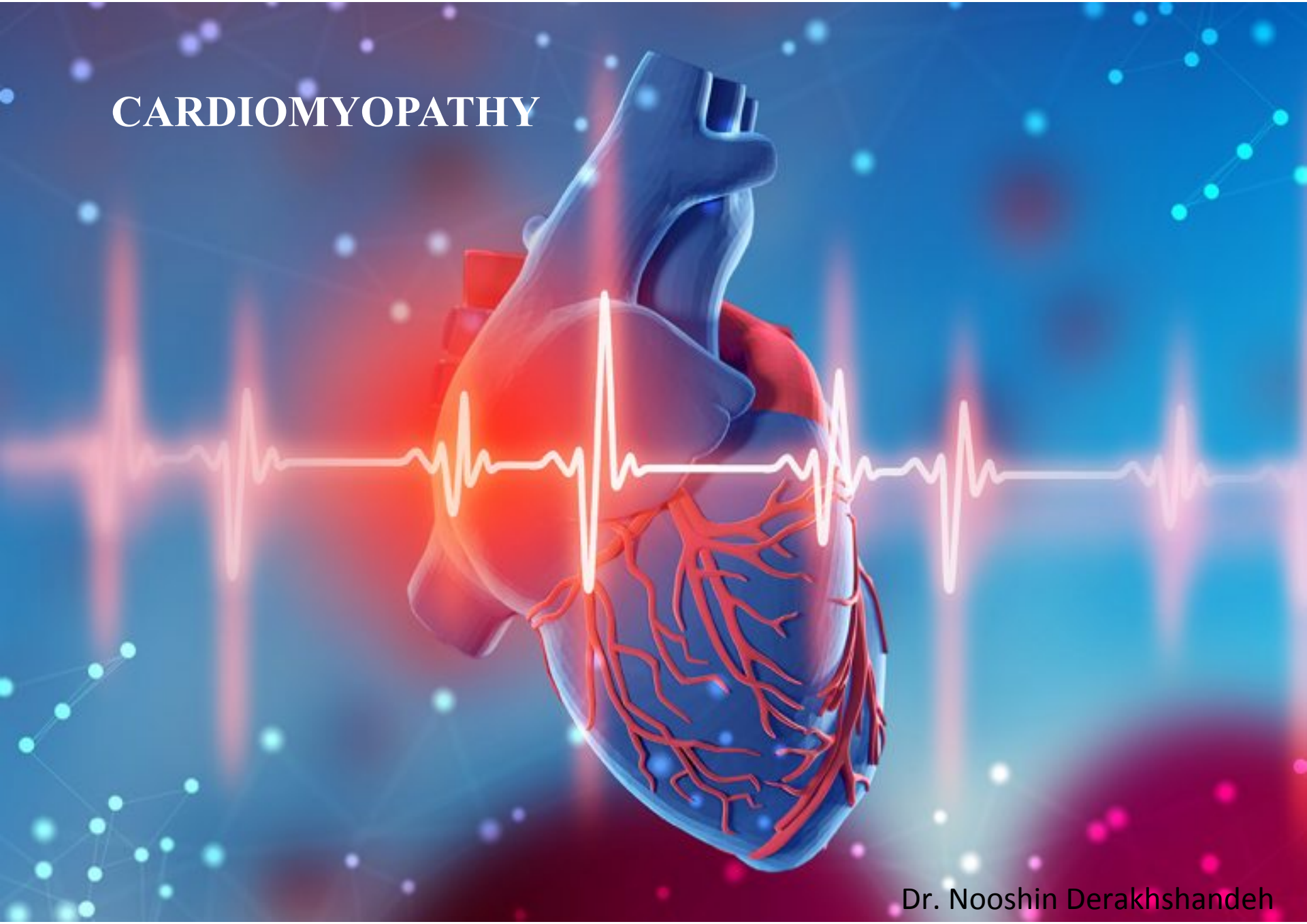
CARDIOMYOPATHY



Dr. Nooshin Derakhshandeh

- **Primary Cardiomyopathy:** Used when the cause is unknown.
- **Secondary Cardiomyopathy:** a specific cause is identified, e.g., (doxorubicin) cardiomyopathy

- **Cardiomyopathy is a major acquired cardiovascular disease in dogs**, following degenerative valve disease and heartworm disease.

- **Dilated Cardiomyopathy (DCM):**

Most common form, particularly idiopathic or primary DCM.
Primarily affects larger breeds.

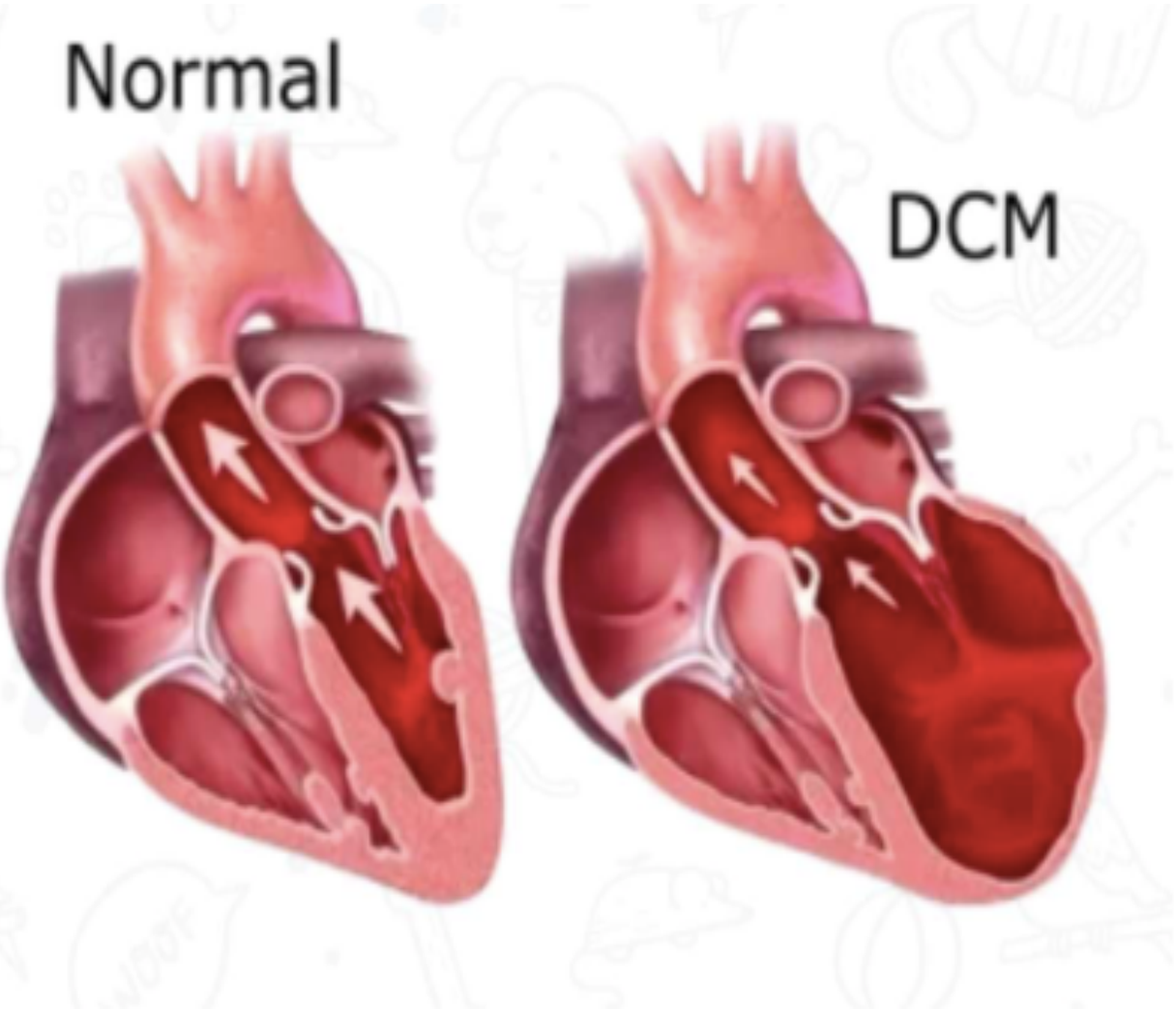
- **Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC):**

Previously known as Boxer cardiomyopathy.
Predominantly affects Boxers; uncommon in other breeds.

- **Less Common Cardiomyopathies:**

Secondary and Infective Myocardial Diseases.
Hypertrophic Cardiomyopathy (HCM): Infrequently recognized in dogs.

DILATED CARDIOMYOPATHY



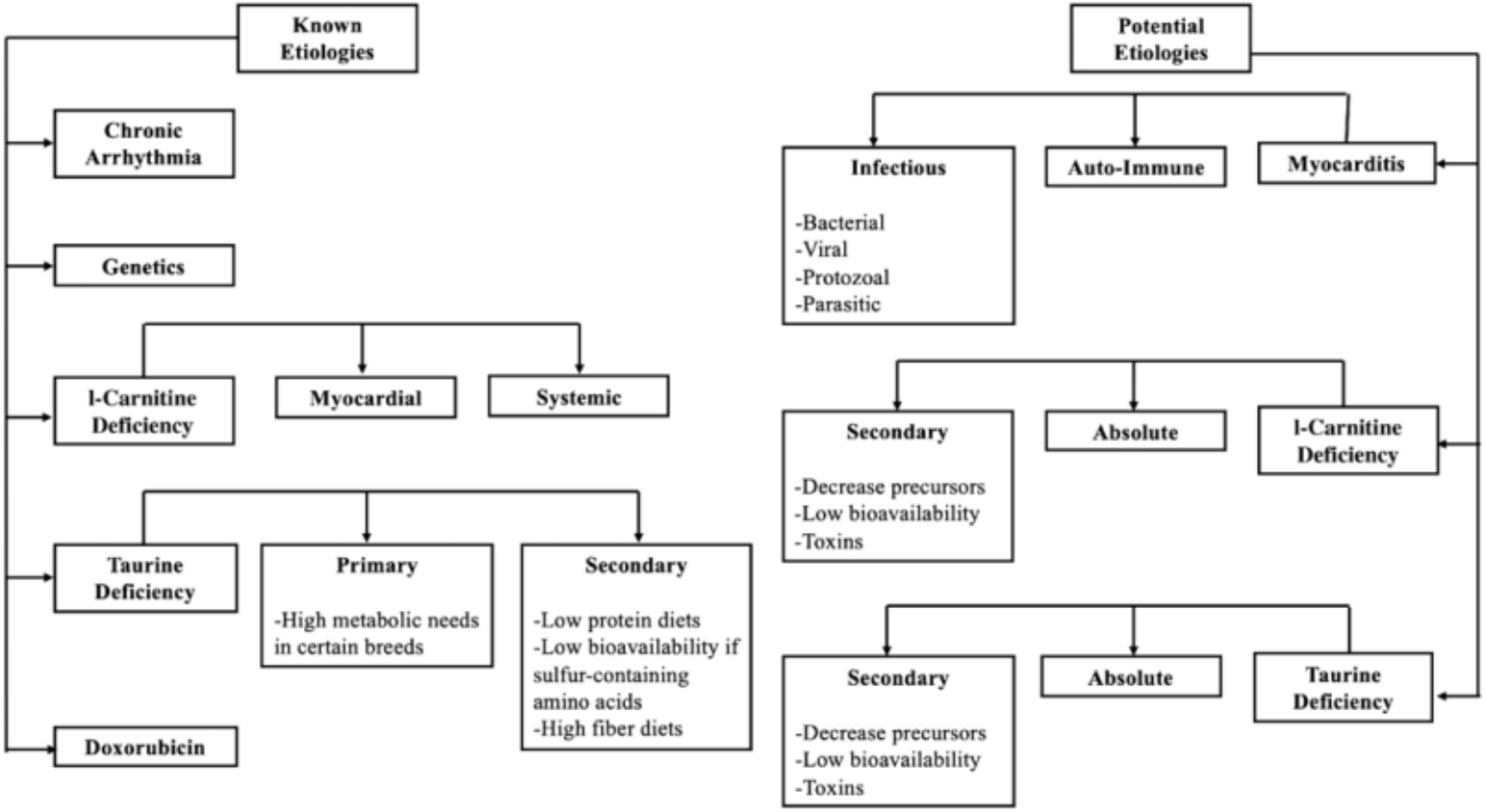


Figure 1. Known and potential etiologies associated with DCM.

Secondary Cardiomyopathies:

- Taurine Deficiency: Can lead to cardiomyopathy and ventricular dilation.
- Doxorubicin Toxicity: A known cause of cardiomyopathy.

- **Myocarditis:** Inflammation of the heart muscle that may result in ventricular dilation.
- **Primary Mitral Valve Insufficiency:** Associated with left ventricular systolic dysfunction, potentially mimicking DCM.

- **Chronic Cardiac Changes and Left-to-Right Shunts:**

Patent Ductus Arteriosus (PDA): Chronic cardiac changes from left-to-right shunts, particularly in adult dogs, can resemble DCM.

- Unknown Cause: A genetic basis is suspected. (Dobermanns)

- Alternative carbohydrate sources like peas and lentils may interfere with amino acid utilization, specifically taurine, affecting heart health.

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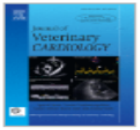

Association of diet with clinical outcomes in dogs with dilated cardiomyopathy and congestive heart failure ☆

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- Dilated cardiomyopathy (DCM) in dogs has been associated with feeding **of grain-free (GF) (low carb), legume-rich die**ts.
- Some dogs with presumed diet-associated DCM have shown improved myocardial function and clinical outcomes following a change in diet and standard medical therapy.
- The median survival time was 344 days for pGF dogs vs. 253 days for GI (grain-inclusive.) (GI)dogs
- Prior GF dogs showed significantly greater improvement in normalized left ventricular internal diastolic diameter and significant decreases in their furosemide and pimobendan dosages over time compared to GI dogs.

- **Diets low in protein,** taurine, and/or taurine precursors

- Diets low in protein, taurine, and sulfur-containing amino acid precursors have been associated with taurine-deficient DCM.

- This may be due to low protein diets being low in essential and nonessential amino acids or vital precursors for carnitine and taurine synthesis.

- when these diets were supplemented with taurine and l-carnitine, DCM clinical signs were reversed and dogs lived longer.

- diets high in fiber, **more specifically insoluble fiber, can decrease the crude protein digestibility in the hindgut**.

- Taurine, carnitine, and other nutrients can be indirectly affected due to the potential decrease in protein digestibility.

- These nutrients are vital in cardiac muscle function. This was observed in medium and large breed dogs given beet pulp.

- **Myocardial Changes in DCM:**

Eccentric Hypertrophy: Ventricular enlargement, predominantly affecting the left side.

Reduced Systolic Function: Marked decrease in left ventricular systolic performance.

Complications of DCM:

Mitral Valve Annulus Dilation: Leads to secondary mitral regurgitation.

Left Atrial Enlargement: **Results from volume overload (preload), impaired ventricular filling, and mitral regurgitation.**

- Typically affects adult dogs.
- **Male** dogs are more commonly affected.
- Breed Specificity: **Portuguese Water Dogs can develop DCM as early as 12 weeks of age.**

- Phases of DCM:

Occult (Asymptomatic) Phase:

- No clinical signs present.
- Presence of arrhythmias or myocardial changes.
- Duration is variable, lasting months to years.

Overt Phase and Clinical Presentation:

- Characterized by exercise intolerance
- congestive heart failure (CHF)
- Gallop rhythm
- syncope.
- Arrhythmia (VPC and AF)
- Sudden Death: In some cases, **particularly in Dobermanns, sudden death may be the first sign of disease**, occurring in approximately 30%–50% of affected dogs.

Increased Sympathetic Tone: Results in peripheral vasoconstriction.

Pale mucous membranes and slowed capillary refill time.

Weak and rapid femoral arterial pulse.

Signs of Heart Failure in DCM

- Left- and/or Right-Sided CHF Symptoms:
- Tachypnea, increased breath sounds, and pulmonary crackles.
- Jugular venous distention or pulsations.
- Presence of pleural effusion or ascites, and/or hepatosplenomegaly.

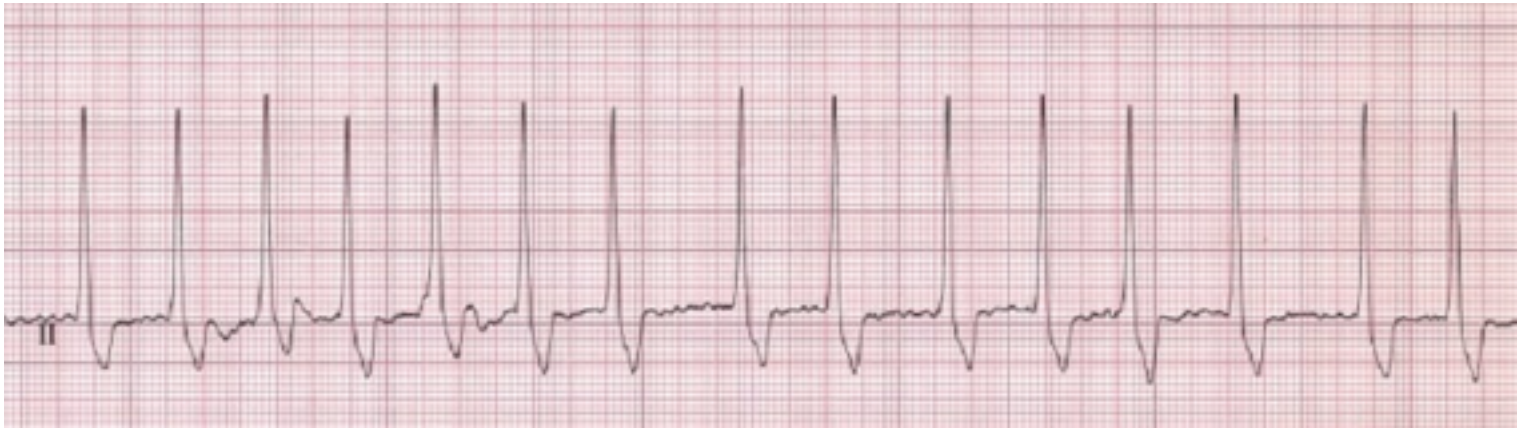
Heart Sounds and Murmurs

- **Muffled Heart Sounds:** Due to pleural effusion or poor cardiac contractility.
- **Third Heart Sound (S3 Gallop):** Classic finding, possibly obscured by irregular rhythm.
- **Systolic Murmurs:** Soft to moderate intensity, indicating mitral and/or tricuspid regurgitation.

- **Atrial Fibrillation (AF) in DCM**

Common in giant-breed dogs and those with severe left atrial enlargement.

Affects approximately 30% of Doberman Pinschers and over 80% of giant breed dogs with DCM.



- **Ventricular premature complexes**

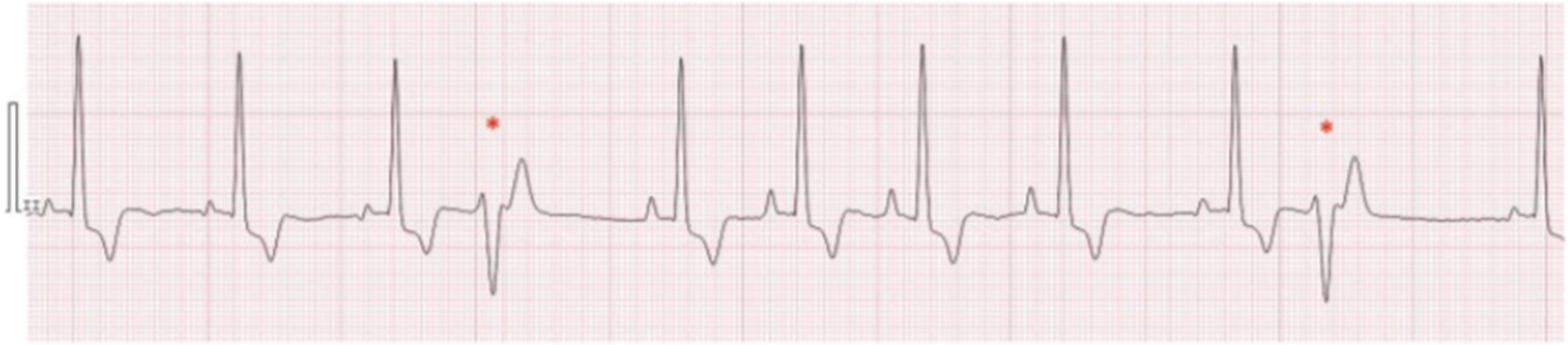


Figure 11.2 Monomorphic ventricular ectopic beats. This trace shows a sinus rhythm interrupted by two examples of single ventricular premature beats (*). The QRS of the ventricular beats has the same morphology, is wider than the normal complexes (100 ms) and has a right bundle branch block conformation. [8-year-old, male Siberian Husky with a patent ductus arteriosus] (50 mm/s, 10 mm/mV)

Physical Examination Findings in DCM:

1. Systolic Murmur and S3 Gallop:

Grade 1 to 3/6 Systolic Murmur: Due to mitral insufficiency from annular dilation, noted over the left apex.

S3 Gallop Sound: Indicates left ventricular dilation.

2.Pulse and Rhythm Abnormalities

Weak Femoral Pulse: Resulting from decreased stroke volume due to poor contractility.

Irregular Rhythm and Pulse Deficits: Common due to ventricular arrhythmias and atrial fibrillation (AF).

Normal heart sound

- The first heart sound (S1) is caused by passive closure of the mitral (left atrioventricular) and tricuspid (right atrioventricular) valves.

- **S1 is longer, louder, duller, and lower pitched than the second heart sound (S2).** It is loudest over the mitral and tricuspid areas and in young, thin animals and those with high sympathetic tone (e.g., fear), tachycardia, systemic hypertension, anemia, or mitral regurgitation.

- **Splitting of S1 is caused by asynchronous closure of the mitral and tricuspid valves.** It can be split normally in large breeds of dogs or abnormally with right bundle branch block, atrial or ventricular premature beats, cardiac pacing, or stenosis of the mitral or tricuspid valve.

- S2 is produced by passive closure of the aortic and pulmonic valves. It is short, high pitched, and sharp. It is loudest over the aortic and pulmonic areas.

- A split S is caused by closure of the pulmonic valve after the aortic valve. This occurs in pulmonary hypertension (e.g., severe heartworm disease or right-to-left PDA), right bundle branch block, ventricular premature beats originating in the left ventricle, atrial septal defect, pulmonic stenosis, and mitral stenosis.

- Paradoxical splitting of S2 is caused by delayed closure of the aortic valve. This results from left bundle branch block, premature beats originating from the right ventricle, subaortic stenosis, severe systemic hypertension, and left ventricular failure.

Abnormal heart sound

- The third heart sound (S3) is caused by rapid ventricular filling and is not heard in normal dogs or cats. It is lower pitched than the S2 and is heard best in the mitral valve area. It occurs during diastole after S2.

- S3 in dogs indicates dilated ventricles, which most commonly occur with **dilated cardiomyopathy,** decompensated **mitral or tricuspid regurgitation**, **large ventricular** or **atrial septal defects**, and large **PDA**. In cats it is associated with **dilated cardiomyopathy, severe anemia, and severe hyperthyroidism**.

- S4 is present in dogs or cats when the **atrium is dilated in response to ventricular diastolic dysfunction, such as hypertrophic cardiomyopathy or third-degree heart block**. It may also be heard in dogs with ruptured chordae tendineae.

- **A gallop rhythm** is an S3, an S4, or a combination of the two. Gallops have a low frequency and can be difficult to hear. **A gallop can be an early sign of heart failure, preceding clinical signs.**

- **Systolic clicks** are short, mid- to high-frequency clicking noises that occur in systole between S1 and S2 .They are usually loudest over the mitral and tricuspid areas. A systolic click may come and go and may change its position in systole (gets closer to or further away from S2) and may change its intensity.
- A systolic click can be hard to differentiate from a gallop, especially if the animal's heart rate is fast.
- The precise cause of the click is unknown, but it may be caused by the **mitral valve prolapse in dogs with early mitral regurgitation** because many of these animals have a mitral regurgitation murmur later in life. This is a benign finding and not usually associated with heart failure.

- An electrocardiogram is necessary to distinguish among sinus, atrial, and ventricular tachycardia.

- **Atrial and ventricular premature beats generate extra sounds that mimic S3 and S4**. It is difficult to differentiate between the two types of premature beats, as well as between S3 and S4 during physical examination. An electrocardiogram may be necessary.

- Sick sinus syndrome will have long pauses where the heart is not beating at all. If the pause lasts long enough, the dog will faint. Sometimes there are also periods of tachycardia, which gives it the name “bradycardia/tachycardia syndrome.”

- Sick sinus syndrome was first described in older female Miniature Schnauzers but has been found in multiple breeds of dogs.

- **Murmurs** are caused by turbulent blood flow through the heart and vessels.

- The turbulence can be caused by disruptions of blood flow through **holes in the heart** (e.g., ventricular or atrial septal defect**), a stenotic valve** (e.g., aortic, pulmonic, mitral, or tricuspid stenosis), **an insufficient valve** (e.g., mitral, tricuspid, aortic, or pulmonic regurgitation), or an **abnormal arterial venous connection near the heart** (e.g., PDA), or it can be caused by altered blood viscosity or changes in blood vessel diameter.

- Functional murmurs are divided into **physiologic and innocent murmurs.**

- **Physiologic murmurs** have a known cause, such as increased cardiac output or decreased blood viscosity and occur with anemia, hypoproteinemia, fever, increased blood pressure, pregnancy, hyperthyroidism, or an athletic heart.

- **Innocent murmurs** have no known cause and are not associated with any cardiac problem.
- These murmurs are soft systolic murmurs (no louder than grade 3) and usually occur in young animals.
- They can be located over any valve area but are most frequent over the mitral and aortic areas.
- Also, these murmurs should disappear by the time of the animal's last vaccinations (5 months of age).

- **Pathologic murmurs** are caused by underlying heart or vessel disease, such as stenosis of valves, outflow tract, or great vessels; valvular regurgitation; or abnormal intracardiac or extracardiac shunts.

- the murmur should be identified as to its timing in the cardiac cycle **(e.g., systolic, diastolic, continuous).**

- **the duration of the murmur** (e.g., early systolic, holosystolic, pansystolic) should be noted.

- the site at which the murmur is loudest (PMI) (e.g., valve area) and where it radiates because of blood flow through the defect (e.g., other valve areas where it can be heard) should be noted.

- the intensity or loudness of the murmur can be evaluated on the basis of the following scale:

grade 1/6 can only be heard after listening for several minutes and sounds like a prolonged S1;

grade 2/6 is very soft but can be heard immediately;

grade 3/6 is low to moderate in intensity;

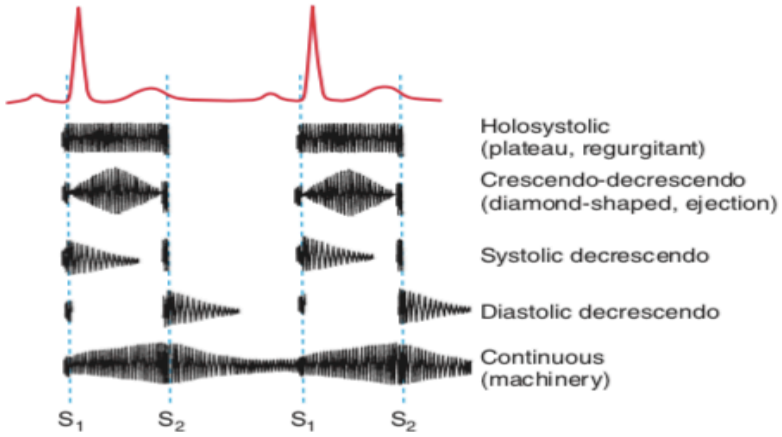
grade 4/6 is very loud, but a thrill cannot be palpated on the thorax;

grade 5/6 is very loud, and a thrill can be palpated on the thorax;

grade 6/6 can be heard without a stethoscope or with the stethoscope slightly off the thoracic wall.

Grading of Heart Murmurs

GRADE	MURMUR
I	Very soft murmur; heard only in quiet surroundings after prolonged listening
II	Soft murmur but easily heard
III	Moderate-intensity murmur
IV	Loud murmur but no precordial thrill
V	Loud murmur with a palpable precordial thrill
VI	Very loud murmur with a precordial thrill; can be heard with the stethoscope lifted from the chest wall



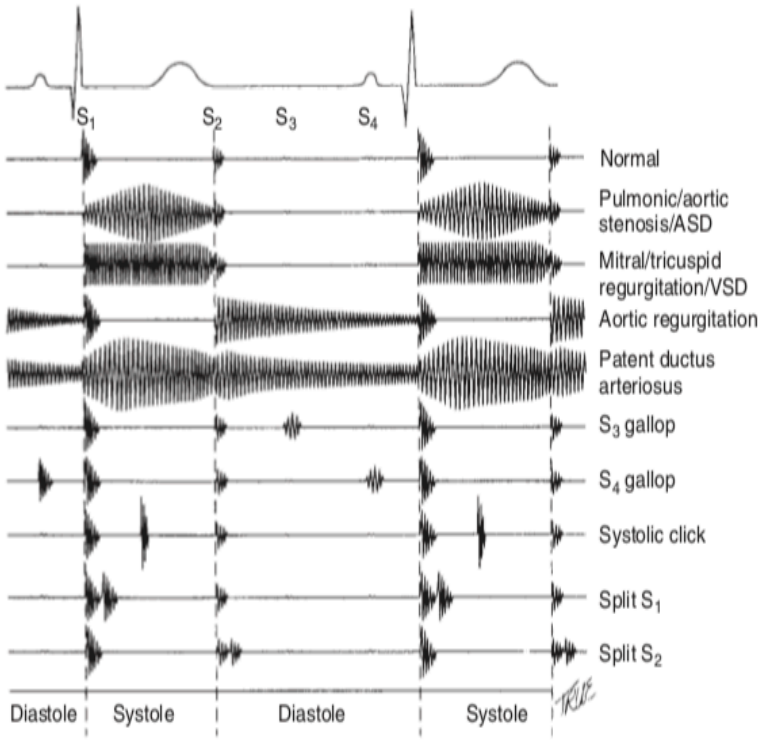


Figure 2-16 Cardiac cycle with electrocardiogram and phonocardiogram schematized. Both normal and abnormal sounds are included. *ASD,* Atrial septal defect; *VSD,* ventricular septal defect. *(From Atkins CE: Abnormal heart sounds. In Allen DG, editor:* Small animal medicine, *Philadelphia, 1991, Lippincott Williams & Wilkins.)*

- Systolic murmurs can occur in early (protosystolic), middle (mesosystolic), or late (telesystolic) systole or throughout systole (holosystolic).

- **Diastolic murmurs** generally occur in early diastole (protodiastolic) or throughout diastole (holodiastolic). Murmurs at the end of diastole are termed *presystolic.*

- **Continuous murmurs** begin in systole and extend through S2 into all or part of diastole.

- **A holosystolic (plateau-shaped) murmur** begins at the time of S1 and is of fairly uniform intensity throughout systole. Loud holosystolic murmurs may mask the S1 and S2 sounds. AV valve insufficiency and interventricular septal defects commonly cause this type of murmur because turbulent blood flour occurs throughout ventricular systole.

- A crescendo-decrescendo or diamond-shaped murmur starts softly, builds intensity in midsystole, and then diminishes; **S1 and S2 can usually be heard clearly before and after the murmur.** This type is also called an ***ejection murmur*** because it occurs during blood ejection, usually because of ventricular outflow obstruction.

- A decrescendo murmur tapers from its initial intensity over time; it may occur in systole or diastole.

- Continuous (machinery) murmurs occur throughout systole and diastole.

- Systolic murmurs can be decrescendo, holosystolic (plateau-shaped), or ejection (crescendo-decrescendo) in configuration. It can be difficult to differentiate these by auscultation alone.

- **Diastolic murmurs are uncommon in dogs and cats.**

- **Aortic insufficiency from infective endocarditis** is the most common cause, although **congenital malformation or degenerative aortic valve disease occasionally occurs.**

- Clinically relevant pulmonic insufficiency is rare but would be more likely in the face of pulmonary hypertension. These diastolic murmurs begin at the time of S2 and are heard best at the left base. They are decrescendo in configuration and extend a variable time into diastole, depending on the pressure difference between the associated great vessel and ventricle. Some aortic insufficiency murmurs have a musical quality.

3. Arrhythmias as Early Indicators: Often detected before other symptoms.

- ECG and Holter monitor screening, particularly in predisposed breeds: **Detection of ventricular premature complexes in asymptomatic dogs, especially Dobermanns, should prompt suspicion of occult cardiomyopathy.**

- **Sinus Tachycardia, AF, and Ventricular Tachycardia: Frequently observed in dogs with DCM.**

- **Giant Breeds (Great Dane, Irish Wolfhound): More likely to develop atrial fibrillation (AF).**

- **Dobermanns: Predisposed to ventricular arrhythmias, ranging from single premature complexes to life-threatening ventricular tachycardia.**

4. Thoracic Radiography:

Generalized **cardiomegaly** may be observed.

Not sensitive to mild cardiac changes, better for diagnosing CHF in the overt stage.

Pulmonary venous distension and pulmonary edema can be visualized.

5. Common ECG Changes:

Left ventricular enlargement indicated by R-wave amplitude > 3.0 mV.

- **Limitations: A normal ECG does not rule out DCM.**

FIG 7.1
Radiographic example of dilated cardiomyopathy with congestive heart failure (and subsequent resolution) in a 5-year-old male Doberman Pinscher. Lateral **(A)** and dorsoventral **(B)** views showing left ventricular and left atrial enlargement, pulmonary venous distension, and moderate diffuse pulmonary edema, consistent with left-sided congestive heart failure. Following medical therapy for congestive heart failure, thoracic lateral **(C)** and dorsoventral **(D)** radiographs of the same patient show resolution of pulmonary edema, with persistent cardiomegaly.

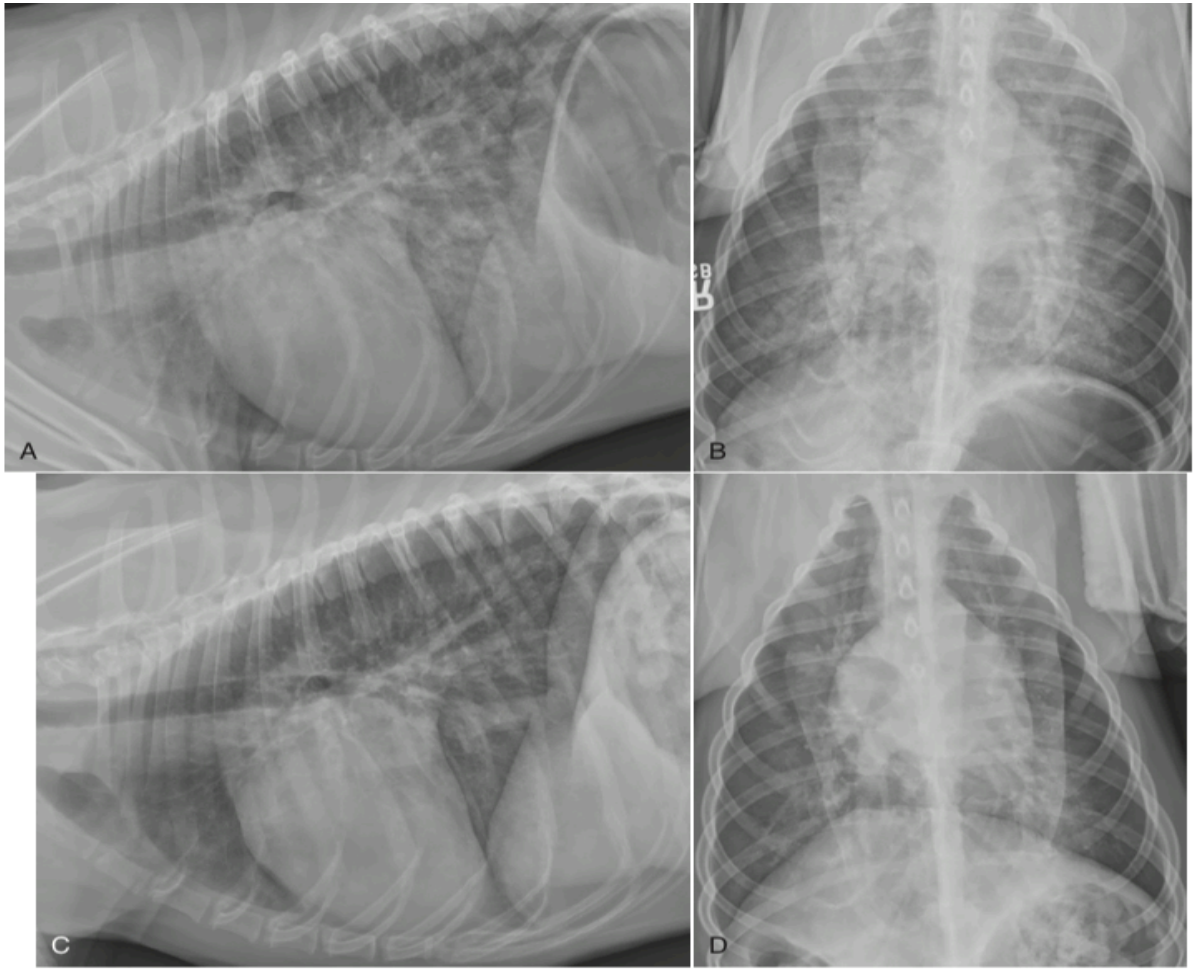
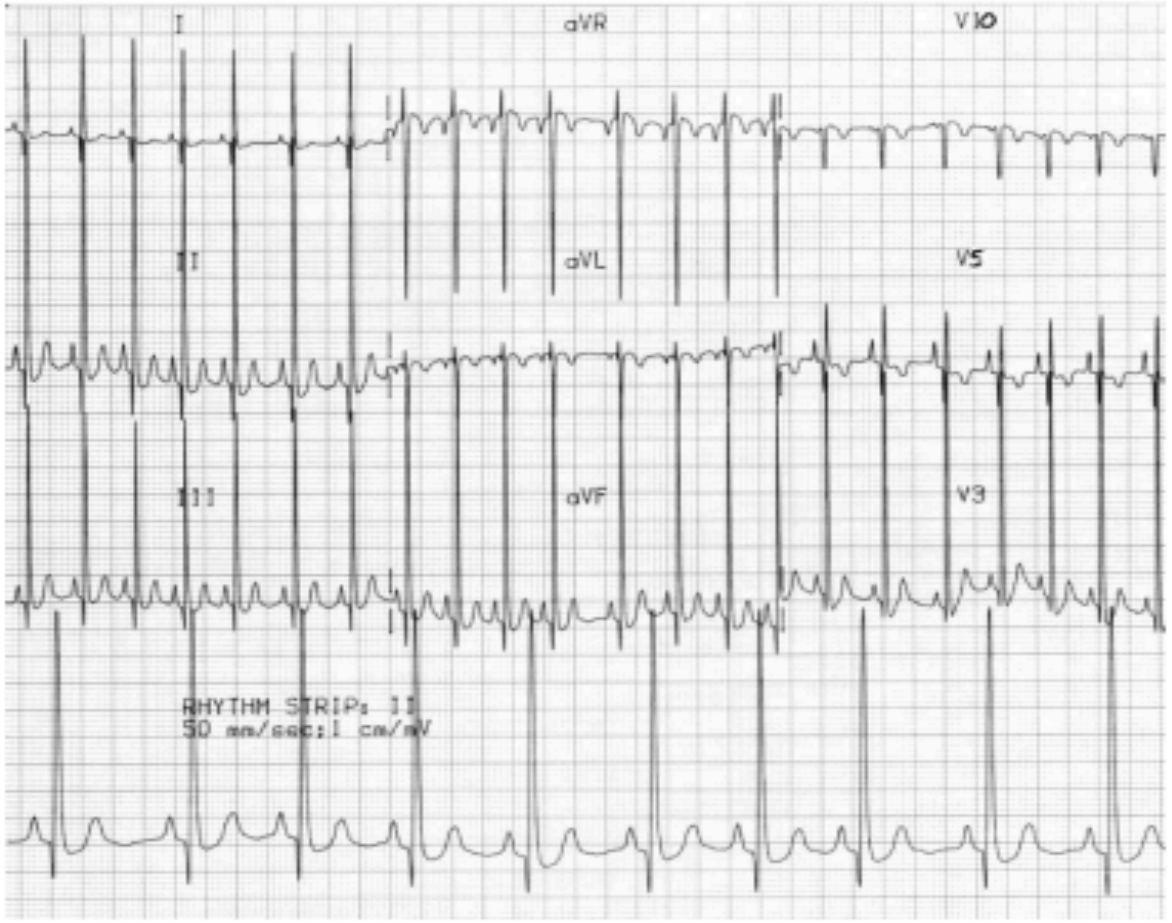


FIG 7.1

Left ventricular enlargement in a dog



6. **Importance of Echocardiography:** Essential Tool: Crucial for evaluating cardiac enlargement and function, particularly valuable in **the occult stage of the disease.**

- Echocardiographic Signs of DCM:

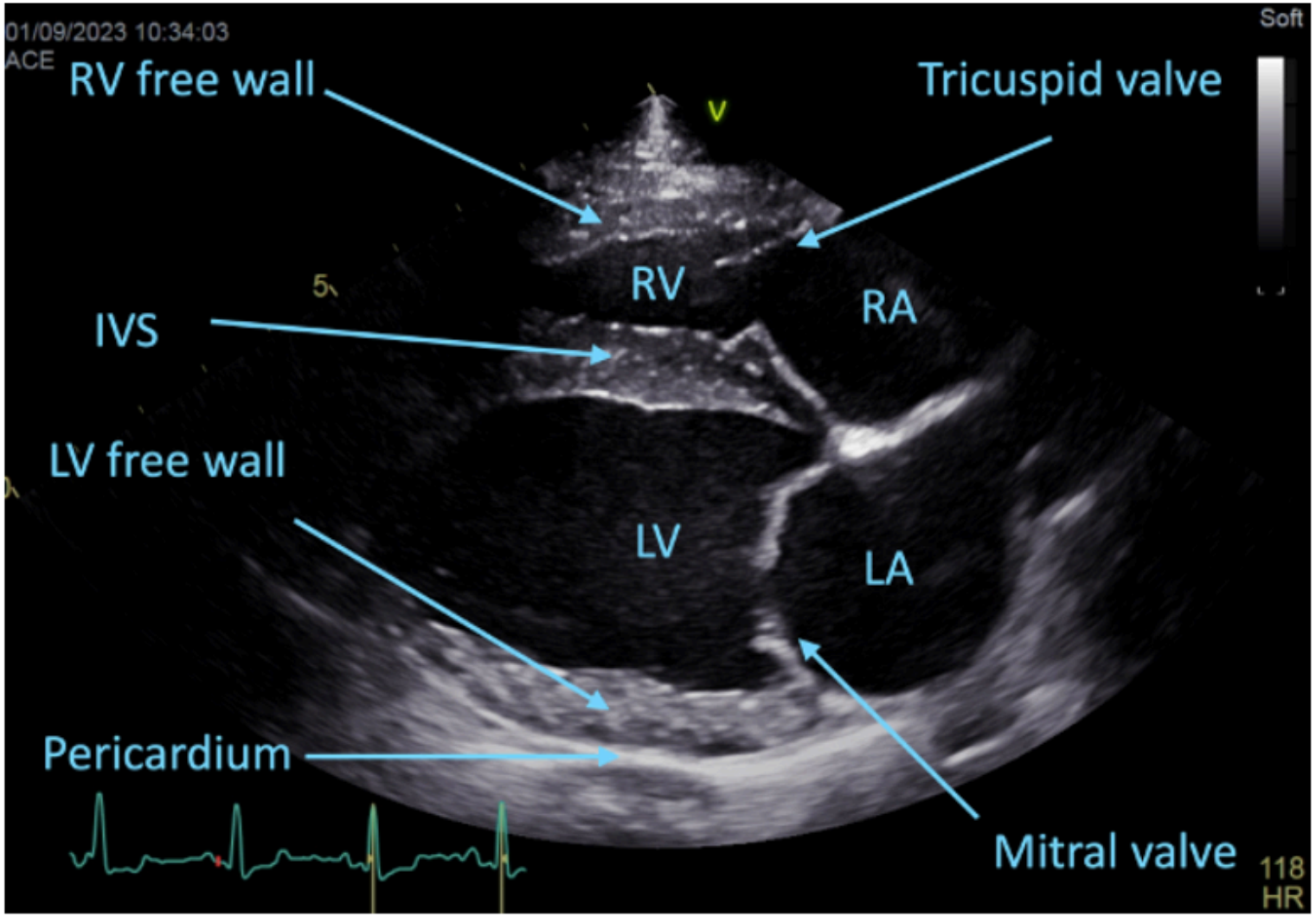
Systolic Dysfunction: **Reduction in fractional shortening (FS) and ejection fraction.**

Increased E-point-to-Septal Separation: A key measurement in assessing cardiac function.

Left Ventricular End-Systolic Diameter: Notably increased in DCM.

- Both **echocardiogram** and **Holter monitor** evaluations are crucial for detecting the **occult phase** of DCM.

B-mode Echocardiography



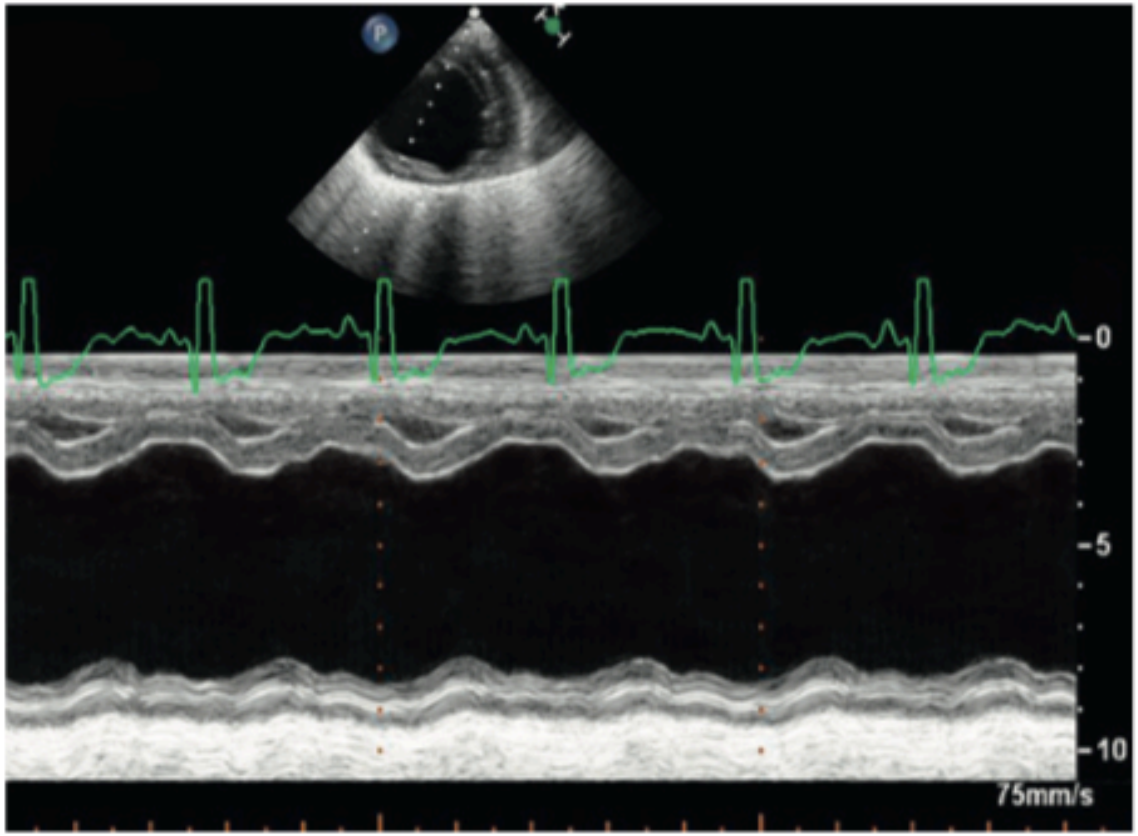


FIG 7.2
M-mode echocardiogram from a Doberman Pinscher with dilated cardiomyopathy at the level of the left ventricular papillary muscles. Note attenuated wall motion (fractional shortening ~18%) and the increased left ventricular dimensions in both diastole and systole.

M-mode calculation:

End diastolic volume	$EDV = \frac{7\ LVIDd^3}{2.4 + LVIDd}$
End systolic volume	$ESV = \frac{7\ LVIDs^3}{2.4 + LVIDs}$
Stroke volume	SV = EDV - ESV
Cardiac output	CO = SV x heart rate
Fractional shortening	$FS = \frac{LVIDd - LVIDs}{LVIDd}$
Ejection fraction	$EF = \frac{SV}{EDV}$
Percent of septum thickening	$PST = \frac{Std - Sts}{STd}$
Percent of posterior wall	$PWT = LVPWd - LVPWs$

Measurements

Chamber dimensions

- LV diameter-through M-mode
- LV wall thickness
- Lt. Atrial & Aortic dimensions – M mode & 2D measures

Systolic function

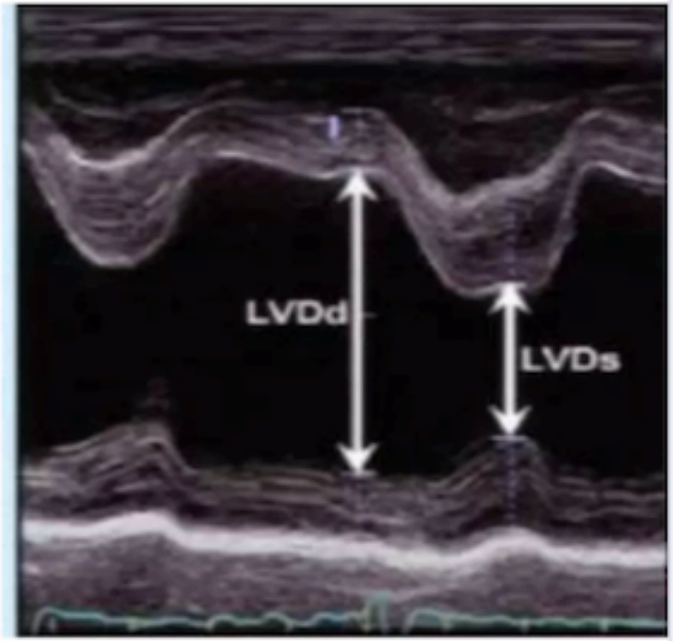
- Lt.V. Fractional shortening (FS%)
- E point Septal Separation (EPSS)
- End-Systolic Volume Index(ESVI)
- Ejection fraction(EF%)
- Systolic Time Intervals

Diastolic function

- Doppler Echo-Choice for evaluation

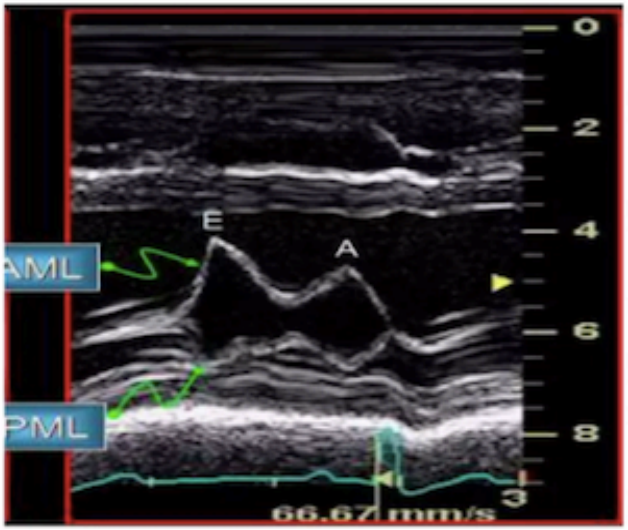
Fractional shortening

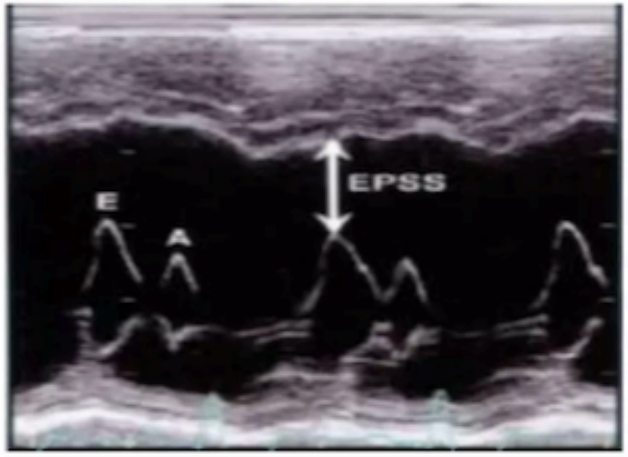
- **FS –Common Echo index of systolic function.**
- **FS % = (LVDd-LVDs) x 100**
- **LVDd.**
- **Mean FS % range – 25-40 %**
  - **>30% in the dog**
  - **>40% in the cat**
  - **>45% if MR is compensated**



EPSS

- M-mode measure – at Mitral valve.
- Distance between **ventricular septum** &Peak opening of **Ant.Mitral.Volve**
- Indicator of **Lt.V filling & function**.
- Normal dog EPSS- **<7.7 mm.**
- Range in giant breeds – **5-8mm**





- **Comprehensive Echocardiographic Examination:**

Modalities Used: 2D, M-mode, and Doppler echocardiography

Goals of Examination:

Identify the primary lesion and assess its severity.

Detect coexisting abnormalities.

Evaluate the size and function of all four cardiac chambers.

Role of Echocardiography in Management

Provides critical data for assessing disease progression.

Monitoring: Enables evaluation of the response to treatment during follow-up examinations.

- **Obtaining Transthoracic Echocardiographic Images:**

Right Side Parasternal Window: Located between the sternal border and costochondral junction from the 3rd to the 6th intercostal spaces.

Left Side Windows: Caudal/Apical Views: Near the sternal border between the 5th and 7th intercostal spaces.

Cranial Views: Between the sternum and costochondral junction at the 3rd or 4th intercostal space.

- Preferred Transducers: Small contact surface transducers are recommended for precise imaging.

Optimal Transducer Frequency:

Cats and Small Breed Dogs: 8–10 MHz

Medium-Size Dogs: 5–8 MHz

Large Breed Dogs: 2–5 MHz

Standard Echocardiographic Measurements:

- Interventricular septum thickness
- Left ventricular internal diameter
- Left ventricular posterior wall thickness

Measurement Phases:

- End-Diastole: Measured at the onset of the QRS complex on ECG, or the frame following mitral valve closure.
- End-Systole: Occurs at the end of the T wave or the frame preceding mitral valve opening (smallest left ventricular internal diameter).

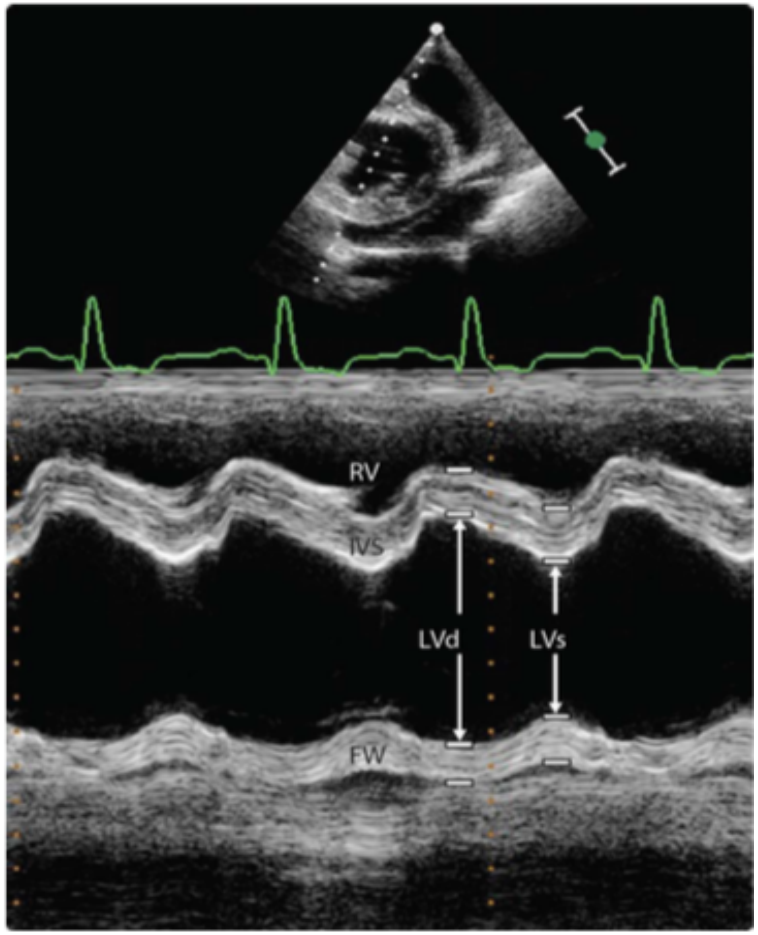


FIGURE 8.15 M-mode view of the left ventricle at the chordae tendineae level. The M-mode cursor is placed perpendicular to the interventricular septum and bisects the left ventricular free wall between the two papillary muscles. FW, left ventricular free wall; IVS, interventricular septum; LVd (LVIDd), left ventricular internal diameter in diastole; LVs (LVIDs), left ventricular internal diameter in systole; RV, right ventricle.

- **Right Ventricle Overview:**

Described as crescent-shaped or pyramid-shaped.

Smaller size and thinner walls compared to the left ventricle, making assessment challenging.

- Qualitative Assessment:

Right ventricular internal diameter is approximately one-third of the left ventricle.

Right ventricular free wall thickness is one-third to one-half that of the left ventricular free wall.

Right Ventricle's Inflow and Outflow Regions

- **Left Atrium Overview**

Oval-shaped chamber positioned caudal to the aorta and above the left ventricle.
Divided into the body and the auricle, with the auricle being heavily trabeculated.

- Imaging and Function of the Left Atrium

Best viewed from a right parasternal short-axis view at the aortic valve level.

Linear measurements are used to calculate the left atrium-to-aortic ratio (LA:Ao).

Contributes 15%–30% of left ventricular filling at end-diastole.

Receives blood from five to eight pulmonary veins, typically not visible on echocardiogram, but inflow can be studied by pulsed-wave Doppler.

Left Atrium-to-Aortic Ratio (LA:Ao)

Independent of body weight.

In healthy dogs and cats, the ratio is between 1.3 and 1.5, usually not exceeding 1.6.

- **Right Atrium Overview:**

Ovoid structure divided into a main chamber and the right auricle.

Compared to the left atrium in size.

In healthy animals, the left and right atria are of comparable size.

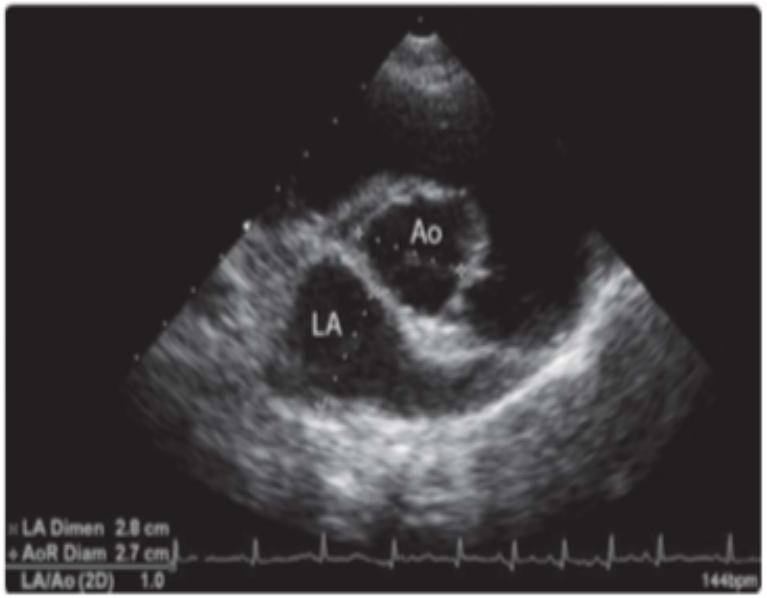


FIGURE 8.16 Measurement of the left atrium and aortic valve annulus from a 2D right parasternal short-axis view. The LA:Ao ratio should be less than 1.6 in healthy animals. Ao, aorta; LA, left atrium.

- **Ambulatory Electrocardiography (Holter Systems):**

Allows for 24- to 72-hour ECG recordings while the animal engages in normal activities within a familiar environment.

Essential for capturing arrhythmias that may not be evident during a standard ECG in a clinical setting.

Designed to collect ECG data and can be manually triggered to save an ECG recording.

Particularly useful for animals with infrequent episodes of weakness or syncope, allowing owners to capture data during events.

- **Indications for Holter Recordings:**

Detects arrhythmias as causes of unexplained syncope.

Quantifies arrhythmias and monitors response to antiarrhythmic therapies.

Screening Tool: Used for predisposed breeds to detect specific cardiac diseases, such as arrhythmogenic right ventricular cardiomyopathy (ARVC) in Boxers.

After clipping away some fur and cleansing the skin, several electrode patches are adhered to the skin over the right and left chest areas and along the sternum. The Holter monitor is a small digital unit with a main cable and 7 wires. The wires snap onto the electrode patches. It is then wrapped up with soft bandage materials around your pet's ribcage. If an appropriately sized vest is available, your pet will be outfitted with a vest over the wrap.

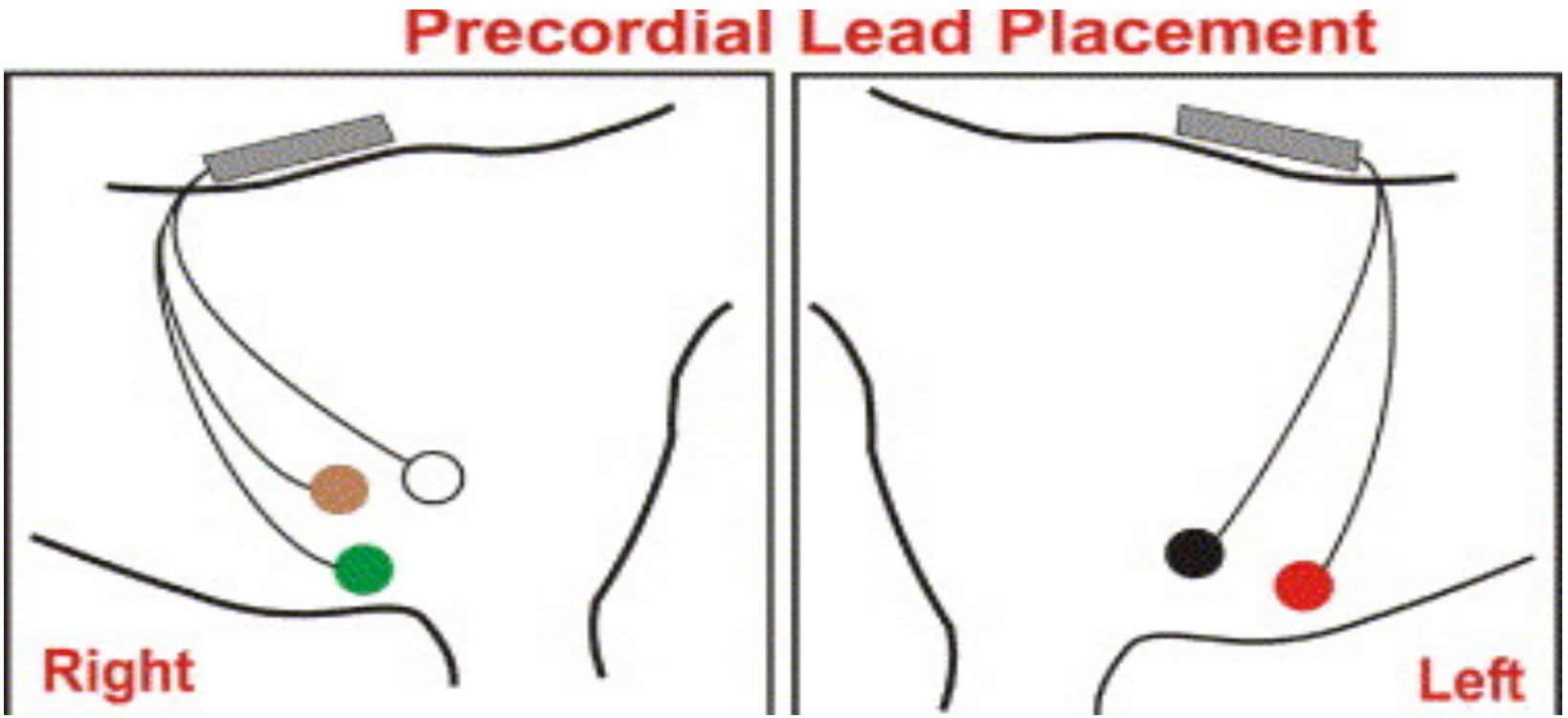


TABLE 8.8 ECG features of the normal dog and cat

	Dog	Cat
Heart rate	– 60–170 bpm for adult dogs – 60–140 bpm for giant breeds – Up to 180 bpm for toy breeds – Up to 220 bpm for puppies	– Range 140–220 bpm – Mean 197 bpm
Rhythm	– Normal sinus rhythm – Sinus arrhythmia – Wandering SA pacemaker	– Normal sinus rhythm
P wave	– Duration: Maximum 0.04 sec; 0.05 s in giant breeds – Amplitude: Maximum 0.4 mV – Mean electrical axis: –18 to +90 degrees	– Duration: Maximum 0.04 sec – Amplitude: Maximum 0.2 mV
PR interval	– Duration: 0.06–0.13 sec	– Duration: 0.05–0.09 sec
QRS complex	– Duration: Maximum 0.07 sec – R-wave amplitude: Maximum 2.5 mV in small breeds, 3 mV in large breeds – Mean electrical axis: +40 to +100 degrees	– Duration: Maximum 0.04 sec – R-wave amplitude: Maximum 0.9mV – Mean electrical axis: 0 to +160 degrees
ST segment	– No depression: Not more than 0.2 mV – No elevation: Not more than 0.15 mV	– No marked depression or elevation
T wave	– Positive, negative, or biphasic – Not greater than one-fourth amplitude of R wave	– Positive, negative, or biphasic – Most often positive – Maximum amplitude 0.3 mV
QT interval	– Duration: 0.15–0.24 s at normal HR, varies with HR	– Duration: 0.16–0.22 s at normal HR, varies with HR

Adapted from Santilli R, Moïse NS, Pariaut R, Perego M (2018). *Electrocardiography of the Dog and Cat*, 2nd ed. Edra, Milano. All measurements made in lead II.

Treatment Outline for Dogs With Dilated Cardiomyopathy

Occult CM (Stage B)

Client education (about disease process and early heart failure signs)
Routine health maintenance
Manage other medical problems
Pimobendan
ACE inhibitor
±Consider β-blocker titration (e.g., atenolol or metoprolol)
Antiarrhythmic therapy, if indicated (e.g., sotalol or mexiletine for ventricular tachyarrhythmias; digoxin and diltiazem combination therapy for atrial fibrillation; see Chapter 4)
Avoid high-salt foods; consider moderately salt-restricted diet
Monitor for early signs of CHF (e.g., resting respiratory rate [see p. 74], activity level)

Mild to Moderate Signs of CHF (Stage C, Chronic/Outpatient Care)*

Furosemide, dose as needed
Pimobendan
ACE inhibitor
Spironolactone
Antiarrhythmic therapy, if indicated (e.g., sotalol or mexiletine for ventricular tachyarrhythmias; digoxin and diltiazem combination therapy for atrial fibrillation; see Chapter 4)
Client education and manage concurrent problems, as previously mentioned
Complete exercise restriction until after signs abate
Moderate dietary salt restriction
Consider dietary supplement (fish oil, ±taurine or carnitine, if indicated)
Monitor resting respiratory rate (see p. 74) ±heart rate at home

Severe CHF Signs (Stage C, Acute/Hospitalized Care)*

Supplemental O_2
Cage rest and minimal patient handling
Furosemide (more aggressive doses, parenteral)
Pimobendan (continue or add as soon as oral administration possible)
Consider dobutamine, especially if persistent hypotension (see Box 3.1, p. 62)
Antiarrhythmic therapy, if necessary (e.g., lidocaine for ventricular tachycardia, PO loading or IV diltiazem (or digoxin) for uncontrolled AF, see text and Table 4.2, p. 90)
Consider cautious use of a vasodilator (nitroprusside, hydralazine, or amlodipine) for adjunct afterload reduction, if necessary, and if blood pressure is not low; beware hypotension
Thoracocentesis, if moderate- to large-volume pleural effusion

Chronic Recurrent or Refractory Heart Failure Strategies (Stage D)*

Ensure that therapies for stage C are being given at optimal doses and intervals, including furosemide, pimobendan, ACE inhibitor, spironolactone
Rule out complicating factors: arrhythmias, renal or other metabolic abnormalities, systemic arterial hypertension, anemia, and other complications
Increase furosemide dose/frequency as needed (and as renal function allows)
Increase pimobendan dose frequency to q8h and/or increase dose
Consider adding digoxin for additional inotropic support
Add (or increase dose of) adjunctive diuretics (e.g., spironolactone, hydrochlorothiazide); monitor renal function and electrolytes closely
Consider additional afterload reduction (e.g., amlodipine or hydralazine); monitor blood pressure closely
Strictly curtail exercise
Further restrict dietary salt intake
Thoracocentesis (or abdominocentesis) as needed
Hospitalize as needed for acute CHF therapy (see Box 3.1)
Manage arrhythmias, if present (see Chapter 4)

ACE, Angiotensin-converting enzyme; *AF*, atrial fibrillation; *CHF*, congestive heart failure; *IV*, intravenous

- **Stage B (Occult) Dilated Cardiomyopathy Treatment**

Pimobendan:

- Shown to delay progression to congestive heart failure (CHF) or sudden death in Doberman Pinschers with echocardiographic evidence of occult DCM.

Angiotensin-Converting Enzyme Inhibitors (ACEI):

- Recommended for dogs with left ventricular dilation or reduced systolic function.

β-Blockers:

- Proven beneficial in human cardiomyopathy, but lacking clinical trials in canine DCM. **Carvedilol,** a combined β-blocker and α-blocker, has limited and unpredictable oral bioavailability in dogs and is less favored.

ACVIM American College of Veterinary Internal Medicine

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Standard Article | Open Access

Efficacy of Pimobendan in the Prevention of Congestive Heart Failure or Sudden Death in Doberman Pinschers with Preclinical Dilated Cardiomyopathy (The PROTECT Study)

N.J. Summerfield, A. Boswood, M.R. O'Grady, S.G. Gordon, J. Dukes-McEwan, M.A. Oyama, S. Smith, M. Patteson, A.T. French, G.J. Culshaw, L. Braz-Ruivo, A. Estrada, M.L. O'Sullivan ... See all authors

Prospective clinical trial evaluating spironolactone in Doberman pinschers with congestive heart failure due to dilated cardiomyopathy ☆, ☆☆

A. Laskary BSc, S. Fonfara DVM, Dr med vet, PhD, H. Chambers BSc, M.L. O'Sullivan DVM, DVSc

ACVIM American College of Veterinary Internal Medicine

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Carvedilol in Dogs with Dilated Cardiomyopathy

Mark A. Oyama, D. David Sisson, Robert Prošek, Barret J. Bulmer, Mike W. Luethy, Virginia Luis Fuentes

First published: 28 June 2008 | https://doi.org/10.1111/j.1939-1676.2007.tb01949.x | Citations: 14

- **Antiarrhythmic Drug Therapy in Stage B DCM:**

Influenced by arrhythmia frequency and complexity on Holter recording.

Presence or absence of clinical signs such as episodic weakness or syncope.

Goals of Therapy:

- Decrease arrhythmia frequency and severity.
- Increase ventricular fibrillation threshold to reduce clinical signs and risk of sudden death.

Common Antiarrhythmic Medications:

- First-Line Medications:

Sotalol and/or Mexiletine: Commonly used for ventricular arrhythmias in DCM.

- Refractory Cases:

Amiodarone or Procainamide: Sometimes used when first-line treatments are ineffective.


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انالا پریل


۳۰ قرص خط‌دار
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هر قرص خط‌دار حاوی: ۶/۲۵ میلی‌گرم کارودیلول
۶/۲۵


۱۰۰ | قرص خط دار
سوتالول
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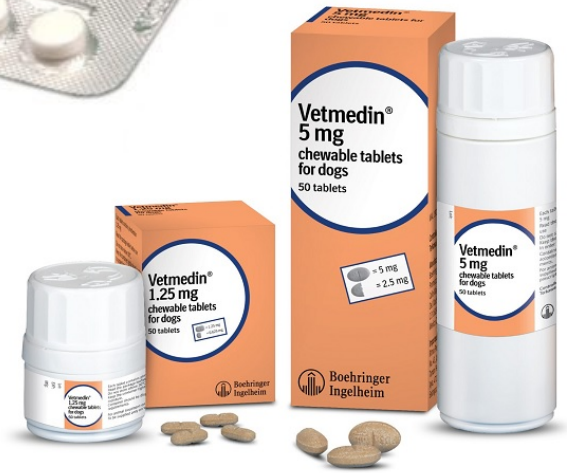
NDC 0093-8740-01
MEXILETINE
HYDROCHLORIDE
Capsules USP
200 mg
100 CAPSULES
TEVA


Vetmedin®
5 mg
chewable tablets
for dogs
50 tablets
Vetmedin®
1.25 mg
chewable tablets
for dogs
50 tablets
Boehringer
Ingelheim

- **Stage C (Clinically Evident) Dilated Cardiomyopathy Treatment**

Therapeutic Goals:

- Improve quality of life and prolong survival.
- Control CHF symptoms, optimize cardiac output, and manage arrhythmias.

Common Medications:

- **Pimobendan, ACEI, and Furosemide:** Used to manage CHF symptoms.
- **Spironolactone:** Often advocated for use.
- Antiarrhythmic Drugs: Administered based on individual patient needs.

- **Acute Therapy for Congestive Heart Failure (CHF):**

Initial Treatment Protocols:

- Parenteral Furosemide: For diuresis.
- Supplemental Oxygen: To improve oxygenation.
- Inotropic Support: To enhance myocardial contractility.
- Vasodilators: Used cautiously based on individual patient needs.
- Thoracocentesis: Indicated if pleural effusion is suspected or confirmed.
- For Poor Myocardial Contractility or Persistent Hypotension:

IV Dobutamine or Dopamine: Infusion for 1 to 3 days.

- **Management of Rapid Atrial Fibrillation (AF)**

Diltiazem and Digoxin: Used to slow ventricular response.

IV Digoxin: Generally avoided; oral formulations preferred.

Diltiazem is more effective but can compromise systolic function; pimobendan should be administered if not already in use.

Digoxin has a slower onset but provides mild positive inotropic effects.

Goal: Reduce ventricular response rate to 150-160 bpm to maximize cardiac output; avoid excessive rate reduction to prevent cardiogenic shock.

- # Long-Term Therapy for DCM

1. Pimobendan (Vetmedin): Oral positive inotrope of choice for managing DCM and CHF.

Mechanism: Phosphodiesterase III inhibitor with Ca++-sensitizing effect, also acts as a vasodilator.

Dosage: Starting at 0.2 to 0.3 mg/kg PO q12h. Can be uptitrated to 0.5 mg/kg PO q8h in progressive cases (off-label use; client consent required).

Benefits: Improves clinical signs and survival.

2. Furosemide Use in Long-Term Therapy

Administered at the lowest effective oral dosage for ongoing management.

3. ACE Inhibitors:

May reduce **progressive ventricular dilation and secondary mitral regurgitation.**

Positive effect on survival in myocardial failure.

Minimizes clinical signs and increases exercise tolerance.

Common ACE Inhibitors: **Enalapril or Benazepril:** Frequently used, with similar effects as other ACEIs.

4. Managing Electrolyte and Acid-Base Abnormalities

- **Hypokalemia**

Treatment Options:

- Potassium-Sparing Diuretic: **Spironolactone is often added to address hypokalemia.**
- Dietary Potassium Supplementation: Considered if additional potassium is needed.

5. Spironolactone as Adjunctive Therapy

Mechanism and Benefits:

Aldosterone antagonist with potential mild diuretic effects.

Helps prevent cardiovascular fibrosis and abnormal remodeling.

Therapeutic Strategy: Used in combination with ACEI, furosemide, and pimobendan for chronic DCM management.

6. Management of Atrial Fibrillation in DCM

Combination of **oral diltiazem and digoxin** is recommended for controlling ventricular response rate.

Digoxin for Heart Rate Control

- Use with Pimobendan: **While pimobendan is preferred for inotropic support, digoxin remains indicated for heart rate control in AF.**
- Dosage: Oral maintenance dose: 0.003 to 0.005 mg/kg PO q12h.
- Typical dose for Dobermans (~40 kg): Approximately 0.125 mg total dose, PO, q12h.
- **Toxicity is uncommon at low doses but monitoring is crucial due to narrow therapeutic index.**
- Measure serum digoxin concentration 7 to 10 days after initiation or dose change; draw samples 6 to 8 hours post-dose.

Diltiazem for Rate Control:

- Cardiac-specific **calcium-channel blocker** effective at slowing AV nodal conduction, reducing heart rate in AF.
- Dosage Considerations: **Start with a low initial dose and gradually titrate to effect or maximum recommended level.**
- Extended-release formulations (Diltiazem ER/XR, Dilacor) allow for twice-daily dosing in chronic administration.

Adjunct Therapy Options:

Amlodipine or Hydralazine:

Useful for additional afterload reduction.

Careful arterial blood pressure monitoring is essential.

Vasodilators must be used cautiously due to low cardiac reserve in affected dogs.

Hydralazine Considerations

- **More likely to cause hypotension, reflex tachycardia, and neurohormonal activation.**

- Dosing: Initiate at a low dose, increase to maintenance level if well-tolerated.

- Monitor for signs of hypotension: worsening tachycardia, weakened pulses, lethargy.

Additional Therapies for DCM:

- **Omega-3 Fatty Acids:** May be beneficial in certain cases.
- **L-Carnitine:** Considered for dogs with low myocardial carnitine concentrations.
- **Taurine:** Recommended for dogs with low plasma concentrations.
- **Long-Term β-Blocker Therapy:** Possible utility, though optimal recommendations require further study.


100 Scored F.C. Tablets
Diltiazem Arya 60
Diltiazem
Each tablet contains:
Diltiazem HCl 60mg
ARYA Pharmaceutical Co.
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100 Tablets/Comprimés
APO-HYDRALAZINE
Hydralazine Hydrochloride Tablets USP
Comprimés de chlorhydrate
d'hydralazine USP
50 mg
APOTEX INC.
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هر قرص روکش دار خط دار حاوی:
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STANDARD ARTICLE | Open Access

Clinical efficacy of a benazepril and spironolactone combination in dogs with congestive heart failure due to myxomatous mitral valve disease: The BEnazepril Spironolactone STudy (BESST)

Melissa Coffman, Emilie Guillot, Thomas Blondel, Catherine Garelli-Paar, Shuo Feng, Susanne Heartsill, Clarke E. Atkins

First published: 24 May 2021 | https://doi.org/10.1111/jvim.16155 | Citations: 8

ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY (BOXER CARDIOMYOPATHY)

Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC):

- Myocardial disease primarily characterized by ventricular arrhythmias and syncope.
- **Ventricular size and function are typically normal at the onset of arrhythmias.**

Affected Breeds:

- **Boxers,** but also documented in breeds such as **English Bulldogs.**
- Typically adult-onset but can be diagnosed **in dogs as young as 1 year.**

Boxer Genetics:

- One identified genetic mutation affects the striatin gene.
- **Boxers with the striatin mutation show a higher number of ventricular premature complexes (VPCs) on 24-hour Holter recordings.**
- ARVC can occur in Boxers without the striatin mutation.

Pathological Characteristics of ARVC:

- Characterized by fatty or fibro-fatty myocardial replacement.
- **Predominantly affects the right ventricle**, with lesser involvement of the left ventricle and both atria.

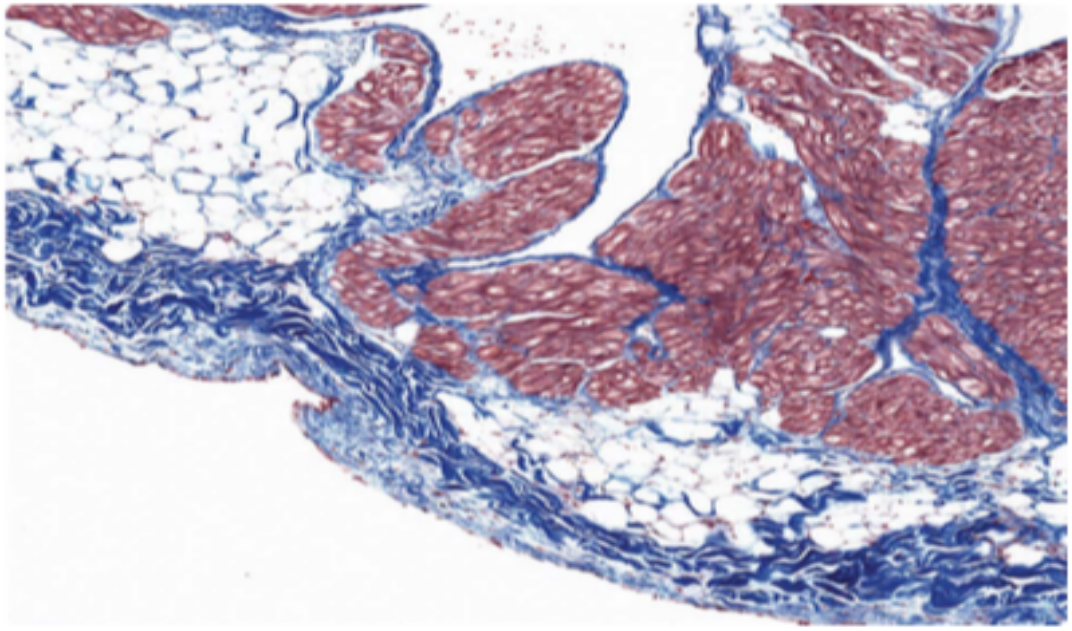


FIGURE 8.37 Histopathology of the myocardium of a Boxer with arrhythmogenic right ventricular cardiomyopathy revealing fatty replacement of the myocytes and interstitial fibrosis. The Masson trichrome stain shows collagen in blue. Magnification ×70.

Clinical Presentation of ARVC

- Ventricular tachycardia
- Right ventricular enlargement
- Syncope and sudden death

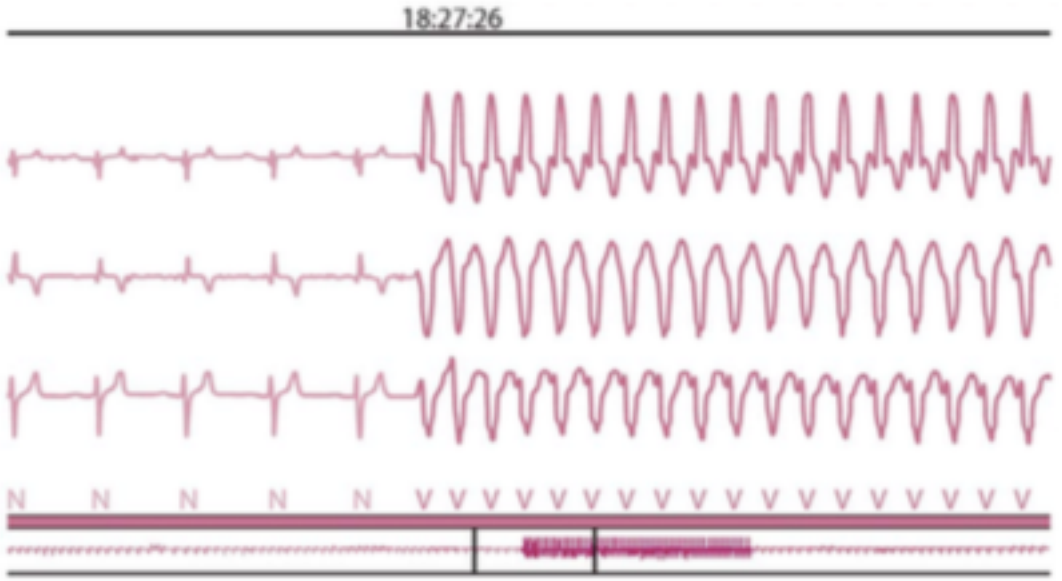


FIGURE 8.38 Holter recording from a Boxer with arrhythmogenic right ventricular cardiomyopathy showing a run of ventricular tachycardia.

Table 2.2 ECG criteria for heart enlargement in the dog and cat

	Dog	Cat
Left atrial enlargement		
P wave	>0.4 mV >0.04 s Notched	>0.04 s
Right atrial enlargement		
P wave	>0.4 mV	>0.2 mV
Left ventricular enlargement		
R wave	>2.5 mV in lead II, aVF (>3.0 mV in large breed dogs) >1.5 mV in lead I	>0.9 mV in lead II
QRS duration*	>0.06 s	>0.04 s
Right ventricular enlargement		
S wave	>0.05 mV in lead I >0.35 mV in lead II	S wave in leads I, II, III and aVF
Electrical axis	Right shift (>+100°)	Right shift (>+160°)

Source: Adapted from Tilley LP, Smith WK (2008). Electrocardiography. In: Tilley LP, Smith WK, Oyama MA, Sleeper MM (eds). *Manual of Canine and Feline Cardiology*, 4th edn. Saunders Elsevier, St Louis.

*QRS duration >0.08 s in the dog and >0.06 s in the cat can be associated with left or right bundle branch blocks. See text for more detail.

Disease Progression Stages:

- **Asymptomatic:** Ventricular arrhythmias are incidental findings.
- **Syncope Stage:** Patient presents with syncope associated with ventricular arrhythmias.
- **CHF Stage:** Signs of congestive heart failure (CHF) due to systolic dysfunction, with a history of weakness or syncope.

- **Prognosis and Risks:**

Sudden Death: Often the first and only sign of the disease, similar to Dobermanns with DCM. More common from ventricular arrhythmias than CHF complications.

Common Physical Examination Findings:

- **Detection of arrhythmias:** single premature beats, paroxysms of tachycardia.
- **Systolic murmur** due to mitral or tricuspid insufficiency from annular dilation.

Electrocardiographic Changes in ARVC:

- Ventricular premature complexes (VPCs) in various forms: singles, couplets, triplets, or bigeminy.
- Ventricular tachycardia with left bundle branch block pattern and positive QRS deflection in leads II, III, and aVF.
- Severe cases may lead to syncope or sudden death.
- 80% day-to-day variation in ventricular beats observed.

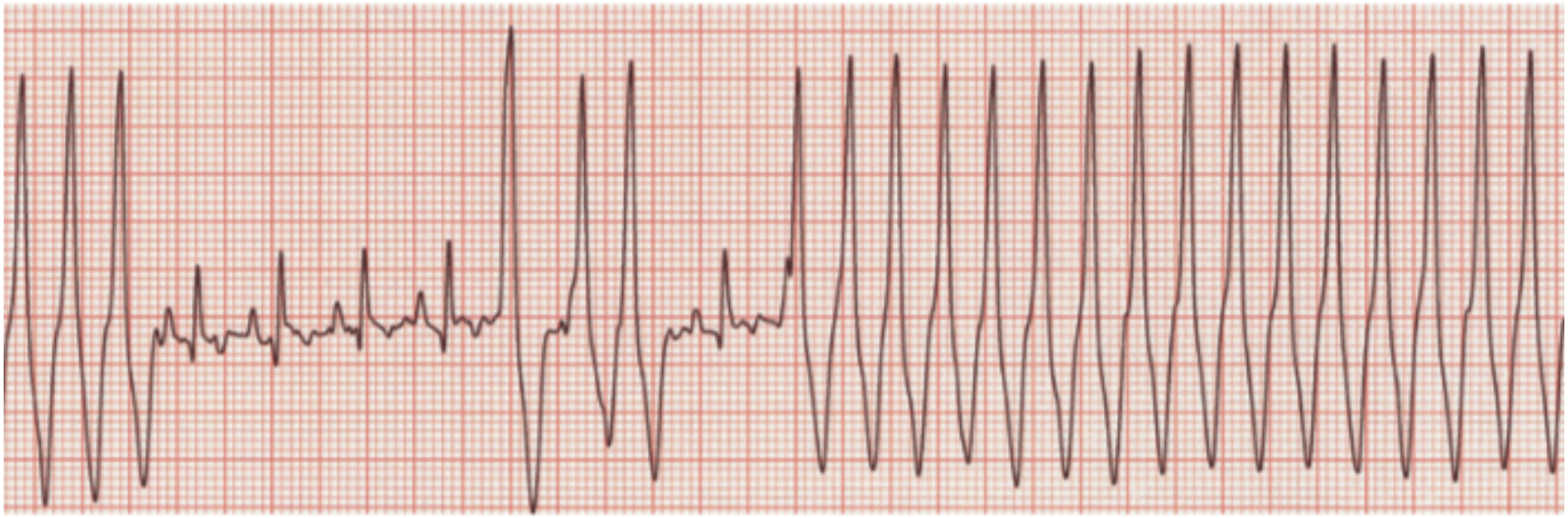


FIG 7.4
Paroxysmal ventricular tachycardia at a rate of almost 300 beats/min in a Boxer with arrhythmogenic right ventricular cardiomyopathy. Note the typical upright (left bundle branch block–like) appearance of the ventricular ectopic complexes in the caudal leads. Lead II, 25 mm/sec.

Holter Monitoring:

- 24-hour monitoring recommended for arrhythmia detection, especially with auscultation evidence or clinical signs like syncope.

- Repeat Holter recording or use of event monitor to increase detection likelihood.

Echocardiography in ARVC

- Echocardiograms often normal in asymptomatic Boxers and dogs with syncope.

- Occasional signs of depressed ventricular function and chamber dilation.

- Atrial enlargement may occur due to ventricular dysfunction, more pronounced in CHF cases.

Treatment Goals for ARVC

- Aim to alleviate clinical signs**; no specific cure for ARVC.**
- **Mild Cases: In the absence of clinical signs and mild arrhythmia burden, treatment may not be necessary.**

- **Antiarrhythmic Therapy**

Sotalol Monotherapy: Most common treatment for ventricular arrhythmias.

Combination Therapy**: If sotalol alone is insufficient, combine with mexiletine or atenolol.**

Impact of Antiarrhythmic Drugs: Can reduce clinical signs and improve quality of life. However, Antiarrhythmic medications do not reduce the risk of sudden death from ARVC.

Monitoring and Therapy Assessment

- Holter Monitoring: Use 24-hour Holter monitoring to assess response to antiarrhythmic therapy.

- ARVC progresses over several years, and the rate of progression varies between dogs.

- In dogs with severe arrhythmia or syncope but normal cardiac function, a 2- to 3-year survival is not unusual when the clinical signs are controlled with antiarrhythmic therapy.

- Survival is usually less than 1 year once myocardial failure and CHF occur.

Table 1 Proposed Holter criteria for the diagnosis of arrhythmogenic right ventricular cardiomyopathy in Boxer dogs.

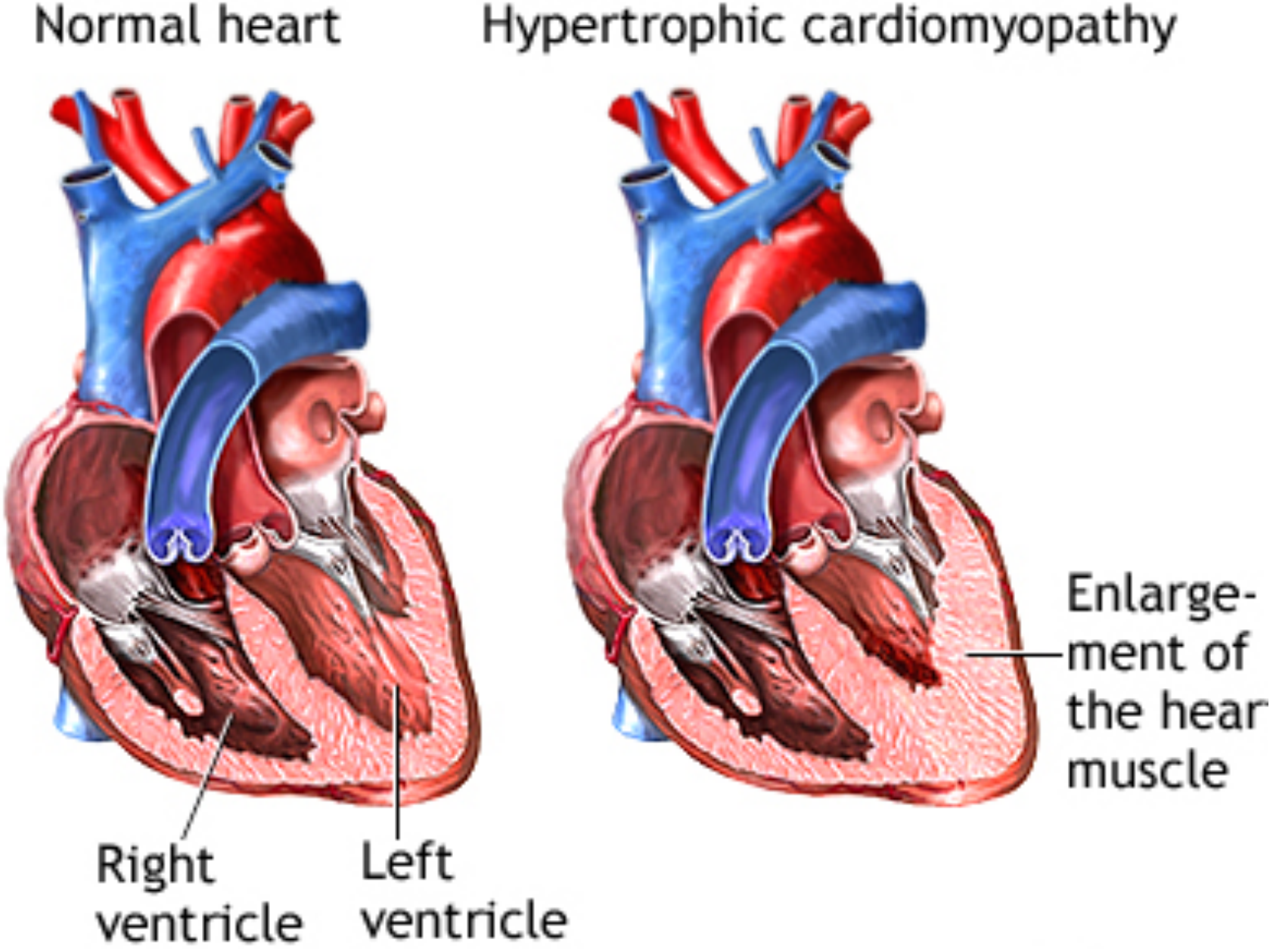
VPCs per 24 h	Interpretation
0–20	Normal
20–100	Equivocal
100–300	Suspicious/likely affected
100–300 with complexity (couplets, runs, or R-on-T); or 300–1000	Very likely affected
>1000	Affected; consider treatment

Modified from Meurs KM. Vet Clin North Am Small Anim Pract. 2017 Sep; 47(5):1103–1111.
VPC: ventricular premature complexes.

Spontaneously arising disease

Vaccine-Associated Anaphylactic Shock in a Springer Spaniel Dog with Arrhythmogenic Right Ventricular Cardiomyopathy

Luca Bertola [†] [*], Andrea Cappelleri [†] [*], Raffaella MA. Tomba [‡], Elisa Dotti [‡], Mario Caniatti [*], Paola Dall'Ara [*], Camilla Recordati [†] [*]


Normal heart
Hypertrophic cardiomyopathy
Enlarge-
ment of
the hear
muscle
Right
ventricle
Left
ventricle

- **Hypertrophic Cardiomyopathy (HCM):**

Rare primary myocardial disease **in dogs**

Characterized by **concentric hypertrophy of the left ventricle without an identifiable cause.**

Clinical Presentation of HCM:

- Predominantly affects male dogs.
- Most diagnoses occur before 3 years of age.
- Hypertrophy Patterns: Dogs typically exhibit symmetrical left ventricular hypertrophy, unlike the asymmetrical pattern seen in humans and cats.

DDX:

- systemic hypertension and other causes **of increased afterload**, such as SAS.
- **Infiltrative myocardial disease,** in particular lymphoma, may be responsible for concentric ventricular hypertrophy.

Diagnosis

- Diagnosis relies on an **echocardiogram to demonstrate left ventricular concentric hypertrophy in the absence of an obstruction to ejection of blood in the aorta or systemic hypertension.**

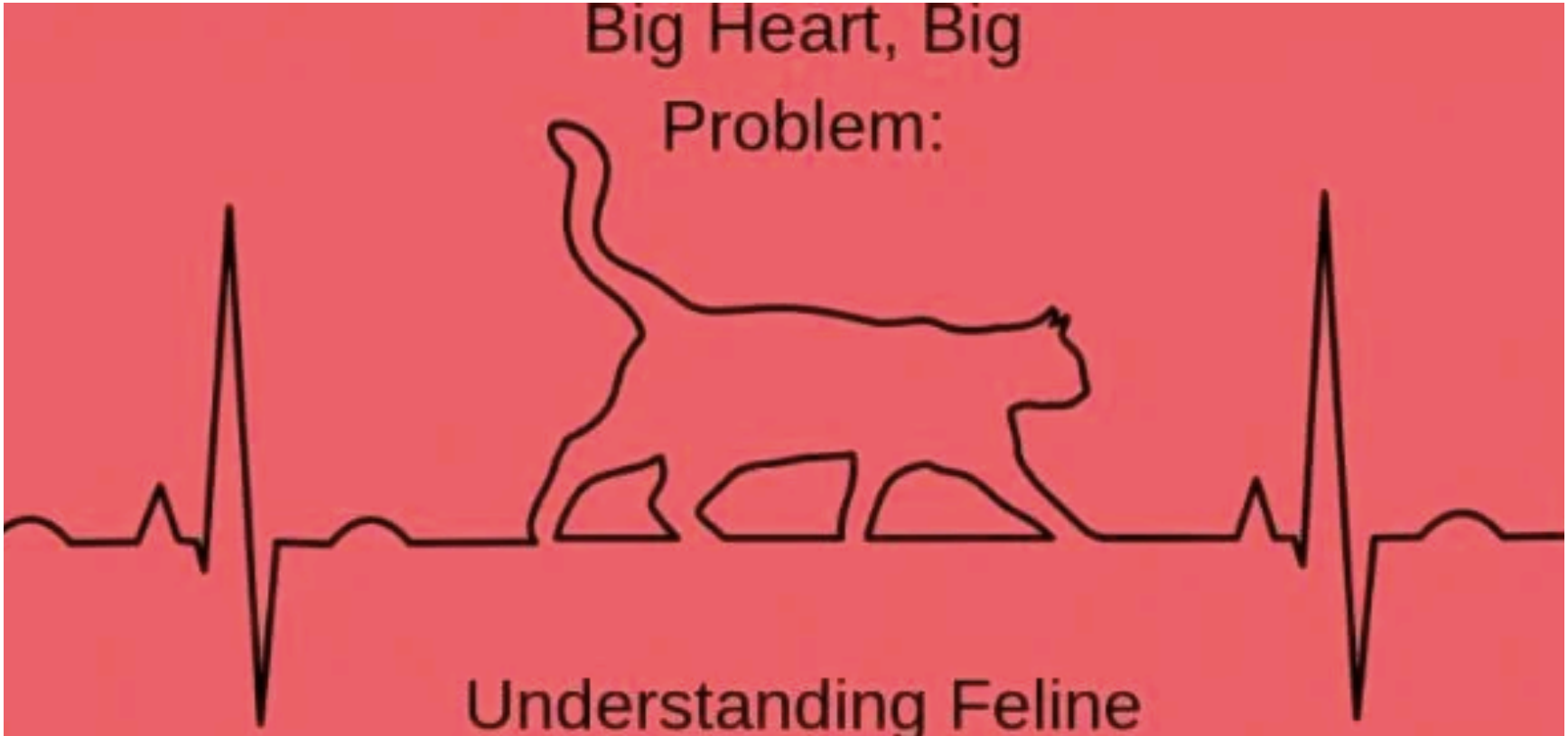
Management

- Treatment is primarily aimed at reducing or eliminating the (left ventricular outflow tract obstruction (LVOTO)) with **beta-blockers (e.g., atenolol: 1 mg/kg q.12–24h.)**, which slow the heart rate and decrease contractility.

Prognosis

The primary concern is sudden death; less commonly, dogs may develop CHF secondary to diastolic dysfunction and severe left atrial enlargement.

Big Heart, Big Problem:



Understanding Feline Hypertrophic Cardiomyopathy

1. **Hypertrophic Cardiomyopathy (HCM) i**n Cats: Most common form of cardiac disease in felines.

- Prevalence: Most common cardiomyopathy, accounting for 60% of cases.
- Higher prevalence in **certain breeds** (Maine Coon, Ragdoll, Norwegian Forest Cat).
- **Males are predisposed** (65%–70% of cases), possibly due to genetic or hormonal influences.

2. Restrictive Cardiomyopathy (RCM):

- **Characterized by restrictive diastolic filling with normal left ventricular dimensions and wall thickness.**

- Myocardial form and endomyocardial form (endocardial scar causing obstruction).
- **Prevalence: Second most common cardiomyopathy (20% of cases).**

3. Dilated Cardiomyopathy (DCM): Associated with dilation and impaired contractility of the left or both ventricles.

4. Arrhythmogenic Cardiomyopathy (ACM): Progressive fibrofatty replacement leading to dilated, hypocontractile right ventricle with arrhythmias. May also affect the left ventricle.

- Dilated Cardiomyopathy (DCM):

Historically linked **to taurine deficiency**; now rare due to dietary supplementation.

Current cases linked to nontraditional diets **(vegetarian, home-cooked, or canine food).**

- Restrictive Cardiomyopathy (RCM) and Arrhythmogenic Cardiomyopathy (ACM):

Possibly associated with **inflammatory processes from infectious, neoplastic, or immune-mediated disorders.**

The Role of Taurine in Cardiac Health in Dogs and Cats

Jennifer A. Larsen, DVM, MS, PhD, DACVN*, Andrea J. Fascetti, VMD, PhD, DACVIM (SA), DACVN
Department of Molecular Biosciences, School of Veterinary Medicine, University of California, Davis, 1089 Veterinary Medicine Drive, Davis, CA 95616, USA

KEYWORDS
• Taurine • Dilated cardiomyopathy • Nutritional requirements • Sulfur amino acids • Canine • Feline

KEY POINTS

- Taurine deficiency has been linked to development of dilated cardiomyopathy in both cats and dogs.
- In contrast to cats that have a dietary taurine requirement, dogs have the metabolic capacity to synthesize taurine from cysteine and methionine, so taurine is not a required amino acid for them.
- In species able to do so, endogenous synthesis of taurine is highly variable between individuals. Synthesis is impacted by an individual's nutritional state, protein intake, and cysteine availability.
- The precise physiologic roles of taurine remain largely uncharacterized, although a major function is the conjugation of bile acids, for which both dogs and cats are obligated to use solely taurine.
- Dietary factors that influence availability and utilization of sulfur-containing metabolites, pathway intermediates, methyl donors such as choline, and enzyme cofactors such as vitamins potentially also play a role in the development of dilated cardiomyopathy suspected to be related to diet, and full characterization of these impacts largely remain unexplored.

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The effect of taurine supplementation on the renin–angiotensin–aldosterone system of dogs with congestive heart failure

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The role of taurine in the treatment of congestive heart failure (CHF) in dogs without systemic deficiency is unexplored. Taurine might have beneficial cardiac effects aside from deficit replacement. We hypothesized that oral taurine supplementation administered to dogs with naturally-occurring CHF would suppress the renin-angiotensin aldosterone system (RAAS). Oral taurine was administered to 14 dogs with stable CHF. Serum biochemical variables, blood taurine concentrations, and comprehensive analysis of RAAS variables were compared before and 2 weeks after taurine supplementation added to background furosemide and pimobendan therapy for CHF. Whole blood taurine concentrations increased after supplementation (median 408 nMol/mL, range 248–608 before and median 493 nMol/mL, range 396–690 after; *P* = .006). Aldosterone to angiotensin II ratio (AA2) was significantly decreased after taurine supplementation (median 1.00, range 0.03–7.05 before and median 0.65, range 0.01–3.63 after; *P* = .009), but no other RAAS components significantly differed between timepoints. A subset of dogs showed marked decreases in RAAS metabolites after supplementation and these dogs were more likely to have been recently hospitalized for CHF treatment than dogs that did not show marked decreases in classical RAAS metabolites. Overall, taurine only lowered AA2 in this group of dogs, however, response heterogeneity was noted, with some dogs showing RAAS suppression.

HCM

- Genetic Mutations:

Mutations in the myosin-binding protein C (MyBPC3) gene identified in Maine Coon and Ragdoll breeds.

- **Secondary Hypertrophy Causes:**

Pressure overload (outflow obstruction, systemic hypertension) (increasing preload)

Hyperthyroidism

Infiltrative myocardial diseases

- **Diastolic Dysfunction in HCM and RCM:**

Primary cause of clinical signs in both HCM and RCM.

Left ventricular hypertrophy impairs/delays relaxation.

Interstitial/endomyocardial fibrosis increases stiffness, reducing distensibility.

Impaired filling results in elevated pressures, leading to left atrial enlargement, pulmonary venous hypertension, and congestive heart failure (CHF).

Clinical Presentation and Auscultatory Findings

Asymptomatic Phase:

- Cats may be presented due to auscultatory abnormalities (heart murmur or gallop sound).
- Prevalence of Murmurs: Nearly 50% of healthy cats with murmurs may not have cardiac disease.
- Presence of a murmur warrants further evaluation.

Silent Disease:

- 70% of cats with HCM may not have auscultatory abnormalities.

Occult Cardiomyopathy and Clinical Signs

- Often not identified until clinical signs appear, especially for RCM, DCM, ACM, nonspecific phenotype.
- Dyspnea, tachypnea, lethargy, poor appetite, hind limb paresis, collapse, sudden death.
- Clinical signs are not inevitable in individuals with HCM.

DDX

- Secondary to systemic diseases: hypertension, hyperthyroidism, hypersomatotropism.
- Degenerative valve disease, congenital heart disease, myocarditis.
- Respiratory Signs Differentials:

Feline lower airway disease (asthma), pneumonia, noncardiogenic pleural effusion or pulmonary edema, hemorrhage, pulmonary thromboembolism (PTE).

Diagnosis:

Diagnosis based on imaging and electrocardiographic tests.

Excludes other etiologies such as hyperthyroidism and systemic hypertension.

Echocardiography as the Gold Standard

Echocardiographic Evaluation:

Diastolic wall thickness > 6 mm on 2D or M-mode echocardiography.

Some cats may have wall thickness between 5 and 6 mm.

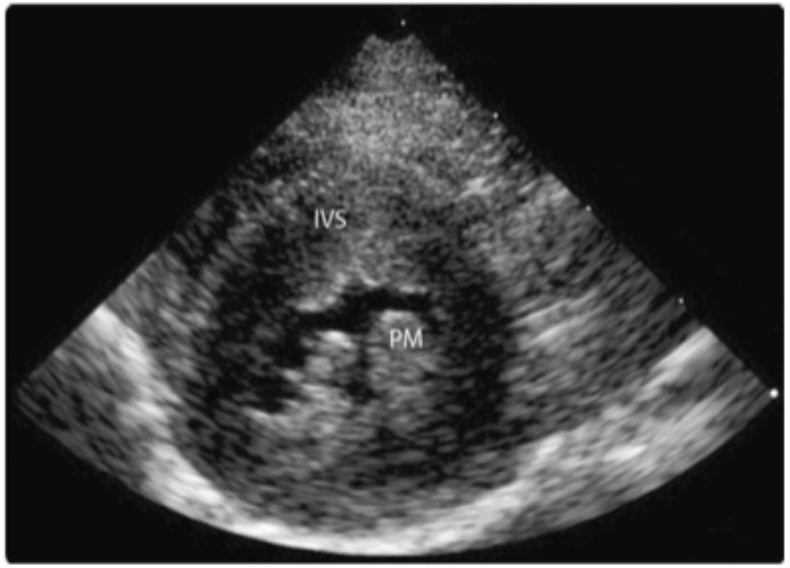

FIGURE 8.39 Right parasternal short-axis echocardiographic view from a cat with hypertrophic cardiomyopathy. There is concentric hypertrophy of the interventricular septum and the left ventricular free wall. The papillary muscles are prominent. IVS, interventricular septum; PM, papillary muscle.

Thoracic Radiographs in Cardiomyopathy Diagnosis

- May identify cardiomegaly, but changes can be subtle without atrial enlargement.
- Classic Appearance: "Valentine shape" indicates severe biatrial enlargement.
- Pulmonary venous congestion
- Patchy or diffuse interstitial to alveolar patterns
- Pleural effusion

Electrocardiogram (ECG) Findings

- Chamber Enlargement

- Common Arrhythmias and Abnormalities: Various arrhythmias and conduction abnormalities.

- Left anterior fascicular block is most common in cats with HCM.

- Supraventricular Tachycardia and Atrial Fibrillation: May occur secondary to severe atrial enlargement, though AF is rare.

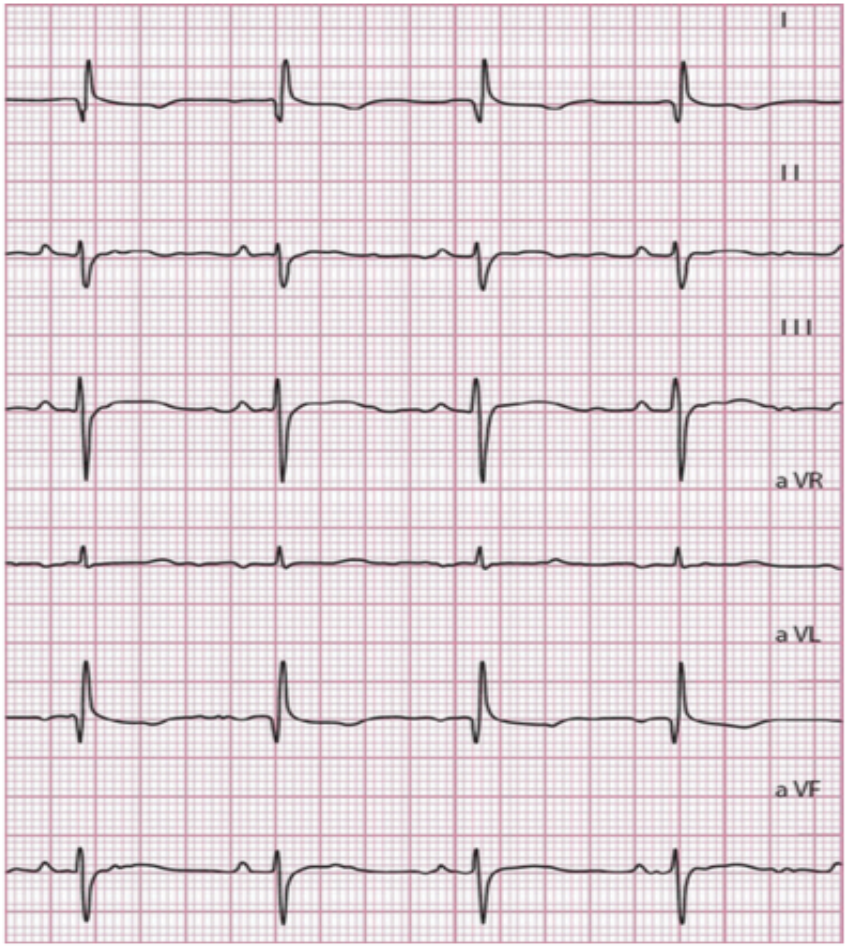

FIGURE 8.41 ECG from a cat with hypertrophic cardiomyopathy showing a sinus rhythm with a left axis deviation (lead I mostly positive, lead a VF mostly negative), also called left anterior fascicular block.

- Biomarkers in Feline HCM Diagnosis

NT-proBNP:

- A sensitive and specific screening tool for subclinical HCM.
- Can differentiate between CHF-related respiratory signs and primary respiratory disease.
- Elevated levels warrant further diagnostic evaluation.
- Normal NT-proBNP should not rule out cardiac disease if other supportive findings exist.

Cardiac Troponin I (cTnI)

- May be elevated in cats with moderate-to-severe HCM, with or without CHF.

TABLE 8.13 Drugs used in the management of cardiomyopathy in cats

Drug	Dosage	Comment
Atenolol	0.5–2 mg/kg p.o. q.12–24h.	In cats with dynamic left ventricular outflow tract obstruction with relatively normal LA size Consider long-term treatment if improvement of hypertrophy Start with low dose and increase slowly over a few weeks Consider for complex ventricular arrhythmias
Enalapril, benazepril	0.25–0.5 mg/kg p.o. q.12–24h.	Start q.24h.
Furosemide	1–2 mg/kg IM or IV q.1–8h. 1–3 mg/kg p.o. q.8–24h.	Monitor BUN, creatine, and blood potassium concentration
Torsemide	0.1–0.2 mg/kg p.o. q.24h.	Monitor BUN, creatine, and blood potassium concentration
Pimobendan	0.25 mg/kg p.o. q.12h.	Administered in the absence of significant LVOTO when CHF is present
Clopidogrel	18.75 mg p.o. q.24h.	In cats after episode of thromboembolism or if severe left atrial enlargement +/– visible thrombus or SEC

Stage A - At Risk Cats

- Cats predisposed to cardiomyopathy based on breed (e.g., Maine Coon, Ragdoll).
- Genetic testing for specific mutations (if applicable).
- Routine echocardiograms recommended for monitoring.

Stage B1 - Low-Risk Cardiomyopathy

- Cats with evidence of cardiomyopathy but without moderate-to-severe left atrial (LA) enlargement.
- Considered low risk for congestive heart failure (CHF) or arterial thromboembolism (ATE).
- Typically, no treatment recommended.
- Atenolol may be considered for severe left ventricular outflow tract obstruction (LVOTO), though it does not prolong survival.

Stage B2 - Increased Risk of CHF or ATE

- Asymptomatic cats with cardiomyopathy and increased risk for CHF or ATE.
- Moderate-to-severe LA enlargement, low LA fractional shortening (FS%).
- Clopidogrel for thromboprophylaxis; consider additional antithrombotic drugs (aspirin, factor Xa inhibitors).
- ACE inhibitors have not been shown to extend time to CHF; pimobendan studies are lacking.
- Atenolol for complex ventricular ectopy.
- Diltiazem, atenolol, or sotalol for atrial fibrillation (AF) with fast ventricular response rate.

Stage C - Cats with CHF or ATE

- Cats currently experiencing or with a history of congestive heart failure (CHF) or arterial thromboembolism (ATE).
- Furosemide: Primary diuretic therapy.
- Clopidogrel: Antithrombotic therapy.
- ACE Inhibitors: Used by some cardiologists, though evidence of effectiveness is limited; combined with furosemide in non-advanced age or non-azotemic cats.
- Pimobendan: Considered in the absence of significant left ventricular outflow tract obstruction (LVOTO).
- Spironolactone: May also be considered.

ATE Management:

- Provide pain medication and antithrombotic therapy (clopidogrel, heparin).
- Taurine supplementation in cats with dilated cardiomyopathy (DCM) phenotype.
- Regular monitoring of serum creatinine and electrolyte concentrations.
- Potassium supplementation for hypokalemic cats.

Stage D - Refractory CHF

- Cats refractory to CHF treatment, requiring high doses of furosemide (>6 mg/kg/day).
- Torsemide: Consider replacing furosemide, starting at 0.1–2.0 mg/kg orally every 24 hours.
- Pimobendan: Recommended for cats with decreased global systolic function.
- Taurine: Recommended unless taurine levels are normal.

Prognosis for Cats with HCM

- Asymptomatic cats may live several years post-diagnosis.
- Some cats with mild HCM do not experience progression and live a normal lifespan.
- Others may experience aggressive disease progression, sudden death at a young age, CHF, or thromboembolic complications.

Risk Factors for Cardiac Death

- Extreme hypertrophy (> 9 mm)
- Decreased left ventricular systolic function (FS < 30%)
- Decreased left atrial systolic function (↓ left atrial FS%)
- Survival Rates: Approximately 80% survive to discharge after acute CHF.
- Impact of Systolic Dysfunction: Historically poor prognosis, potentially improved with pimobendan.
- General Prognosis: First episode of heart failure: 6–12 months survival, variable outcomes.
- Some cats survive 2–3 years.

- Prognosis in Thromboembolic Complications:

Poor long-term prognosis with average survival of 6 months.

- Up to 30% mortality during immediate hospitalization.

- Some cats survive for several years.