

An Everyday Approach to Canine Degenerative Mitral Valve Disease

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(Cardiology)



Degenerative mitral valve disease (DMVD) is a common canine cardiac disease; approximately 30% of dogs over age 10 possess the characteristic left-sided systolic murmur of DMVD and resulting mitral regurgitation (MR).¹ DMVD is most common in small breeds, with some breeds being highly predisposed. For example, virtually all Cavalier King Charles spaniels will develop DMVD during the course of their lifetimes.

Developing a standardized approach to patients with DMVD can help the clinician concentrate on the most important diagnostic aspects of disease as well as provide useful information about monitoring and prognosis. This overview will focus on the role of the physical examination and thoracic radiographs and how information from these tests can aid in the everyday diagnosis and management of DMVD.

DIAGNOSIS OF DMVD

Diagnosis of DMVD is one of the easiest clinical aspects of the disease. A presumptive diagnosis can be made on the basis of patient signalment and physical examination, wherein the hallmark finding is an adult small-breed dog with a left-sided systolic murmur. In these instances, it is reasonable to assume that the source of the murmur is MR and hence, DMVD is present. Once a diagnosis has been made, the next question is: what is the severity of DMVD?

ASSESSING SEVERITY OF DMVD

Pathophysiology

As DMVD worsens, the valve leaflets degenerate, resulting in worsening MR. The reduction in forward cardiac output triggers activation of several neurohormonal pathways, including the renin-angiotensin-aldosterone system and sympathetic nervous system. For instance, reduction in renal perfusion results in renin release from the macula densa, which results in downstream production of angiotensin II via angiotensin-converting enzyme (ACE).

Angiotensin II and its related molecule aldosterone act to conserve sodium and water and expand plasma volume. In this way, cardiac output is maintained and there is an increase in cardiac stroke volume and, over time, ventricular size. Thus, the degree of volume retention and subsequent eccentric cardiac hypertrophy is a marker of MR severity.

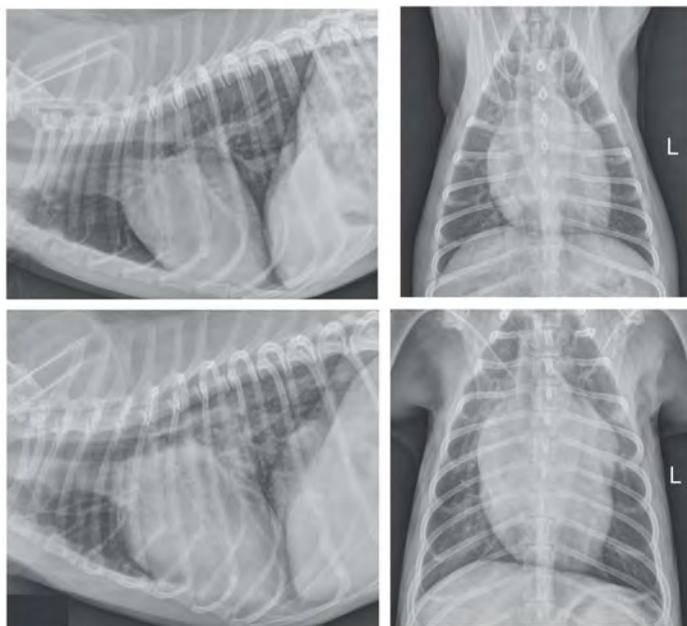


Figure. Right lateral and dorsoventral radiographs from a 12-year-old male Maltese with DMVD, demonstrating progressive left heart enlargement. The lower row of radiographs were taken approximately 12 months after the upper row. Based on the marked change in heart size over this period of time, the risk of future congestive heart failure was high and increased owner monitoring for early signs of heart failure was warranted. The owner began keeping a log of the dog's respiratory rate and effort and was instructed to contact the veterinarian if the rate began to increase above the baseline value. Recheck physical examination and radiographs in 3 months were recommended.

View the [Guidelines for the Diagnosis and Treatment of Canine Chronic Valvular Heart Disease \(2009\)](https://onlinelibrary.wiley.com/doi/10.1111/j.1939-1676.2009.0392.x/full) at onlinelibrary.wiley.com/doi/10.1111/j.1939-1676.2009.0392.x/full.

Thoracic Radiography

Measuring heart size is extremely useful in assessing severity of DMVD (**Figure**). Thoracic radiography is a relatively simple and cost-efficient tool for assessing heart size and identifying the presence of pulmonary venous distention or edema.

Measurement systems, such as the vertebral heart size (VHS) system, are useful in reducing inter-observer variability and tracking longitudinal changes in a patient's heart size. The VHS technique has been well-described.² Normal VHS size in dogs is < 10.7; severe heart enlargement and hence, severe DMVD, is usually considered to be > 12.

I recommend performing baseline thoracic radiographs to assess heart size on every dog with a newly discovered murmur. Baseline radiographs establish the initial degree of disease severity and assess rate of disease progression during subsequent follow-up (recheck radiographs should be performed 6–12 months after initial detection and are often repeated every 3–12 months depending on rate of progression).

Electrocardiography & Echocardiography

The value of electrocardiography (ECG) and echocardiography (echo) in the diagnosis and monitoring of DMVD varies from case to case. Clearly, ECG is the gold standard for assessment of arrhythmias, but ECG criteria for heart enlargement (eg, increased R wave amplitude indicating left ventricular enlargement) are relatively insensitive.

Echo is the gold standard for assessment of cardiac structure and function, but in most cases of DMVD, especially those that are relatively mild, the information obtained by echo rarely adds to what was already known from the physical examination and thoracic radiographs. While echo is helpful in some cases to detect concurrent cardiac disease, such as tricuspid regurgitation or pulmonary hypertension, lack of echo should not affect the ability to make an initial assessment of disease severity based on radiographic heart size.

In most cases of DMVD, appropriate diagnostic, monitoring, and therapeutic decisions can be made without echo provided that radiographs are performed.

MONITORING PROGRESSION OF DMVD

It is reassuring to remember that most dogs with DMVD will not develop enough MR to cause congestive heart failure or clinical signs.³ The preclinical course of disease can be many years and only a minority of dogs will progress to the point of heart failure.

Classifying Patients

By performing longitudinal examinations of heart size, clinicians can gradually stratify dogs into (1) those that have slowly progressive disease and are unlikely to develop signs versus (2) those with more rapidly progressing disease that are at risk for future heart failure. In general, it is very uncommon for dogs to develop congestive heart failure with VHS < 11.5.

High-Risk Patients

Once VHS \geq 11.5, both absolute VHS and rate of increase in VHS are linked to risk of heart failure. In these patients, more frequent rechecks and increased owner vigilance for subtle signs of early heart failure, such as mild increase in respiratory rate or effort while at rest, decreased activity tolerance, or lethargy, is recommended. Previous studies⁴ have shown that a rate of VHS increase > 0.25 vertebra over a 3-month period suggests that risk for heart failure is high.

SLOWING PROGRESSION OF DMVD

Unfortunately, there are no proven interventions that slow the rate of DMVD progression or significantly delay the onset of heart failure in preclinical DMVD.

Role of Ace Inhibitors

While 2 large studies^{5,6} failed to demonstrate that ACE inhibitors significantly affected the primary endpoint of first-onset heart failure, the majority of cardiologists and members of the consensus panel on DMVD recommend ACE inhibitors prior to onset of heart failure, after cardiac enlargement is detected.⁷ In contrast, most cardiologists do not advocate use of furosemide prior to onset of congestive heart failure.

Monitoring & Prompt Therapy

Other medications are being assessed in ongoing clinical trials, but until these trials are completed, I recommend that dogs at high risk for heart failure are simply monitored more closely, both by the clinician and owner. Detection of subtle signs of heart failure as previously mentioned followed by prompt initiation of conventional therapy can prevent development of severe and life-threatening heart failure that requires emergency visits, intravenous treatment, and prolonged hospitalization.

TREATMENT OF DMVD

Clinical Signs

In dogs with severe DMVD, progressive MR and chronic neurohormonal activation ultimately leads to congestion in the form of pulmonary edema. Dogs often present for respiratory signs, ranging from:

- Mild increase in respiratory rate
- Increased effort with abdominal component
- Fulminant respiratory distress.

Coughing is often considered a sign of heart failure, yet most dogs with the isolated complaint of cough

suffer from primary airway or respiratory disease as opposed to heart failure. Etiology of the cough can be differentiated by paying close attention to:

- Radiographic heart and pulmonary vein size
- Type and distribution of pulmonary pattern
- Evidence of upper airway collapse.

Medical Therapy

Once a diagnosis of heart failure is made, 4 medications comprise the cornerstones of treatment for heart failure secondary to DMVD.

1. **Diuretics:** The most commonly prescribed diuretic is furosemide, which is typically administered at **2 to 3 mg/kg/day PO** in dogs with mild heart failure and up to **4 to 6 mg/kg/day PO** in dogs with more severe signs. Boluses of intravenous furosemide, **2 to 4 mg/kg IV Q 2 to 4 H**, are used for rapid diuresis in dogs with life-threatening failure.
2. **ACE Inhibitors:** ACE inhibitors directly address activity of the renin–angiotensin–aldosterone system and work in tandem with diuretics to control congestion. Enalapril, **0.5 mg/kg PO Q 12 H**, is the most commonly prescribed ACE inhibitor and is generally well-tolerated. Renal biochemistry values and electrolytes are usually checked before and 3 to 5 days after starting diuretics and ACE inhibitors or changing doses.
3. **Inodilators:** The inodilator agent pimobendan counteracts DMVD and MR through 2 different mechanisms: the first through calcium sensitization and increased myocardial contractility; the second through vasodilation. Pimobendan improves survival in dogs with heart failure secondary to DMVD and when compared directly against benazepril, extends longevity by almost 2-fold (median survival, 4 months longer in pimobendan group) over ACE inhibitors.⁸

These 3 medications—furosemide, pimobendan, and enalapril—comprise what cardiologists refer to as triple therapy and are considered standard of care for heart failure secondary to DMVD. In my practice, if owners are constrained to administering less than 3 medications, such as instances in which the dog is difficult to pill, the priority of administration is furosemide, pimobendan, and then enalapril.

4. **Aldosterone Antagonists:** The aldosterone antagonist, spironolactone, is the fourth agent commonly used for treatment of heart failure. It provides additional blockade of the renin–angiotensin–aldosterone system and produces modest diuresis. In a European study, spironolactone, **2 mg/kg PO Q 24 H**, was shown to significantly increase survival in dogs with heart failure due to DMVD⁹; it is currently approved for use in dogs in the European Union. Cardiologists have been slower to adopt spironolactone as standard therapy, primarily due

to the somewhat atypical nature of the study cohort. Additional studies to better clarify the utility of spironolactone are currently ongoing.

IN SUMMARY

- The diagnosis of DMVD is relatively simple and based on auscultation and signalment.
- Thoracic radiography is an excellent modality to ascertain baseline and progressive heart size, which directly correlates to severity of disease. In addition, in dogs with clinical signs, radiographs help distinguish cardiac from respiratory causes.
- Dogs with large hearts and rapidly increasing heart size are at high risk for heart failure; increased owner and veterinary monitoring is warranted.
- There are no drugs that have been unequivocally proven to slow progression of disease in the pre-clinical stage.
- If and when heart failure develops, therapy with furosemide, pimobendan, and an ACE-inhibitor (+/-spironolactone) should be instituted with attention paid to renal blood analysis, patient respiratory effort and rate, and activity level. ■

Disclosure Statement

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ACE = angiotensin-converting enzyme;
DMVD = degenerative mitral valve disease;
ECG = electrocardiography; echo = echocardiography;
MR = mitral regurgitation; VHS = vertebral heart size

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IN SUMMARY

You now have the tools to create and leverage social media and maximize client communication. Step by step you will grow in confidence, strengthen your plan and message, and take advantage of the opportunity the future holds with regard to social media resources. ■

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Sheila Grosdidier, RVT, PHR, is a partner/consultant with Veterinary Management Consultation, Inc, and specializes in staff training/utilization, client service excellence, and technical proficiency within veterinary practices. She is also an editorial advisory board member for Today's Veterinary Practice.

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