal or persistent AF despite appropriate rate control and it is a diagnosis of exclusion. Holter analysis needs to rule out inadequate rate control and for confirmative diagnosis, restoration of sinus rhythm should reverse the DCM phenotype. Mechanisms associated with the pathophysiology of AF-induced cardiomyopathy include irregular cycle lengths and loss of atrial contraction.

Large and giant breed dogs are most commonly affected with AF and similarly, large breed dogs are also frequently diagnosed with a DCM phenotype (43.5–50%). Breeds that have been reported to develop AF include Dogue de Bordeaux, Irish Wolfhound, Great Dane, Newfoundland, Mastiff, German shepherd, Rottweiler, Labrador retriever and Australian Shepherds. In a study investigating success rate of DC electrical cardioversion for rhythm control, approximately 64% of the dogs with AF had structural heart disease, with a DCM phenotype being the most common (30%). Nearly all dogs were successfully cardioverted, but duration of sinus rhythm was longer for those not showing structural heart disease (690 vs. 73 days). No data is available about possible development of a DCM phenotype in the group of dogs diagnosed with lone AF, apart from the Irish Wolfhound cohort.

Irish Wolfhounds with lone atrial fibrillation and no obvious structural changes have been the subject of a restrospective, case-control study. Half of the Irish Wolfhounds with AF and no structural heart disease developed a DCM phenotype and age and gender-matched Irish Wolfhounds used as a control had a lower rate of DCM incidence compared to the AF group. Similarly to what has been speculated in people with AF, heart rate at the initial visit did not correlate with survival, despite Irish Wolfhounds with AF had faster heart rates compared to controls (144 bpm versus 118). This study could not prove (or disprove) that AF could cause a DCM phenotype, but it further shows that dogs with AF may be more prone to develop a dilated, poorly contracting heart in a breed where DCM phenotype is commonly identified.

Another retrospective study also showed that systolic dysfunction was identified in 60% (50/83) of dogs that presented in AF; in the group of dogs with echocardiographic changes, a DCM phenotype was identified in 49% (41/83) of dogs. Heart rate was not predictive of outcome in this study as well, and similarly fractional shortening was not a useful criterion.

Loss of atrial kick, fast and irregular heart rate can worsen or precipitate clinical signs in dogs. It is common to approach a dog referred for congestive heart failure signs and concomitant rhythm disturbances, and treatment should aim at treating congestive heart failure and address the rhythm disturbance. Because CHF will cause sympathetic activation and a faster heart rate, it is prudent to start addressing CHF in the first place and re-evaluate the (likely) need for antiarrhythmic treatment in these dogs.

This can be true not only in large-breed dogs, but also in small breed dogs. Atrial fibrillation was a poor prognostic factor in a study of small breed dogs with degenerative mitral valve disease. Another study similarly found that small breed dogs with atrial fibrillation had a poorer prognosis compared to large breed dogs (median survival time 1.1 mo vs 32 mo for large breed dogs). Paroxysmal AF was also identified recently in dogs with degenerative mitral valve disease.

In contrast with people, there is no data about true AIC due to AF in dogs, as reverse remodeling is barely never seen in our patients due to concurrent structural heart disease. In general, it is always reasonable to consider the possibility of structural heart disease that cannot be confidently diagnosed in a dog presenting with AF, even in dogs that successfully were cardioverted or that show lone AF. These patients will need to be monitored over time.

Ventricular arrhythmias

In people, the diagnosis of premature ventricular complex–induced cardiomyopathy is presumptive based on the presence of frequent ventricular ectopic beats, an existing cardiomyopathy, and the lack of an alternative etiology for the cardiomyopathy. Even in people (and less so in dogs), the exact percentage of VPC burden (the total percentage of VPCs over normal beats) leading to AIC is not strictly clear-cut; in people, values below 4% of total VPC count were associated with a DCM phenotype, but cut-off variables to suggest a higher risk of AIC range from 16 to 25%. Some authors would consider a VPC burden of 10% as a criteria to define VPC-induced cardiomyopathy. The risk of sudden cardiac death in this population is not well studied.

The mechanism associated with the pathophysiology of VPC-induced cardiomyopathy include heart rate irregularity and post-extrasystolic potentiation, LV dyssynchrony, AV dyssynchrony, and increased heart rate. VPC suppression is considered successful if burden is decreased by >80% of baseline VPCs, as it likely represents a true effect of treatment rather than spontaneous VPC variability.

Most of the available data about VPCs number and Holter analysis is associated with Boxer dogs with a clinical diagnosis of ARVC. Other conditions associated with an abnormal number of VPC include DCM phenotype, myocarditis as well as systemic conditions (e.g., pheochromocytomas, sepsis, gastric-dilation volvulus, hemoabdomen and others). In most of these scenarios, the heart may look abnormal on echocardiography, but few data is available about VPC- associated cardiomyopathy in the strict human cardiology form: dogs with structural heart disease would not be included in this group, whilst dogs with systemic conditions may not be reassessed as frequently once the systemic condition has been successfully managed.

However, these data show the importance of efficient arrhythmia management in patients with frequent ventricular arrhythmia.

Conduction disturbances (Bundle branch block and atrioventricular blocks)

Dyssynchrony can have long-term sequelae in people, causing LV dilation and dysfunction. Cardiac resynchronization therapy is a beneficial procedure performed in people with left

ventricular dyssynchrony due to conduction delays (mainly left bundle branch block) and evidence of heart failure.

Data about the long-term impact of ventricular dyssynchrony in dogs are lacking, but data about the effect of single- versus dual chamber pacemaker implant do not support the notion that single-chamber pacemakers (which could cause dyssynchrony) were associated with a shorter survival. Furthermore, another study including 154 dogs with mainly a single-chamber pacemaker found that only 6% of them developed signs of congestive heart failure after pacemaker implantation.

Chronic bradycardia due to third degree AV block can be associated with left ventricular dilation. Systolic function is most of the time normal, so a DCM phenotype is not often observed. Although half of the dogs needing a pacemaker may have additional forms of heart disease, it very rarely involves a DCM phenotype. In some case series detailing pacemaker implantation in dogs, 10 - 18% of the dogs presented with congestive heart failure, and only 2% of the dogs had a DCM phenotype on initial evaluation.

The presence of congestive heart failure signs (mainly right sided) may be due to bradycardia, and this will be associated with complete resolution of congestive heart failure after pacemaker implantation. Some dogs may however not improve despite pacemaker implantation if myocardial dysfunction is irreversible.

Similarly, some dogs with normal echocardiographic exams before pacemaker-implantation may develop congestive heart failure after pacemaker implantation. The development of congestive heart failure may not be strictly related to LV dyssynchrony or AV block chronicity but is likely the progression of an underlying (undiagnosed) myocardial disease that might have been present alongside with the rhythm disturbance.

Management of dogs with DCM and arrhythmias

The decision to treat arrhythmias (regardless of the clinical status) does not rely on guidelines, but there are some scenarios that allow for more straightforward decisions, and others that may require a case-to-case approach. The aim of treatment should include resolution of clinical signs (if present), reduction of arrhythmia complexity, and possibly reduction in the arrhythmic burden. Antiarrhythmic drugs can however be proarrhythmic, so the decision to treat should balance the expected benefit over possible risks.

It is also important to remember that no antiarrhythmic drug can prevent 100% the risk of sudden cardiac death, possibly only reducing the risk. Most owners would probably feel more relieved if sudden death occurs whilst their pet is receiving an antiarrhythmic drug (which would mean they did everything they could to avoid that), but some others would rather not add an additional medication if they knew that this medication will not certainly prevent death. Client communication and levelling expectations in these cases is important, also in light of the possible trauma associated with discovering that their pet died suddenly, drugs side effects or the need for more frequent tests and rechecks if an antiarrhythmic treatment is started.

In general, in a dog with echocardiographic changes, the presence of numerous ventricular premature beats (VPC count greater than 1000-2000 single VPC) or, more importantly, complex

arrhythmias (defined as presence of couplets or triplets with fast ventricular coupling interval- ie >250 bpm, runs of ventricular tachycardia or bigeminal/trigeminal rhythm, as well as by some authors the presence of R-on-T phenomenon) should prompt the clinician to decide for antiarrhythmic treatment. Some authors would also decide to treat if >50 couplets, triplets, VT, or a fast instantaneous rate (FR) of VPC 280 bpm is observed.

When no complex arrhythmias are noted and there are only single isolated, ventricular premature complexes, the decision (and VPC count cut-off) to offer treatment is more labile. Most people would agree that a VPC count greater than 1000-2000 VPC/24 hr is abnormal and may consider treatment considering the risk of arrhythmia-induced cardiomyopathy and further worsening left ventricular function, but this is less well defined.

When single VPC counts are lower with no complex arrhythmias, the decision to treat should also probably consider patient signalment and an extra-cardiac cause for the arrhythmia should be ruled out: for example, a Dobermann Pinscher is at high risk of sudden cardiac death (presumed arrhythmogenic) and should be at least monitored closely with frequent Holter analysis; similarly, pheochromocytoma or abdominal masses can increase VPC count below 1000 VPC/24hr but above 50-100 or even 300 over 24 hours. Monitoring is probably a reasonable approach with a repeat cardiac evaluation earlier than normal in these scenarios (ie, in 3 months' time), alongside additional testing.

There is limited data about antiarrhythmic treatment for ventricular arrhythmia in dogs. The drugs more commonly used include mexiletine (5-8 mg/kg q 8hr PO) or sotalol (1-2mg/kg q 12hr PO, up-titrated from 1 to 2 if decreased systolic function is observed) or a combination of the two. Mexiletine is probably preferred as it has no effect on systolic function, but it can be difficult to source cheaply in some countries. Amiodarone can also be considered for refractory cases that do not respond to the combination sotalol/ mexiletine. Procainamide and flecainide use have also been reported. Dobermann Pinscher have been reported to be sensitive to amiodarone, with nearly half of them showing reversible increase in hepatic enzymes.

In large-breed dogs predisposed to DCM with atrial fibrillation, the decision on when and how to treat lies on the ventricular rate and the presence or absence of echocardiographic changes. Atrial fibrillation will decrease cardiac output by 20-30% due to the loss of atrial kick, which can have a marked impact in dogs already experiencing systolic dysfunction and lower-than-normal cardiac output.

If lone atrial fibrillation is observed on Holter analysis (median HR similar to a normal dog, ie, below 120 bpm and normal HR variability) or on ambulatory ECG in the hospital environment (HR < 150 bpm), the decision should focus on rhythm control rather than rate control. If no structural changes are noted, DC electrical cardioversion or medical treatment could be considered, however the decision to monitor with no treatment is also not unreasonable if the patient is not showing clinical signs. The response to DC electrical cardioversion is variable and in general less rewarding when structural heart disease is present (even if they are not obvious on echocardiography but are suspected given patient's signalment), and when AF is long-standing (the longer the patient is in AF , the least they will respond to rhythm control). Antiarrhythmic drugs reported to achieve cardioversion include lidocaine (mainly for vagally-induced AF) and amiodarone. In people, sotalol has also been used.

If structural changes are already present and the ventricular rate is moderate to fast (> 120bpm median HR on Holter, or >150 bpm on ambulatory ECG), rate control treatment should be offered. If there are infrequent to no ventricular arrhythmias, digoxin is probably the first choice (0.003 mg/kg to 0.005 mg/kg q 12hr PO), which can then be coupled with diltiazem (1.5-3 mg/kg q 12hr PO modified release, 0.5-2 mg/kg q8hr PO standard formulation) for optimal rate control. Digoxin trough levels need to be rechecked 7-10 days after the treatment is started, 6-8 hours post-pill (target levels: half low of the trough ranges- 0.8-1.2 ng/mL). The presence of frequent ventricular arrhythmia may be a cause of concern if digoxin is started and combination with sotalol or amiodarone could be considered to tackle both issues. Some of the ventricular arrhythmia may however be hypoxia-related and may improve once rate control is achieved.

From the available data in dogs with atrial fibrillation and different underlying cardiac conditions, a longer survival was identified in those that showed a median HR of 120 bpm or lower on Holter analysis. In dogs with DCM though, sometimes such low median HR may be too low to allow to maintain adequate cardiac output, so some dogs may become weak or more lethargic with optimal rate control, and would need adjustments to allow for a slightly higher median HR. Additionally, if the patient is in congestive heart failure, this will falsely increase the median HR due to sympathetic activation. Unless HR is very fast on presentation (i.e., greater than 200 bpm), in a patient presenting in acute CHF, CHF management would be advisable prior to fitting a Holter or deciding whether to start antiarrhythmic treatment.

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