Managing a Canine Patient With Heart Failure Before You Get the Echo

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Introduction

Echocardiography will give definitive information about structure and function of the heart and is therefore included in the evaluation of patients with heart disease. Obtaining a diagnostic echocardiogram involves image acquisition by a skilled operator using specialized equipment and therefore may be unavailable when heart disease is first diagnosed by the practitioner, or it may never be available due to constraints including access to someone skilled in the technique, or cost.

The absence of this data, however, does not preclude the diagnosis and treatment of patients with heart disease. The ACVIM consensus statement on chronic valve disease stated that in small-breed dogs with typical murmurs, an echocardiogram is recommended to answer specific questions regarding either cardiac chamber enlargement or the cause of the murmur if those questions are not adequately answered by thoracic radiography, emphasizing thoracic radiography as an essential diagnostic test in these patients. Definitive diagnosis of dilated cardiomyopathy would require echocardiographic evaluation, but these patients may present in congestive failure, compelling the practitioner to make decisions about lifesaving therapeutic interventions before an echocardiogram is performed.

An important component of managing a patient with heart disease is an understanding of the nature of that disease, how severe that disease is, and how likely the disease is contributing to the clinical signs that are seen. The ACVIM consensus panel and the Cardiac Education Group (www.cardiaceducationgroup.org (http://www.cardiaceducationgroup.org/home/)) have recently developed a practical grading system that allows us to organize the continuum of heart disease into stages to facilitate this understanding. The staging system allows us to develop stage-specific recommendations for both diagnosis and treatment.

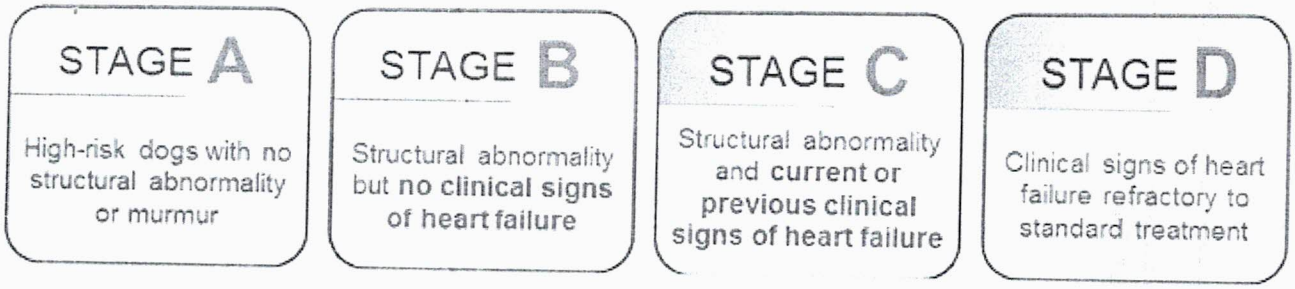
Clinical Tools

Practitioners have three powerful tools that can be used to make confident judgments about the cardiovascular status of a patient:

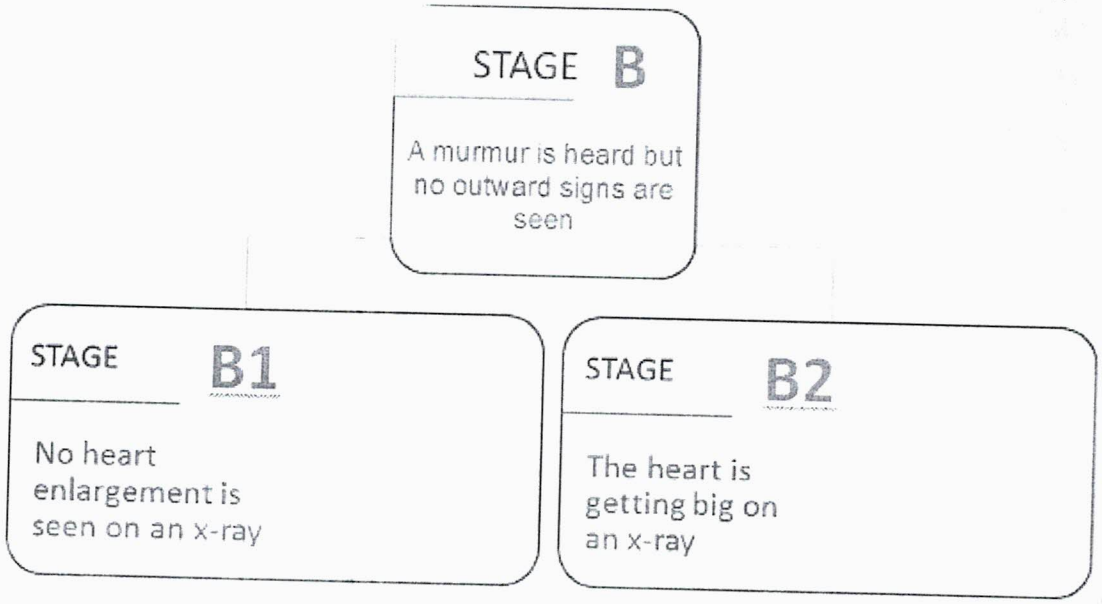
1. A solid history and a thorough physical examination
2. Auscultation of the heart and lungs
3. Thoracic radiography

We will see how these tests allow us to stage our patients and thus make informed decisions about their management, even before we secure a definitive diagnosis with echocardiography.

The ACVIM Staging System



Stage B is further split:



The time spent at each stage varies from dog to dog, but many dogs remain in stage B for 2–3 years.

Stage-Based Case Management

We will not spend a lot of time on stage A, as these patients have no disease but are those at risk for developing it. Management typically involves screening to identify the presence of disease as soon as it develops.

Stage B, i.e., disease is present (a murmur is heard) but no clinical signs have developed, is an important stage for a number of reasons. Firstly, the patient may spend several years in this stage. Secondly, B2 is the phase immediately prior to the development of clinical signs, and recognizing the transition to stage C as soon as possible will facilitate the timely introduction of medications that will improve both the quality and quantity of that patient's life. Below is one suggested management approach for patients once they enter stage B.

Stage B (i.e., Murmur But No Associated Clinical Signs)

Baseline: Grade III?

1. Chest film (measure the vertebral heart score - VHS)
2. Blood work
3. Blood pressure
4. NTproBNP
5. ECG

Stage B1 (Murmur, No Changes on Chest Radiograph, No Clinical Signs)

No treatment is recommended.

1. Recheck within the next year (six months if a new client). That recheck will include:
    a. Another chest radiograph to look for any sign of progression of disease (VHS)

b. Biochemistry
c. Measure BP

2. Client education
   a. Early warning signs - don't discount them
      i. Coughing
      ii. Changes in breathing - RRR > 30
      iii. Difficulty breathing
      iv. Shortness of breath
      v. Changes in behavior
      vi. Lack of energy/tires easily/lethargy
      vii. Exercise intolerance/fainting
      viii. Restlessness, especially at night
      ix. Changes in appetite
   b. Counting a pet's sleeping breathing rate at least once a month and keeping a log
3. Set client expectations
   a. A chronic but progressive disease
   b. They can do a lot to help us manage the disease going forward
      i. Vigilance to clinical signs
      ii. Assessing respiratory rate
      iii. Adhere to follow up schedule

Stage B2 (Murmur with Changes on Chest Radiograph, No Clinical Signs)

~~**No treatment is recommended at this time**~~, **although there is debate about therapeutic interventions in 'late' stage B2. We are entering a time of increased vigilance.** + PIMOBENDAN (EPIC TRIAL)

As B1 but:

1. Increase the recheck frequency to at least every 6 months (including chest radiograph with VHS)
2. Increase the frequency of checking respiratory rate to at least **weekly**

Stage C

As soon as clinical signs develop, a patient enters stage C. Identifying that transition can be challenging. If we have been following a patient as described above, we will have information to support a diagnosis of heart failure, and we will be sensitive to signs that might otherwise be discounted as signs of aging - such as exercise intolerance, lack of interest in playing, being unable to rest comfortably at night - which are, in fact, associated with heart failure.

For example, a patient presents because the owner is concerned that the pet lacks energy. We have followed this patient through stages B1 and B2, and in that time their VHS has gone from 10.7 to 12, and reviewing the owner's log of sleeping respiratory rate shows it has increased recently from a consistent 19 to 28. Repeat chest films show evidence of pulmonary venous distension and an increased interstitial pattern (suggestive of pulmonary edema), but they are inconclusive. Based on all the evidence that we have accumulated, the index of suspicion that this patient's vague signs are due to heart failure and therefore require treatment is much higher.

The ACVIM consensus panel made a recommendation for managing patients with heart failure. These patients should immediately receive a combination of:

1. Furosemide
2. Pimobendan (Vetmedin)

3. ACE inhibitor

These medications are started concurrently, and a patient should be reevaluated 7–10 days after therapy begins or discharge from the hospital, assuming that the patient is stable or continues to improve.

Medication Dosing

- The dose of furosemide depends on the severity of signs and may be as high as 6 mg/kg initially. Maintenance doses of furosemide are typically 1–2 mg/kg BID to TID.
- Pimobendan is administered at 0.25–0.3 mg/kg BID.
- The dose and frequency of the ACE inhibitor will depend on the drug selected.

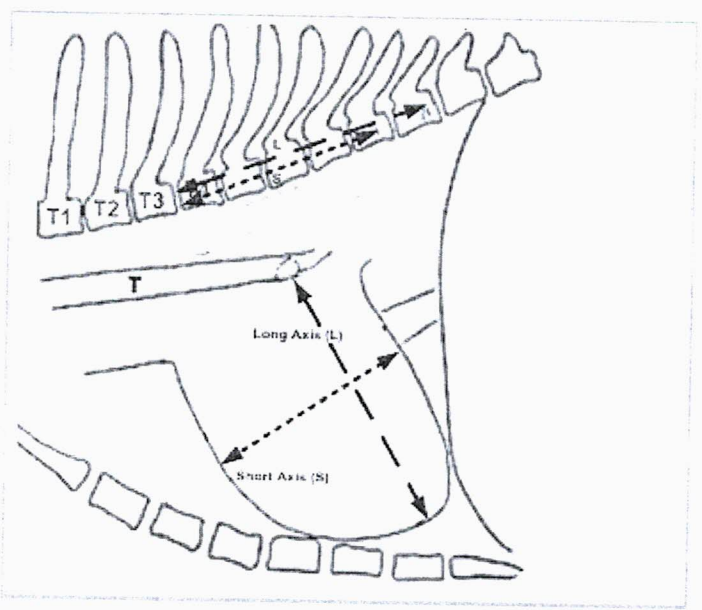
Referral to a Cardiologist

If referral to a cardiologist is possible, it is appropriate at any stage. The cardiologist is trained to help you and your client manage patients with cardiac disease.

Measuring Vertebral Heart Score

To calculate the vertebral heart score, measure the longest axis of the heart (L) from the bottom of the tracheal bifurcation to the cardiac apex (see diagram below). Then transfer this measurement to the vertebrae, starting at the cranial edge of T4 and count the number of vertebrae within that span. For fractions of a vertebral body, estimate this to within 0.1 of a vertebral body unit (i.e., 5.5). Starting at the cranial edge of T4 once more, take the same measurement for the short axis of the heart (S), at 90 degrees to the long axis at the point where the left atrium joins the left ventricle (or the ventral border of the caudal vena cava if this is not visible) and again count the number of vertebrae that fall within the span. Then add the 2 numbers together to get the total VHS.

Each patient makes its own best control. Always use the same technique when assessing a patient longitudinally. As this technique is somewhat subjective, we should look for changes of at least 0.5 of a vertebral body unit.



Normal

Dogs 8.5-10.7

Cats 6.7-8.1

Conclusion

Application of the grading system discussed allows us to manage a patient through the continuum of its disease. The tools we have available to us on a daily basis, including thoracic radiography, allow us to appropriately stage a patient and thus make judgments about therapy, even before we have an echocardiogram available. The ability to track progressive changes

radiographically will build confidence that clinical signs are indeed those of heart failure. When heart failure is diagnosed, the patient should be started immediately on furosemide, pimobendan (Vetmedin), and an ACE inhibitor.

Speaker Information

(click the speaker's name to view other papers and abstracts submitted by this speaker)

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RECOMMENDATIONS
conversations with a cardiologist

CARDIAC EDUCATION group

December 2016

The EPIC Trial: Pimobendan in Preclinical Myxomatous Mitral Valve Disease

What is the EPIC trial?

The EPIC trial was a large randomized, multinational, multicenter study designed to investigate the effect of Vetmedin® (pimobendan) on the progression of myxomatous mitral valve disease in small breed dogs. The trial acronym (EPIC) stands for the "**E**valuation of **P**imobendan **I**n dogs with **C**ardiomegaly caused by preclinical myxomatous mitral valve disease." The results of this trial were first published in the Journal of Veterinary Internal Medicine in September, 2016 (link to manuscript).[1]

What was the purpose of the EPIC trial?

The purpose of the trial was to investigate whether or not pimobendan delayed the onset of congestive heart failure (CHF) or cardiac-related death/euthanasia in dogs with asymptomatic myxomatous mitral valve disease and cardiac enlargement, as compared to placebo. Additionally, the effect of pimobendan on all-cause mortality in this population was evaluated as a secondary endpoint.

What dogs were enrolled in the EPIC trial?

The EPIC trial enrolled 360 dogs with preclinical myxomatous mitral valve disease. To enter the study, cardiac enlargement was required based on the criteria above, meaning all dogs were ACVIM stage B2 with at least moderate cardiac enlargement (link to ABCD handout). Specifically, the inclusion criteria for the trial were:

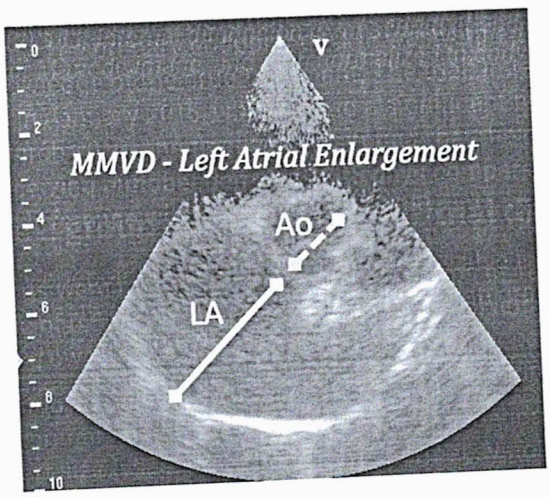
- dogs over 6 years of age,
- with a body weight between 4 and 15kg,
- systolic heart murmur characteristic of mitral regurgitation (maximal intensity at the left cardiac apex) of moderate to high intensity (greater than or equal to grade 3 out of 6),
- cardiac enlargement, defined by:
  - vertebral heart size (VHS) had to equal or exceed 10.5[2]
  - left atrial-to-aortic root short-axis ratio (Figure 1) as measured by 2-dimensional echocardiography had to equal to or exceed 1.6, according to the Swedish method[3]
  - normalized left ventricular internal dimension in diastole (LVIDDN; Figure 1) had to equal or exceed 1.7[4]
    - The allometric formula for calculation of LVIDDN is:

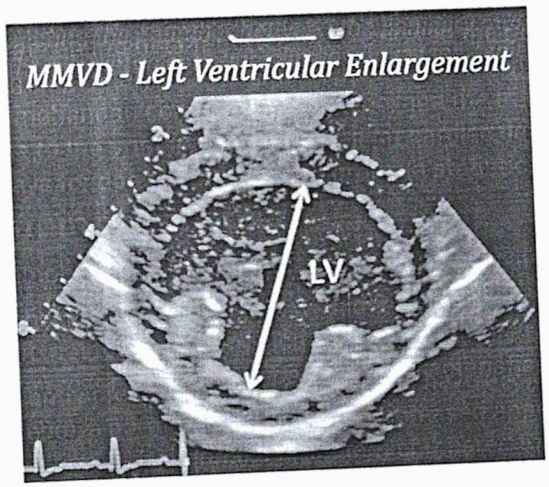
$$\text{LVIDDN} = \frac{\text{measured LVIDd (cm)}}{\text{Weight (kg)}^{0.294}}$$

    - The approximate measured value of LVIDD that delivers a LVIDDN greater than or equal to 1.7 by body weight are shown in Table 1.

Dogs were excluded from the trial if they had other life-threatening disease, clinically significant arrhythmias, severe pulmonary hypertension, prior CHF, or had previously received cardiac medications. Dogs were enrolled in the study from October 2010 to June 2013 and then followed until the trial ended in March 2015.

FIGURE 1





The left atrium (LA) to aortic (Ao) ratio (LA size divided by Ao diameter) is measured at maximal LA size (end of ventricular systole) from a short-axis image. The Ao is measured along the junction of the non-coronary and left coronary valvar sinuses, while the LA is measured along this same line from inner edge to inner edge, without extending the line into a pulmonary vein. The left ventricular internal dimension is measured at end diastole, bisecting the chamber between the papillary muscles on either a 2D or M-mode image, and then normalized to body weight by the allometric equation shown above (see Table 1).

Were the 2 groups within the EPIC trial different from one another?

The 360 dogs were randomly divided into 2 groups of 180 each; one group received pimobendan at the labeled dose (target dose of 0.5 mg/kg/day divided into 2 doses) and the other received a placebo that was identical in appearance. Both the dog owners and the veterinarians caring for each dog were not told which medication the dogs were receiving. An analysis of the groups at the time of enrollment found no difference for all pertinent baseline characteristics.

What were the results of the EPIC trial?

The dogs were monitored and re-evaluated at 1 month and then every 4 months for the duration of the trial. If clinical signs developed at any point in the trial, the dogs underwent clinical evaluation to determine if congestive heart failure was present. The primary endpoint of CHF was verified by a separate endpoint committee who reviewed the radiographs without knowledge of the drug treatment. The trial underwent an interim analysis by an independent monitoring committee in January 2015, which found a significant benefit of pimobendan compared to placebo, and the trial was ended in March 2015. **The final analysis found that the median time to the primary endpoint (CHF or cardiac death) for dogs receiving pimobendan was 1228 days compared to 766 days for dogs in the placebo group (a statistically significant difference with a P value of 0.0038).**

A secondary endpoint of all-cause mortality was also in favor of pimobendan, with a median time from inclusion in the study to death of 1059 days for the pimobendan group compared to 902 days for the placebo group (a statistically significant difference with a P value of 0.012).

When the study results were analyzed, the variables that independently predicted which dogs would have the longest time to the primary endpoint were receipt of pimobendan (vs. placebo), a normal appetite (vs. a decreased appetite), smaller heart size (vs. a larger heart), and normal systolic heart function (% fractional shortening) compared to an increased % fractional shortening. If the echocardiographic variables were excluded, pimobendan (vs. placebo), slower heart rate on initial examination, normal systolic arterial blood pressure (vs. low normal blood pressure), and smaller radiographic heart size (VHS) predicted a longer time to the primary endpoint. Sporadic adverse events, primarily gastrointestinal in nature, were noted throughout the study period with equal prevalence between groups.

What other interesting findings arose from the EPIC trial?

A secondary endpoint was the time to a composite endpoint of left-sided CHF, euthanasia or death for noncardiac reasons, initiation of non-CHF medications (e.g. cough suppressant), or non-confirmed CHF. This analysis was meant to better understand the real-world effect of the drug beyond and including the primary endpoint. **This secondary endpoint was also different between groups in favor of pimobendan at 640 days compared to 406 days in the placebo group at a P value of <0.001.**

Why was the trial stopped prematurely?

At first glance, it seems odd that the trial was ended before the expected timeframe. However, the decision to do so was made before the trial started if the independent interim analysis committee found certain conditions had been met. The rationale for this approach is well established in human medicine; namely, if there is a substantial safety concern that is different between groups, it is better to know sooner and address it. Similarly, if there is a clear outcome advantage for one group compared to the other, it is important to allow all dogs to receive this benefit as soon as it is clearly determined to be real. The final analysis confirmed the conclusion from the interim analysis.

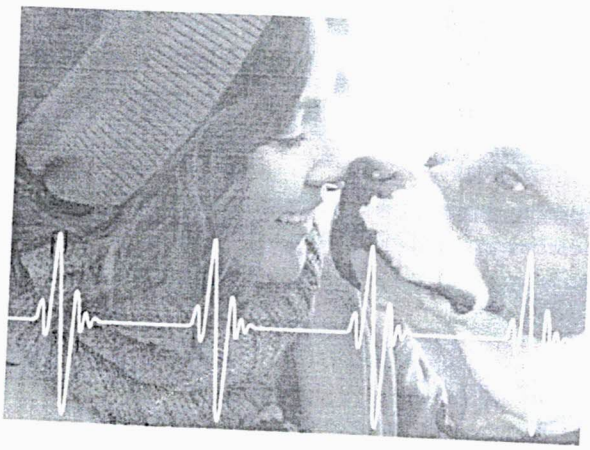


TABLE 1

Dog Weight (kg)	LVIDD Measurement Equal to LVIDDN of 1.7	Dog Weight (kg)	LVIDD Measurement Equal to LVIDDN of 1.7
1	17.0	13	36.1
1.5	19.2	14	36.9
2	20.8	15	37.7
3	23.5	16	38.4
4	25.6	17	39.1
5	27.3	18	39.8
6	28.8	19	40.4
7	30.1	20	41.0
8	31.3	21	41.6
9	32.4	22	42.4
10	33.5	23	42.7
11	34.4	24	43.3
12	35.3	25	43.8

Ventricular enlargement was defined as a normalized left ventricular internal dimension in diastole (LVIDDN) greater than 1.7. For dogs of various size, the actual LVIDD measurement that equals this cut-off value is shown.

What is the significance of these results to the treatment of mitral valve disease?

For the first time in canine medicine, this trial has found a statistically-significant beneficial effect from the administration of a medication in dogs with asymptomatic myxomatous mitral valve disease and substantial cardiac enlargement. **For dogs that meet all the inclusion criteria of the EPIC trial, the data suggest that dogs that receive pimobendan will, on average, remain asymptomatic for ~15 months longer and live for ~5 months longer when compared to dogs that do not.** Analyses of the data tell us the drug appears safe to administer in this population compared to placebo. Overall, the dogs receiving pimobendan were less likely to require initiation of additional cardiopulmonary medications in the preclinical period compared to those taking the placebo.

How should I apply these findings to my clinical practice?

This is the most difficult question to answer about this trial and a matter that remains debated by veterinary cardiologists. We know that myxomatous mitral valve disease is the most common heart disease of dogs and many dogs with the disease will not progress to CHF. Because of this, **the CEG does not recommend administration of pimobendan to every dog with a heart murmur.**

It is the opinion of the CEG that dogs with asymptomatic mitral valve disease be examined closely to identify those dogs that may benefit from pimobendan. From this trial, we know that the dogs with substantial cardiac enlargement will benefit from pimobendan. Thus, **documentation of cardiac enlargement is critical to decide whether treatment is appropriate for a given patient (see algorithm).**

The strictest interpretation of the trial would suggest that all small breed dogs with a left apical systolic murmur of grade 3/6 or louder should have radiographs taken and an echocardiogram performed to determine if substantial cardiac enlargement is present and pimobendan therapy warranted. The practical impacts of this approach are challenging, since not all dogs with a murmur of this type will meet the EPIC inclusion criteria for cardiac enlargement, nor will all dog owners be willing to have these diagnostic tests performed or be able to afford lifetime pimobendan therapy.

How does the CEG advise practitioners to apply these results?

- Documentation of substantial cardiac enlargement (Figure 1 & algorithm) is critical to determine whether or not to start pimobendan in dogs with preclinical mxyomatous mitral valve disease.
- If substantial cardiac enlargement is not documented by echocardiography and the VHS is <10.5, reevaluation is advised in 12 months.
- If the VHS is between 10.5 and 11.5 and an echocardiogram is not performed or does not exceed enlargement criteria, reevaluation is advised in 6 months.
- If an echocardiogram is not available and only thoracic radiographs are performed, the CEG recommends starting pimobendan in dogs with heart murmurs of grade 3/6 or louder only when the VHS exceeds 11.5 vertebral bodies or an incremental increase of greater than 0.5 vertebral bodies per 6 months is accurately documented.
- See algorithm for a schematic representation of this approach.

What about the co-administration of an Angiotensin Converting Enzyme Inhibitor (ACE-inhibitor) or an aldosterone antagonist?

The EPIC trial did not evaluate concurrent use of an ACE-inhibitor (e.g. enalapril or benazepril) or aldosterone antagonist (i.e. spironolactone) in the setting of preclinical myxomatous mitral valve disease. Interpretation of prior veterinary trials (SVEP[5] and VETPROOF[6]) investigating enalapril in preclinical mitral valve disease dogs failed to achieve a statistically-significant benefit compared to placebo. However, in VETPROOF some secondary endpoints were in favor of enalapril and on average there was a modest delay in the onset of CHF by about four months.[6] This delay is clearly inferior to that achieved in the EPIC trial using pimobendan, but we do not know if using both drugs would provide any incremental benefit. In short, cardiologists have not reached a consensus on when to initiate ACE inhibition or aldosterone antagonism at this stage of MMVD. The consensus of the CEG is to add an ACE-inhibitor to pimobendan when severe cardiac enlargement has occurred or if a large incremental change in heart size over time (e.g., greater than 0.5 vertebral bodies per 6 months) is documented despite pimobendan therapy. We also recognize that the cost of medication in a country can influence this decision. Overall, the CEG recommends that you discuss this issue with cardiologists within your referral area and continue to follow the literature for clinical trial evidence.

What about dogs in stage B2 that have been chronically receiving ACE-inhibitors or aldosterone antagonists?

The CEG recommends reevaluation of these dogs with imaging to investigate the degree of cardiac enlargement, as discussed above and in the algorithm. If the criteria discussed in this document are then met, administration of pimobendan is recommended with continuation of the previously-prescribed medication.

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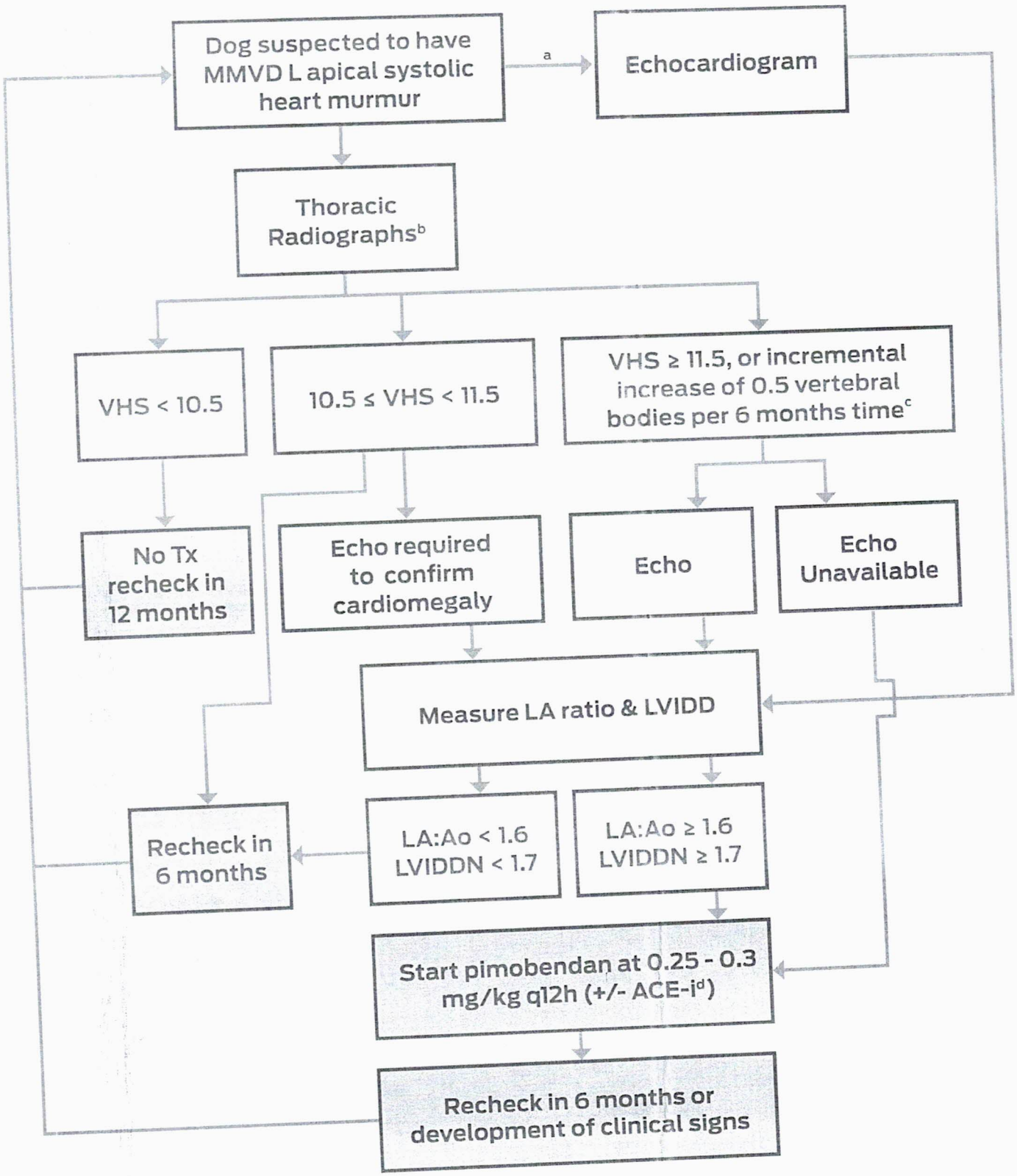
The CEG is sponsored by an educational grant from Boehringer Ingelheim Vetmedica, Inc., and IDEXX Laboratories.

ALGORITHM

CEG Recommendations for Therapy in Preclinical Myxomatous Mitral Valve Disease



Algorithm Footnotes

a. Diagnostic imaging becomes especially meaningful in dogs when the murmur is no longer soft or focal. Imaging studies are always recommended for evaluation of a moderate to loud murmur of mitral regurgitation (grade 3/6 or louder). Echocardiography is also indicated if the cause of the heart murmur is in doubt, as with a younger dog who might have congenital heart disease or in a dog with a breed-risk for dilated cardiomyopathy.

b. While both thoracic radiography and echocardiography can be used to identify cardiac enlargement, the echocardiogram is generally preferred if the cause of the systolic murmur is uncertain and to more precisely measure chamber sizes. If there is evidence of cardiomegaly/remodeling on the echocardiogram, then radiographs are also suggested to measure vertebral heart size (VHS), evaluate the cardiac chambers subjectively, and obtain a "baseline" for the appearance of pulmonary vasculature and parenchyma. In situations where the client declines echocardiography, the thoracic radiograph path can be followed in the primary care practice. ACVIM guidelines recommend **both** imaging methods for optimal evaluation of a dog with suspected MMVD.

c. The EPIC trial did not investigate a cutoff for cardiac enlargement by VHS alone at which to start pimobendan therapy. This algorithm represents the consensus of the CEG to initiate therapy when echocardiography is not available, but is not based on evidence.

d. Cardiologists have not reached a consensus on when to initiate angiotensin converting enzyme (ACE) inhibition or aldosterone antagonism at this stage of MMVD. The consensus of the CEG is to add an ACE-inhibitor to pimobendan when severe cardiac enlargement has occurred or if a large incremental change in heart size over time.

Cardiac Education Group
CardiacEducationGroup.org

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Echocardiography versus Radiography for the Diagnosis and Management of Heart Disease

ACVIM 2017

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Introduction

Thoracic radiography and echocardiography are both invaluable tools in the diagnosis and management of heart disease. Although there is certainly overlap in the information obtained by these two modalities, they should be viewed as complementary, rather than competing tools.

Echocardiography provides two general categories of information about the heart: morphology and function. Morphology of the heart provides information such as chamber dimensions, appearance of the valves, evidence of effusions, and presence of tumors. Morphology is assessed using 2D and M-mode. Systolic and diastolic function is assessed using a combination of these modes and Doppler imaging.

Unfortunately, ultrasound has extremely limited utility for evaluation of air-filled structures, thus thoracic radiography is an invaluable diagnostic tool for evaluation of pulmonary parenchyma and vasculature. Thoracic radiographs also provide a good overall sense of the size of the cardiac silhouette relative to the thorax. Although viewers often use the terms "heart" and "cardiac silhouette" interchangeably, it is important to remember that this structure on a radiograph simply represents the pericardium and its contents. If the cardiac silhouette is interpreted as enlarged, then the next question becomes "what is causing the enlargement"?

Thoracic Radiography Reviewed

Alterations in the cardiac silhouette on thoracic radiographs are useful for suggesting specific chamber enlargements. On lateral views, the cranial aspect of the cardiac silhouette is composed of the right atrium and ventricle and the caudal aspect is composed of the left atrium and ventricle. The cranial dorsal region, the "cranial cardiac waist," is composed of three overlapping structures: the right atrium, pulmonary artery, and aorta. Loss of the cranial cardiac waist can be due to enlargement of any of these structures and evaluation of the orthogonal view is necessary to determine which is involved. The caudal dorsal region, the "caudal cardiac waist," is composed only of the left atrium. In dogs, serial radiographs to assess for progressive left atrial enlargement are very useful for monitoring for progression of degenerative mitral valve disease. In cats, left atrial enlargement is more easily appreciated on the DV/VD projections. Left atrial enlargement causes widening of the heart base, commonly referred to as a valentine-shaped or shield-shaped heart.

A clock-face analogy is often used to identify structures on the DV/VD view. The aortic arch is approximately at 11:00–1:00; the pulmonary artery is from 1:00–2:00; the left auricle is from 2:00–3:00; the left ventricle is 3:00–6:00; the right ventricle is 6:00–9:00; and the right atrium is 9:00–11:00. The left atrium is central and caudal to the bifurcation of the mainstem bronchi.

Various methods have been developed to quantify enlargement of the cardiac silhouette using thoracic radiographs. On lateral views, the cranial-to-caudal width of the heart should span less than 2.5 intercostal spaces for deep-chested dogs, and less than 3.5 intercostal spaces for barrel-chested dogs. The height of the heart from the carina to the apex should

be ≤2/3 of the height of the thoracic cavity. On DV/VD views, the maximal width of the heart should be ≤1/2 the width of the hemi-thorax at the 9th rib. These are relatively subjective measures, and a vertebral heart scale (VHS) system was developed to try to provide a more objective determination of cardiac size. Using the lateral view, the long and short axes of the heart are measured. The measurements are then superimposed over the vertebral column, both starting at the cranial edge of the fourth thoracic vertebral body (T4), and the number of vertebrae covered is summed. Normal dogs have a VHS of 8.5–10.6 although there are some breed variations with Boxers, Dachshunds, Labradors and Cavaliers having normal VHS up to 12. Normal cats have a VHS of 7.2–7.8. I routinely use a combination of methods to interpret cardiac size.

Abnormal lung patterns are described as interstitial, alveolar, or bronchial. Frequently, a combination of patterns is present in the same patient. An interstitial pattern appears as an increase in the overall opacity of the lungs. All normal structures are still visible, but the margins are hazy. Interstitial patterns can be structured (e.g., nodular) or unstructured. An alveolar pattern is characterized by complete loss of margins of the vasculature, and the presence of air bronchograms. An air bronchogram is caused by fluid or cells filling the terminal alveoli, resulting in a uniform soft tissue opacity surrounding the radiolucent bronchus. Alveolar patterns occur with pulmonary edema, pneumonia, hemorrhage, atelectasis, lung lobe torsion, or neoplasia. A bronchial pattern is caused by thickening of the airways, either from inflammation or mineralization of the airways themselves (e.g., asthma, bronchitis) or interstitial fluid tracking along the airway. Radiographically, this appears as "donuts" and "tramlines."

Changes in the pulmonary vasculature are indicative of the underlying pathology. Enlargement of the pulmonary arteries is implicative of pulmonary hypertension. Enlargement of the veins is a hallmark feature of congestive heart failure, and is the result of increased cardiac preload. Symmetrical enlargement of both the arteries and veins indicates a combination of pulmonary hypertension and increased left atrial pressure. In a young animal, this is most likely due to a left-to-right shunting cardiac defect. In an older animal, this is most likely due to a combination of left heart failure and chronic pulmonary disease.

Early cardiogenic pulmonary edema appears as interstitial to bronchointerstitial infiltrate. As failure worsens, the interstitium becomes saturated and an alveolar pattern may be seen. Cardiogenic edema is typically bilaterally symmetrical. In the dog, it is most prominent in the caudal-dorsal and perihilar regions. In cats, it is typically found in the ventral regions, although a diffuse distribution can also be seen. Large volume pleural effusion as a manifestation of heart failure is much more common in cats than in dogs.

Generally, left-sided congestive heart failure is considered to be present and active by the finding of three radiographic changes: 1) left atrial enlargement, usually accompanied by left ventricular enlargement; 2) distention of the pulmonary veins (with or without pulmonary arterial distension); and 3) interstitial to alveolar pattern in a location consistent with heart failure for the species. The presence of these three signs indicates that, at very minimum, therapy with a loop diuretic such as furosemide, and an ACE-inhibitor should be initiated. Most dogs will also benefit from the addition of pimobendan to the above combination.

Echocardiography Reviewed

Echocardiography, in contrast to radiographs, allows very precisely measurements of chamber dimensions. Echocardiograms usually begin with views from the right hemithorax, and the first view commonly acquired is the right parasternal long-axis four-chamber view. The heart is positioned horizontally across the screen with the left atrium (LA) and ventricle (LV) in the far field and the right atrium (RA) and ventricle (RV) in the near field. By making a slight rotation of the transducer, the aorta is brought into view, the so-called "five" chamber view. Here the LA is much smaller, but the aorta is in full view and represents the fifth chamber. From this view the transducer is rotated 90 degrees and the heart is seen in the

short axis. There are three standard short-axis views: 1) LV at the papillary muscles, 2) LV at the mitral valve (MV), and 3) the heart base showing the aortic valve, LA, RA, RV and RV outflow tract (RVOT).

To complement these 2D views, a series of M-Mode views are also acquired. M-Mode uses only one ultrasound beam, requiring less raw data to be processed. Consequently, M-Mode has greater image resolution which usually allows more precise measurements of wall thickness and chamber dimensions than 2D. To measure the LV chamber dimensions and wall measurements, a cursor is placed across the 2D image so that the cursor crosses the interventricular septum (IVS) and LV posterior (or free wall; LVPW) between the papillary muscles. The resulting image is a graphical representation of the heart with Time along the X axis and Depth along the Y axis. The RV and IVS are in the near field and the LV lumen appears in the middle, with the LVPW in the far field. From this image, measurement of the LV can be made in diastole and systole. From the mitral valve view, the motion of the MV can be recorded during the cardiac cycle. From the base of the heart, a measurement of the LA and aortic diameters is made. A ratio of LA to aorta (LA/Ao) can also be calculated.

Diastolic wall thicknesses obtained on M-mode are compared to weight-matched reference ranges. In cats with HCM or systemic hypertension, and dogs with sub-aortic stenosis, these measurements can be markedly increased. Diseases causing eccentric hypertrophy (e.g., mitral regurgitation, dilated cardiomyopathy, and left-to-right shunts) result in chamber dilation. An increase in the LV systolic dimension is implicative of systolic dysfunction. Only one M-Mode measurement is taken from the MV, the "E point" to septal separation (EPSS). The EPSS measures the distance from the MV at its maximal opening to the IVS. This distance increases in the face of systolic dysfunction. Multiple views are used to measure the LA. In addition to the short-axis M-mode, a 2D measurement of the LA can be measured from the same view: the measurement line through the LA extends from and parallel to the commissure of the aortic valve between the non-coronary and left-coronary cusps. An LA/Ao ratio can be calculated and in a healthy patient this ratio is usually <1.25. Increased preload causes the LA to increase in size; however, the aorta remains static. Patients in heart failure frequently have an LA/Ao ratio of >2 or more. Another common measure of 2D LA size is obtained from the long-axis view, at the maximal width of the LA, parallel to the mitral valve annulus.

No single measurement provides a fool-proof method of assessing systolic function. The most common measures used are percent fractional shortening (%FS) and ejection fraction (%EF). Each of these measurements determines the percent change in the LV from diastole to systole. Fractional shortening is a percent change in the linear measurement of the LV diameter from on M-Mode using the following formula: (LVIDd - LVIDs)/LVIDd∗100 = %FS (where LVIDd=LV internal diastolic dimension and LVIDs=LV internal systolic dimension). %FS is the easiest measure of systolic function, but it has important limitations. The %FS measures the change in the LV lumen in a single line, but the heart moves in three dimensions. For a 3-D evaluation of LV systolic function, the %EF is used. To measure the LV volume requires some mathematical assumptions since the LV is not a perfect cylinder, but more of a truncated ellipse. Using a formula called the Modified Simpson's rule, the area of the LV is traced from the long-axis four-chamber view. The computer measures the maximal length and then fills this area with a series of disks approximating the area at various levels of the LV. The area of these disks is summed to generate an approximate volume. Another advantage of the %EF is that the difference between the LV volume in diastole and systole is equal to the stroke volume, which allows for calculations of cardiac output by multiplying stroke volume by heart rate. In most clinical situations, if the %FS is normal, then the patient is considered normal. If the %FS is not normal, then the other assessments of systolic function are utilized. However, keep in mind the most important limitation of all of these measures: they are simply derived from the ratio of the starting point in diastole to the ending point in systole; thus, abnormalities of either of these dimensions will alter the final product. You cannot assume because a patient has an ejection fraction

below 50% that systolic dysfunction is present (i.e., the diastolic dimensions could be small due to volume depletion). Similarly, a normal ratio does not guarantee that systolic function is adequate: a large increase in diastole due to increased preload can result in this ratio being "normal" in a patient with decreased contractility. The bottom line: treat your patient, not the numbers.

In addition to these standard views and measurements, other image planes are often useful. Views of the main pulmonary artery and its branches may allow visualization of heartworms or a patent ductus arteriosus, if present. In the presence of pericardial effusion, a tipped short-axis view may bring a tumor on the tip of the right auricle into view. From the left hemithorax, the heart is seen in a four-chamber view with the atria in the far field and the apex of the ventricles in the near field. From this orientation, accurate Doppler velocity measurement across the valves may be acquired.

A complete discussion of Doppler is beyond the scope of this article. However, Doppler is used to assess direction and velocity of flow in order to determine if regurgitant or stenotic valves are present, identify and quantitate shunts, and assess tissue motion for diastolic abnormalities. Most commonly, Doppler is used to quantify the severity of obstruction at a stenotic valve by measuring the velocity of flow out the valve. The severity of obstruction is directly proportional to the velocity and is related by the formula: pressure gradient = $4 \times V^2$ where V is velocity in m/sec. Understanding normal cardiac physiology is crucial to interpret the findings. For example, normal velocity out of the aorta is about 1.0 to 1.7 m/sec. Using this equation, this indicates a LV to aortic PG of ~ 4 to 12 mm Hg. In subaortic stenosis, this velocity can be markedly increased. If the velocity recorded by CW Doppler measured 5.0 m/sec, then a PG of 100 mm Hg exists.

Which Test When?

The answer is based upon the following question: "**What kind of information are you trying obtain by performing the test?**" The test(s) listed below represents my opinion of the easiest way to answer the question in a routine clinical setting.

Information desired	**Recommended initial diagnostic test**
What is the cause of this patient's murmur?	Echocardiogram
Is this patient with a heart murmur safe for general anesthesia?	Thoracic radiographs ± echocardiogram
Why is this patient coughing?	Thoracic radiographs
Is this patient in heart failure? Should I start medications?	Thoracic radiographs
Did the heart failure medications work? Why is this patient still coughing?	Thoracic radiographs
Why is this patient having syncopal episodes?	Echocardiogram, ECG, ± thoracic radiographs
Why is this patient having labored breathing?	Thoracic radiographs
Does this patient have pericardial effusion?	Echocardiogram
Does this old dog with a murmur have severe heart disease?	Thoracic radiographs ± echocardiogram
Does this young dog with a murmur have severe heart disease?	Echocardiogram ± thoracic radiographs
Is this patient safe for doxorubicin?	Echocardiogram

Is this dog's mitral valve disease getting worse over time?	Thoracic radiographs ± echocardiogram
Does this large breed dog have dilated cardiomyopathy?	Echocardiogram
Does this cat have hypertrophic cardiomyopathy?	Echocardiogram
Does this patient have pulmonary hypertension?	Echocardiogram

In summary, echocardiography provides important information about cardiac morphology and function, whereas thoracic radiographs allow evaluation of pulmonary parenchyma and determination of the functional consequences of heart disease. Thoracic radiographs are irreplaceable for patients with respiratory signs. These two modalities are complementary and invaluable resources for the diagnosis and treatment of heart disease. Knowing what answers a clinician is attempting to learn, will guide the diagnostic approach.

References

References are available upon request.

SPEAKER INFORMATION

(click the speaker's name to view other papers and abstracts submitted by this speaker)

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