REVIEW ARTICLE

Cardiomyopathies in veterinary medicine: A comprehensive review across canine, feline, and equine species

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ABSTRACT

Cardiomyopathies constitute a major cause of morbidity, mortality, and performance limitation across veterinary species. Although the overarching concept of myocardial disease spans veterinary and human medicine, its phenotype, clinical consequences, diagnostic pathways, and therapeutic strategies differ substantially between dogs, cats, and horses. Dilated cardiomyopathy (DCM) and arrhythmogenic cardiomyopathy (ACM/ARVC) dominate in predisposed dog breeds and reflect a combination of heritable, nutritional, and metabolic influences. Feline cardiomyopathy is overwhelmingly defined by hypertrophic cardiomyopathy (HCM), a disease shaped by diastolic dysfunction, left atrial enlargement, and risk of arterial thromboembolism (ATE). Restrictive cardiomyopathy (RCM), though less common, carries a particularly poor prognosis due to its severe impairment of diastolic filling. In horses, clinically significant cardiomyopathies are predominantly acquired, often arising from myocarditis, systemic disease, or maladaptive athletic remodeling associated with high-performance training. Performance decline, exercise-induced arrhythmias, or poor recovery rather than overt heart failure often herald equine myocardial disease. Diagnosis across species is anchored in echocardiography, supported by electrocardiography, Holter monitoring, and cardiac biomarkers such as troponin and NT-proBNP. Therapeutic approaches range from evidence-based interventions—such as pimobendan in preclinical canine DCM and clopidogrel for feline ATE prophylaxis—to largely empirical management of equine myocarditis and arrhythmia. Early identification of high-risk patients, understanding species-specific disease trajectories, and recognizing which interventions meaningfully alter prognosis are central to improving long-term outcomes. This review synthesizes current knowledge regarding the biology, clinical expression, diagnostic evaluation, and evidence-supported treatment of cardiomyopathies in dogs, cats, and horses. Emphasis is placed on practical clinical decision-making, early recognition, and integration of species-specific considerations important for general practitioners, cardiologists, and equine performance clinicians.

Keywords: Cats, dogs, horses, echocardiography, ventricular dysfunction

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List of Abbreviations

ACM/ARVC –	Arrhythmogenic cardiomyopathy / Arrhythmogenic right ventricular cardiomyopathy
ACTH –	Adrenocorticotropic hormone
ATE –	Arterial thromboembolism
CHF –	Congestive heart failure
DCM –	Dilated cardiomyopathy
DNA –	Deoxyribonucleic acid
ECG –	Electrocardiogram
ELISA –	Enzyme-linked immunosorbent assay
HCM –	Hypertrophic cardiomyopathy
HOCM –	Hypertrophic obstructive cardiomyopathy
LA –	Left atrium
LV –	Left ventricle
LVOTO –	Left ventricular outflow tract obstruction
NT-proBNP –	N-terminal pro–B-type natriuretic peptide
PCR –	Polymerase chain reaction
RNA –	Ribonucleic acid
RCM –	Restrictive cardiomyopathy
VPC –	ventricular premature complex
VT –	Ventricular tachycardia

INTRODUCTION

Cardiomyopathies in veterinary medicine encompass a wide spectrum of primary myocardial disorders that impair systolic function, restrict diastolic filling, alter ventricular geometry, or predispose the heart to arrhythmias and sudden death. Although rooted in universal principles of myocardial pathophysiology, veterinary cardiomyopathies express themselves through species-specific patterns influenced by genetics, body size, athletic physiology, and even environmental and nutritional factors (Sasaki et al., 2017; Fuentes et al., 2020; Grzeczka et al., 2022). Dogs predominantly develop dilated cardiomyopathy (DCM) or arrhythmogenic right ventricular cardiomyopathy (ARVC), diseases that are often heritable and may remain occult for long periods (Payne et al., 2015; Meurs, 2017; Meurs et al., 2014; Zibetti et al., 2025). Cats most frequently develop hypertrophic cardiomyopathy (HCM), which is the leading cause of cardiac morbidity in the species and closely mirrors aspects of human HCM in its diastolic abnormalities and risk of thromboembolism (Kittleson and Côté, 2021; Grzeczka et al., 2024). Horses, in contrast, primarily experience acquired cardiomyopathies associated with myocarditis, systemic inflammatory conditions, toxic injury, or the cumulative effects of intensive athletic training (Mogg, 1999; Avison et al., 2025).

The impact of cardiomyopathy extends beyond structural changes to include debilitating clinical syndromes such as congestive heart failure (CHF), life-threatening arrhythmias, poor performance, collapse, and sudden cardiac death (Summerfield et al., 2012; Cunningham and Dos Santos, 2022). These outcomes have substantial welfare, economic, and performance implications, especially for working animals and equine athletes. Early diagnosis is critical but complicated by the often-silent progression of disease, particularly in cats and in certain dog breeds such as Doberman Pinschers (Matos et al., 2024; Shen et al., 2022).

Advances in veterinary cardiology have transformed diagnostic capabilities. Echocardiography is central to phenotype identification, while Holter monitoring and exercise ECG have become indispensable in recognizing arrhythmogenic conditions in dogs and horses (Grden et al., 2020; Meurs, 2017; Meurs et al., 2014; Zibetti et al., 2025). Cardiac biomarkers, including NT-proBNP and troponin, have facilitated screening and risk stratification in both small animals

and equine patients (Guillaumin, 2024; Nath et al., 2024). Recently, a relatively cost-effective, small, practical, and manageable smartphone-based digital stethoscope (DS) device has also been developed. It allows simultaneous recording of electrocardiographic and phonocardiographic traces directly on a smartphone (Bozzola et al., 2024). This represents a significant advantage over the traditional manual stethoscope, whose clinical utility is diminishing-particularly during the auscultation of cats that frequently purr throughout the respiratory cycle (Ferasin et al., 2022; Katica and Hadžimusić, 2023).

This review synthesizes current understanding of cardiomyopathies in dogs, cats, and horses (Table 1), emphasizing comparative pathophysiology, early recognition, evidence-based treatment, and prognosis. The goal is to provide a clinically oriented, species-specific guide for practitioners managing myocardial disease in their daily work.

Table 1 Overview of cardiomyopathy phenotypes across canine, feline, and equine species

DOGS (key phenotypes)	CATS (key phenotypes)	HORSES (key phenotypes)
Dilated CM (DCM)	Hypertrophic CM (HCM)	Myocarditis-related CM
Arrhythmogenic CM (ARVCM/ACM)	Restrictive CM	Exercise-associated maladaptive remodeling
	Risk of ATE	

Legend: ACM – Arrhythmogenic cardiomyopathy; ARVCM – arrhythmogenic right-ventricular cardiomyopathy; ATE – arterial thromboembolism; CM – cardiomyopathy; DCM – dilated cardiomyopathy; HCM – hypertrophic cardiomyopathy

Canine Cardiomyopathies

Dilated Cardiomyopathy

Overview and Pathogenesis

Dilated cardiomyopathy (DCM) is one of the most clinically important cardiac diseases in dogs, particularly in large and giant breeds such as Doberman Pinschers, Great Danes, Irish Wolfhounds, and Boxers (Peterson et al., 2021; Summerfield et al., 2012; Schober et al., 2022). It is characterized by progressive eccentric remodeling, reduced systolic function, activation of neurohormonal pathways, and eventual CHF or sudden arrhythmic death. Microscopically, DCM reflects myocyte degeneration, mitochondrial dysfunction, apoptosis, and interstitial fibrosis (Monnet et al., 1995; Shen et al., 2022).

Genetic predisposition is strongly supported, with heritable variants identified in several breeds (Meurs, 2017; Meurs et al., 2014; Zibetti et al., 2025). Nutritional factors, including taurine deficiency, carnitine deficiency in certain lines, and diet-associated cardiomyopathy linked to grain-free or legume-heavy diets—have gained increasing recognition (Shen et al., 2022). These nutritionally mediated forms may be reversible if identified early.

Clinical Presentation

The early course of DCM is frequently silent. Many dogs show no outward abnormalities until extensive myocardial degeneration has occurred (Summerfield et al., 2012; Sasaki et al., 2017). Owners may report subtle fatigue, decreased stamina, or occasional coughing. In Doberman Pinschers, ventricular arrhythmias often precede structural dilation, making syncope or episodic weakness the first presenting signs (Meurs, 2017; Cunningham and Dos Santos, 2022).

As disease progresses, dogs may develop left-sided CHF with pulmonary edema or biventricular failure with ascites. Auscultation may reveal a systolic murmur due to functional mitral regurgitation, a gallop rhythm, or arrhythmias. However, many dogs in the preclinical stage have completely normal auscultatory findings (Ware and Bonagura, 2020), highlighting the need for breed-based screening.

Diagnosis

Echocardiography is the gold standard for DCM diagnosis (Fig. 1), demonstrating LV dilation, reduced ejection fraction, fractional shortening, and often left atrial enlargement (Schober et al., 2022). Because normal LV dimensions differ among breeds, breed-specific reference ranges are essential (Fuentes et al., 2020).

Holter monitoring is critical, especially in Doberman Pinschers, to detect ventricular premature complexes (VPCs) and runs of ventricular tachycardia (VT) that may precede echocardiographic changes (Meurs, 2017; Cunningham and Dos Santos, 2022). A burden ≥300 VPCs/day is often considered abnormal in this breed (Meurs et al., 2014).

Cardiac biomarkers add value: NT-proBNP correlates with myocardial stretch, while cardiac troponin indicates active myocardial injury (Guillaumin et al., 2024).

Dietary history and taurine/carnitine profiling are indicated in potential nutritional DCM (Shen et al., 2022).

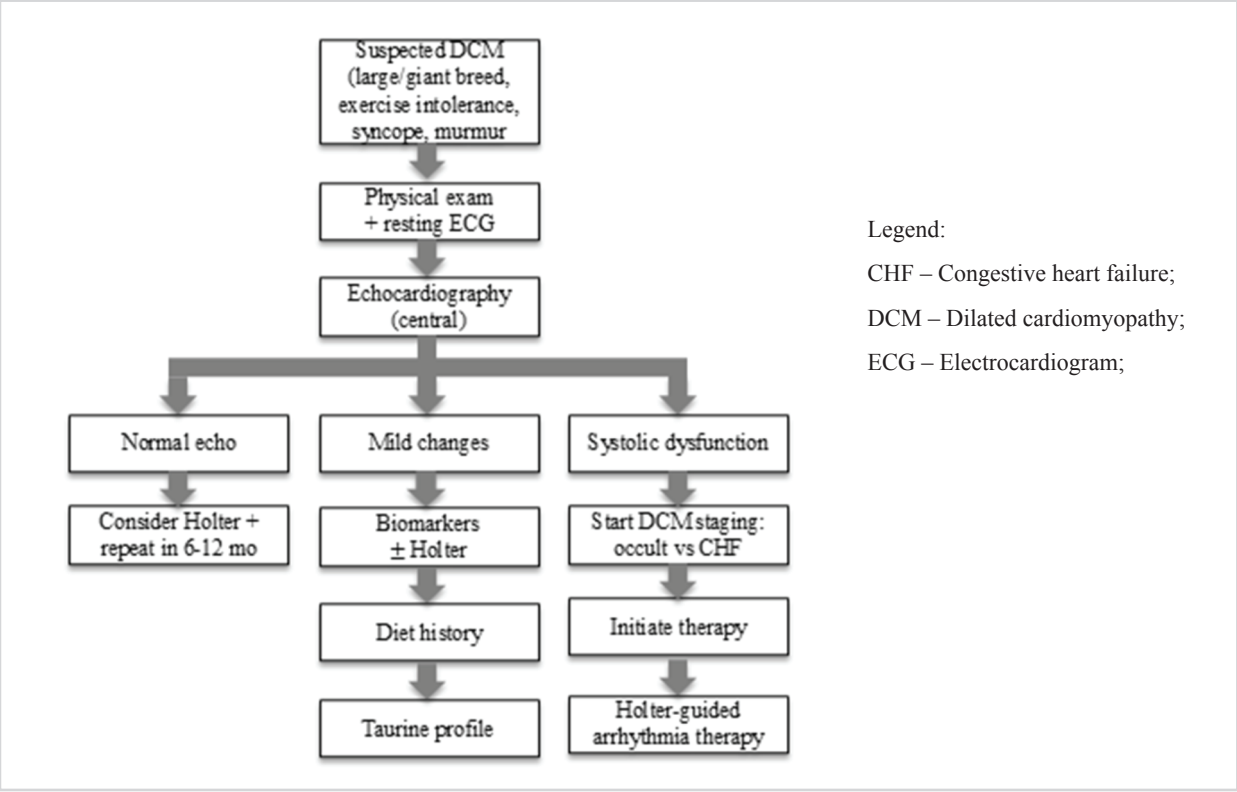


Figure 1 Diagnostic approach to suspected canine dilated cardiomyopathy

Therapy

Evidence-supported therapy for DCM is best established in dogs. The Protect study demonstrated that pimobendan significantly delays the onset of CHF or sudden death in Doberman Pinschers with occult DCM (Summerfield et al., 2012). Pimobendan is now the standard treatment once systolic dysfunction is documented.

In CHF, treatment includes furosemide, pimobendan, ACE inhibitors, and spironolactone (Fuentes et al., 2020). Arrhythmias requiring treatment may be managed with sotalol, mexiletine, or combination therapy (Cunningham and Dos Santos, 2022).

Nutritional DCM may improve with dietary correction and taurine/carnitine supplementation (Shen et al., 2022).

Prognosis

Outcomes vary by breed and disease stage. Doberman Pinschers often have the poorest prognosis due to malignant arrhythmias (Meurs, 2017). Once CHF develops, median survival typically ranges from several months to 1–2 years depending on response to therapy (Summerfield et al., 2012).

Arrhythmogenic Cardiomyopathy (ARVC/ACM)

Overview and Pathophysiology

Arrhythmogenic cardiomyopathy—classically arrhythmogenic right ventricular cardiomyopathy (ARVC)—is most commonly recognized in Boxer dogs but can occur in other breeds, including English Bulldogs and mixed breeds (Meurs, 2017; Cunningham and Dos Santos, 2022). It is defined by fibrofatty infiltration of the myocardium, particularly the right ventricle, creating an arrhythmogenic substrate that predisposes to ventricular premature complexes (VPCs), ventricular tachycardia (VT), syncope, and sudden death (Meurs et al., 2014). Genetic predisposition is strong (Ruiz-Guerrero and Barriales-Villa, 2018). A mutation in the striatin gene has been identified in Boxers, although genotype–phenotype correlations vary, and a negative genetic test cannot exclude disease (Adin et al., 2019; Meurs, 2017; Shen et al., 2022).

Clinical Presentation

ARVC is frequently occult during early stages. Many affected Boxers appear outwardly healthy and maintain normal activity levels despite significant arrhythmia burden (Cunningham and Dos Santos, 2022). The disease often manifests first as episodic syncope, weakness, or collapse. Owners may describe transient "staring spells," occasional disorientation, or short-lived episodes of unsteadiness following exertion (Meurs et al., 2014; Zibetti et al., 2025). Sudden death may be the initial presentation.

On examination, arrhythmias may be intermittently auscultated as irregular pulses or pulse deficits. Because resting ECG may be normal, ARVC often requires more intensive rhythm evaluation.

Diagnosis

Twenty-four-hour Holter monitoring is the cornerstone of ARVC diagnosis (Fig. 2). In Boxers, >100 VPCs/day is considered suspicious, and >300 VPCs/day increases concern for ARVC, while >1,000 VPCs/day or runs of VT strongly support diagnosis (Meurs, 2017; Cunningham and Dos Santos, 2022). Morphology of VPCs may be monomorphic or polymorphic, and complexity often correlates with disease severity.

Echocardiography can be normal early in the course but may later show right ventricular dilation, decreased right ventricular systolic function, or biventricular enlargement (Ware and Bonagura, 2020). In some dogs, ARVC progresses to a DCM-like phenotype with significant systolic dysfunction (Meurs, 2017).

Cardiac troponin may be mildly elevated in some cases, reflecting myocyte injury (Guillaumin, 2024).

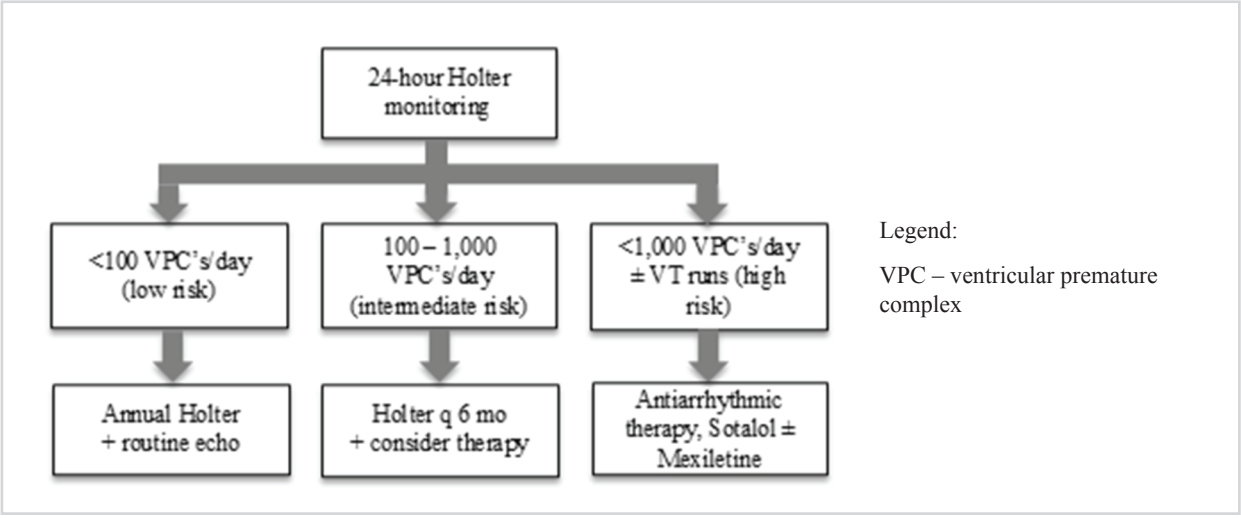


Figure 2 Holter-based ARVC risk stratification in Boxers

Treatment

Treatment focuses on suppression of ventricular arrhythmias and prevention of syncope or sudden death. Sotalol is typically first-line therapy and is effective in reducing VPC burden in many Boxers (Cunningham and Dos Santos, 2022). For severe cases, sotalol is combined with mexiletine. Atenolol is less effective for ARVC unless dynamic outflow obstruction also exists (Shen et al., 2022).

Exercise restriction is strongly recommended due to risk of VT during intense activity.

Dogs with structural progression may also require pimobendan and standard CHF therapy if systolic dysfunction evolves (Fuentes et al., 2020).

Prognosis

Prognosis varies considerably. Dogs with low to moderate arrhythmia burden can live many years with antiarrhythmic therapy (Cunningham and Dos Santos, 2022). Dogs with malignant VT, syncope, or biventricular involvement have significantly shorter survival times (Meurs, 2017).

Feline Cardiomyopathies

Hypertrophic Cardiomyopathy (HCM)

Overview

Hypertrophic cardiomyopathy is the most common cardiac disease of domestic cats, with prevalence estimates ranging from 10% to over 15% depending on population (Fuentes et al., 2020; Grzeczka et al., 2024). It is a primary myocardial disorder characterized by concentric left ventricular hypertrophy, impaired diastolic relaxation, and increased left atrial pressure that predisposes cats to CHF and arterial thromboembolism (ATE) (Guillaumin, 2024).

Genetic mutations in MYBPC3 are well documented in Maine Coon and Ragdoll breeds (Grzeczka et al., 2024), although the majority of domestic cats with HCM remain genetically uncharacterized.

Pathophysiology

Microscopically, HCM exhibits myocyte disarray, interstitial fibrosis, and small-vessel disease leading to ischemia and diastolic impairment (Casu and Prodi, 2014). These abnormalities increase ventricular stiffness, elevate filling pressures, and result in atrial stretch and enlargement (Sasaki et al., 2017). Some cats develop dynamic left ventricular outflow tract obstruction (LVOTO) due to systolic anterior motion (SAM) of the mitral valve—a subset known as HOCM (Fuentes et al., 2020).

Clinical Presentation

HCM is often silent. Many cats remain clinically normal for years, with diagnosis occurring incidentally during routine examination or pre-anesthetic screening (Hogan et al., 2015). When present, early signs include subtle lethargy, decreased jumping, or intermittent tachypnea during stress.

Progressive diastolic failure leads to CHF, manifesting as acute respiratory distress with open-mouth breathing, tachypnea, pulmonary edema, or pleural effusion (Fuentes et al., 2020). A gallop sound is a valuable and specific auscultation finding. Dynamic obstruction may produce a systolic murmur, although murmurs are not specific for HCM.

Aortic thromboembolism presents suddenly with hind limb pain, paralysis, hypothermia, and absent femoral pulses (Guillaumin, 2024).

Table 2 Echocardiographic differentiation of HCM, HOCM, and RCM in cats

HCM	HOCM	RCM
Thick LV walls	LV wall thickening	Normal/mild thickening
Small LV cavity	SAM of mitral valve	Severe LA enlargement
LA enlargement	LVOTO gradient ↑	Restrictive filling
Diastolic dysfunction	± Systolic murmur	Diastolic failure

Legend: ACM – CM – cardiomyopathy; HCM – hypertrophic cardiomyopathy; HOCM – hypertrophic obstructive cardiomyopathy; LA – left atrium; LV – left ventricle; LVOTO – left ventricle outflow tract occlusion; RCM – restrictive cardiomyopathy; SAM – systolic anterior motion

Diagnosis

Echocardiography is required to diagnose HCM (Table 2). Key findings include increased LV wall thickness (>5.5 mm in most cats), preserved or hyperdynamic systolic function, and left atrial enlargement (Fuentes et al., 2020). Doppler-echo evaluation identifies diastolic dysfunction or dynamic LVOTO.

Thoracic radiographs help confirm CHF but cannot differentiate cardiomyopathy forms.

NT-proBNP is valuable for distinguishing cardiac from respiratory causes of dyspnea (Guillaumin, 2024). Troponin elevation may indicate active myocardial injury and correlates with severity.

Treatment

Asymptomatic HCM

There is no proven disease-modifying therapy for asymptomatic cats without significant left atrial enlargement (Fuentes et al., 2020). Atenolol may be used in obstructive forms but has not demonstrated long-term benefit in non-obstructive disease.

Symptomatic HCM and CHF

Cats in CHF require:

- Oxygen
- Furosemide
- Thoracocentesis for pleural effusion

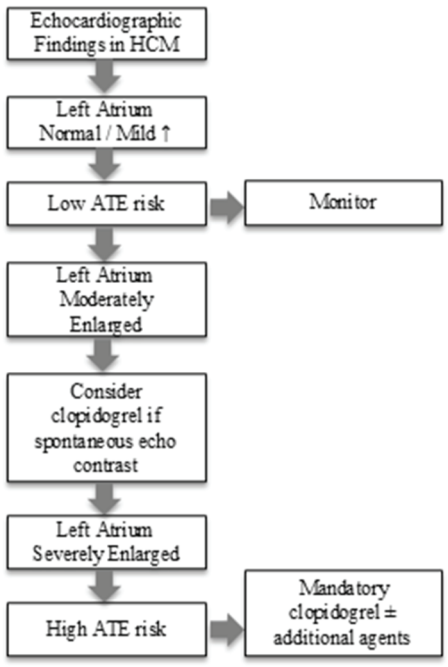
ACE inhibitors may be added, although evidence for their benefit remains mixed (Fuentes et al., 2020).

Atenolol or diltiazem can be used in HOCM to reduce LVOTO and improve filling (Casu and Prodi, 2014).

ATE Prevention

Clopidogrel is the preferred antithrombotic agent (Fig. 3), supported by the FAT CAT trial demonstrating superiority to aspirin (Hogan et al., 2015). Candidates include cats with:

- Severe LA enlargement
- Spontaneous echo contrast
- Previous ATE
- Markedly elevated NT-proBNP (Guillaumin, 2024)



Legend:

ATE – Arterial thromboembolism;
HCM – Hypertrophic cardiomyopathy

Figure 3 Risk stratification algorithm for arterial thromboembolism in cats

Prognosis

Prognosis is variable. Cats with mild HCM may live many years without progression (Fuentes et al., 2020). Severe left atrial enlargement or ATE confers a poor prognosis. Median survival after first CHF episode ranges from 6 months to 2 years, depending on management and severity (Hogan et al., 2015).

Restrictive Cardiomyopathy (RCM)

Overview and Pathophysiology

Restrictive cardiomyopathy is the second most important feline cardiomyopathy and is characterized by severe diastolic dysfunction with nondilated but stiff ventricles (Casu and Prodi, 2014). Endomyocardial fibrosis is a hallmark feature. Because the ventricle cannot fill properly, left atrial enlargement is typically marked.

Clinical Presentation

RCM frequently presents with CHF, significant left atrial enlargement, and high risk of ATE (Fuentes et al., 2020). Clinical signs mirror advanced HCM, including tachypnea, dyspnea, anorexia, or acute hind limb paresis.

Diagnosis

Echocardiography shows large atria, normal or minimally thickened ventricular walls, and restrictive transmitral flow patterns (Casu and Prodi, 2014). Radiographs reveal pulmonary edema or pleural effusion in CHF.

Treatment and Prognosis

Therapy parallels HCM with CHF: diuretics, cautious vasodilators, and antithrombotic therapy (Hogan et al., 2015). Ventricular compliance cannot be corrected, making prognosis guarded to poor. Median survival is typically shorter than HCM, often measured in months (Fuentes et al., 2020).

Equine Cardiomyopathies

Overview

Unlike dogs and cats, horses rarely develop primary inherited cardiomyopathies. Instead, most clinically important myocardial diseases in horses are acquired, arising from myocarditis, systemic disease, inflammation, toxins, or maladaptive remodeling secondary to intense athletic training (Mogg, 1999; Nath et al., 2024).

Cardiac disease in horses often first appears as decreased performance, poor recovery after exertion, abnormal sweating, or exercise-induced arrhythmias rather than overt signs of CHF (Avison et al., 2025).

Equine Cardiomyopathies (Continued)

Etiology and Pathogenesis

Myocarditis

Myocarditis is the most frequent cause of clinically relevant cardiomyopathy in horses and may arise from viral, bacterial, parasitic, immune-mediated, fungi or toxic etiologies (Mogg, 1999; Nath et al., 2024., Šaljić et al., 2020). Viral causes historically included equine influenza and equine herpesvirus, whereas bacterial sources include *Streptococcus spp., Clostridium spp.*, and endotoxemia associated with colitis or septicemia (Mogg, 1999). Parasitic burdens, including *Strongylus vulgaris* migration, may also contribute.

Inflammation results in myocyte necrosis, interstitial edema, and conduction abnormalities that predispose the horse to arrhythmias and exercise intolerance (Nath et al., 2024). Chronic myocarditis leads to myocardial fibrosis and long-term systolic dysfunction.

Systemic or Metabolic Injury

Systemic inflammatory disease, colic, endotoxemia, heat stroke, or severe electrolyte disturbances can cause myocardial injury through oxidative stress, calcium dysregulation, or microvascular dysfunction (Mogg, 1999). Horses with neurologic disease may develop catecholamine-associated myocardial injury similar to stress-induced cardiomyopathy in humans (Vitale et al., 2025).

Exercise-Associated Maladaptive Remodeling

Elite equine athletes undergo marked physiologic cardiac remodeling characterized by increased chamber size, wall thickness, stroke volume, and vagal tone (Avison et al., 2025). In some individuals, the transition from physiologic to maladaptive remodeling occurs when chronic high-intensity training produces fibrosis, conduction abnormalities, or arrhythmogenic substrate (Nath et al., 2024). The challenge for clinicians is distinguishing adaptive remodeling from early cardiomyopathy (Fig. 4).

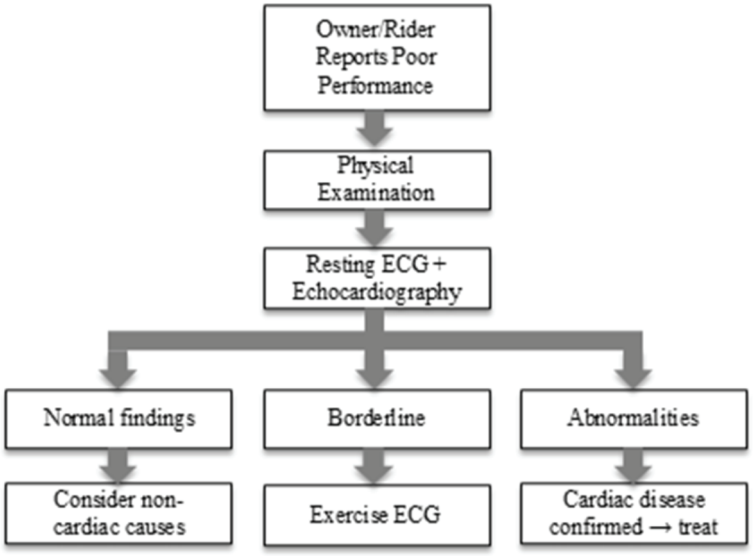


Figure 4 Algorithm 400 for Evaluating Performance Decline in Horses

Clinical Presentation

Clinical manifestations vary but often begin with:

- Performance decline (Avison et al., 2025)
- Exercise intolerance or early fatigue
- Excessive sweating or delayed recovery
- Exercise-induced arrhythmias (Nath et al., 2024)
- Coughing or respiratory effort (advanced CHF)
- Collapse or sudden death (severe arrhythmogenic disease)
- General weakness
- Tachycardia
- Uneven and irregular pulse
- Dyspnea
- Arterial hypotension

Because respiratory disease, orthopedic pain, or poor conditioning can mimic these findings, cardiac disease must be considered within a broad differential.

Diagnosis

History and Physical Examination

A detailed training history is essential. Duration of conditioning, competition level, frequency of high-intensity exercise, and recent systemic illness guide suspicion (Avison et al., 2025). Auscultation may reveal murmurs (often physiologic) or arrhythmias. Second-degree AV block at rest is common in healthy horses; persistence during exercise suggests underlying pathology (Nath et al., 2024).

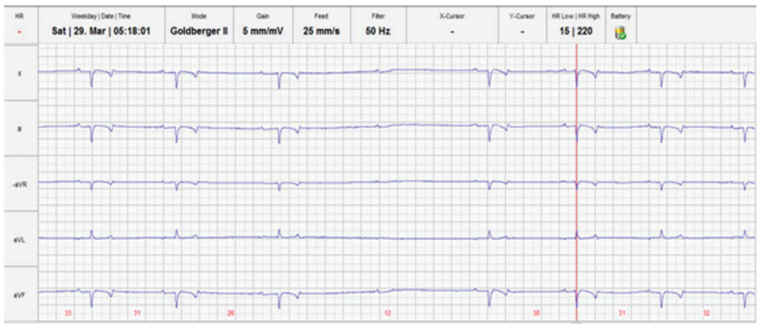


Figure 5 Second-degree AV block in a sport horse at rest

Electrocardiography

Resting ECG may identify premature atrial or ventricular complexes, but has limited sensitivity. **Exercise ECG** or continuous telemetry during exertion is critical for detecting arrhythmias that only manifest under load (Nath et al., 2024). Sustained or complex VT is highly concerning.

Echocardiography

Echocardiography evaluates chamber size, systolic function, wall motion, and pericardial effusion. Horses with maladaptive remodeling may demonstrate regional akinesia, hypokinesia, or reduced LV fractional shortening (Mogg, 1999; Nath et al., 2024). Significant atrial enlargement or valvular regurgitation (especially tricuspid) also raises concern.

Biomarkers

Cardiac troponin (Fig. 5) is an important indicator of myocardial injury (Nath et al., 2024). Mild increases may follow strenuous exercise, but persistent or marked elevations require further evaluation. NT-pro BNP is less well validated in horses but may assist in identifying myocardial stress.

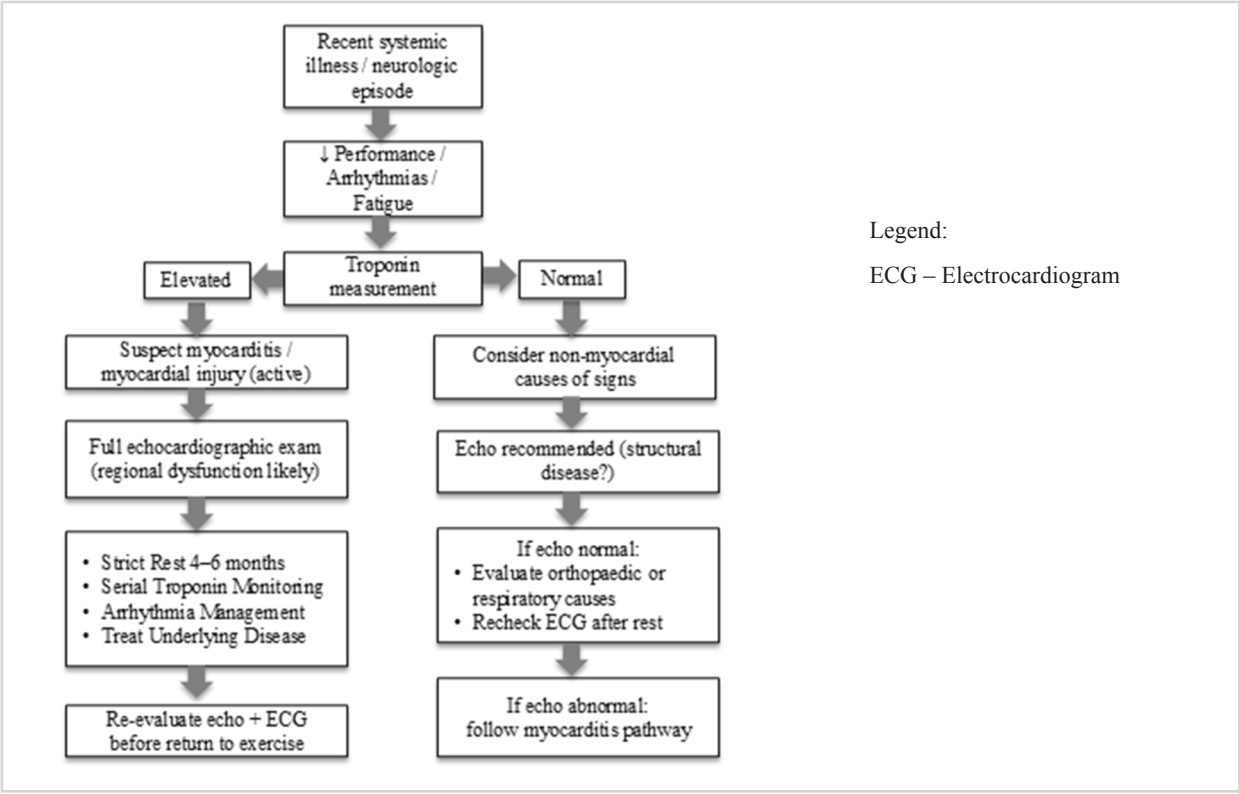


Figure 5 Diagnostic and management pathway for myocarditis-associated cardiomyopathy in horses

Additional Diagnostics

Endomyocardial biopsy is rarely feasible in equine practice due to procedural risks. Bloodwork may reveal inflammation, CK elevation, or electrolyte abnormalities.

Approach and Technique

Endomyocardial biopsy in horses is most commonly performed on the right ventricle.

The horse is kept standing and sedated

Vascular access is obtained via the jugular vein

A biopsy forceps (bioptome) is advanced into the heart under:

- echocardiographic guidance and/or
- fluoroscopic control (Decloedt et al., 2016).

Risks and Complications

Endomyocardial biopsy in horses is associated with several potential risks, including:

- Cardiac arrhythmias during the procedure
- Myocardial or ventricular wall perforation
- Hemopericardium
- Thromboembolism
- Infection (Decloedt et al., 2016).

Therapeutic Considerations

General Management Principles

Treatment of equine cardiomyopathy is often empirical and directed toward:

- **Resolving underlying disease**
- **Supporting myocardial recovery**
- **Controlling arrhythmias**
- **Preventing collapse during exercise**

Myocarditis Management

Strict rest is essential and often lasts 4–6 months (Mogg, 1999). Anti-inflammatory treatments, such as corticosteroids or NSAIDs, must be used judiciously due to variable evidence and risk of adverse effects. Specific therapy is indicated when an infectious cause is identified (Vitale et al., 2025).

Serial troponin measurements help track myocardial recovery.

Arrhythmia Management

Atrial fibrillation (AF) and ventricular arrhythmias can accompany cardiomyopathy.

- **AF** may be treated with quinidine or electrical cardioversion depending on duration and athletic expectations (Mogg, 1999).
- **Ventricular arrhythmias** require rest, correction of electrolyte disturbances, and in severe cases, antiarrhythmic therapy such as sotalol (Vitale et al., 2025).
- Exercise restriction is mandatory when ventricular arrhythmias are present.

Congestive Heart Failure

CHF is less common in horses but treated using furosemide, judicious vasodilators, and supportive care (Mogg, 1999). Long-term prognosis is guarded.

Comparative and Integrative Considerations Across Species

Distinguishing Primary vs Secondary Cardiomyopathy

Although cardiomyopathies in dogs, cats, and horses diverge in etiology, expression, and prognosis (Fig. 6), several unifying diagnostic and therapeutic principles apply across species. Understanding these integrative themes can improve clinical reasoning and the veterinarian's ability to recognize disease early.

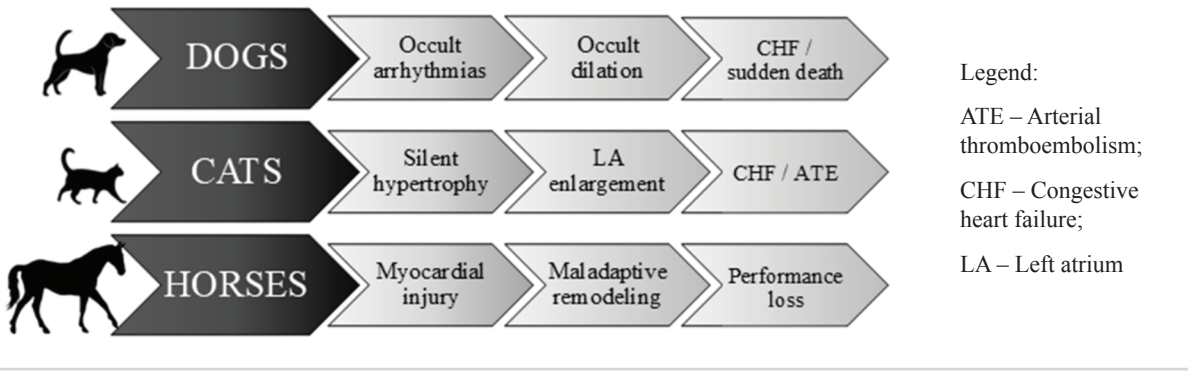


Figure 6 Comparative timeline of cardiomyopathy progression in dogs, cats, and horses

A recurring challenge is differentiating primary cardiomyopathy from secondary myocardial changes caused by systemic disease, pressure overload, endocrine disorders, or nutritional imbalance.

- **Cats:** Systemic hypertension, hyperthyroidism, followed by myocarditis, tumor infiltration (lymphoma), and pseudohypertrophy cause concentric hypertrophy that mimics HCM (Fuentes et al., 2020).
- **Dogs:** Taurine deficiency, carnitine imbalance, or diet-associated DCM can resemble idiopathic DCM but may be reversible (Shen et al., 2022).
- **Horses:** Systemic inflammatory disease or endotoxemia frequently precedes myocardial injury (Nath et al., 2024).

Correct classification determines prognosis and treatment approach.

Common Diagnostic Strategies

Echocardiography

Echocardiography is central across species. In dogs, LV dilation and reduced systolic function support DCM, whereas in cats, LV concentric hypertrophy with normal systolic function supports HCM (Fuentes et al., 2020). In horses, echo helps differentiate physiologic remodeling from cardiomyopathy-associated dysfunction (Nath et al., 2024).

ECG and Holter Monitoring

Holter monitoring is particularly valuable in:

- **Dobermans** for detecting occult DCM (Summerfield et al., 2012)
- **Boxers** for diagnosing ARVC (Meurs, 2017; Cunningham and Dos Santos, 2022)

In horses, exercise ECG is vital for arrhythmia detection (Avison et al., 2025).

Biomarkers

NT-proBNP supports differentiation between cardiac and non-cardiac dyspnea in cats and early detection of DCM in dogs (Guillaumin, 2024). Troponin is useful in detecting myocarditis across species (Nath et al., 2024; Vitale et al., 2025).

Therapeutic Principles Across Species

When Treatment Meaningfully Improves Outcomes

- **Dogs (DCM):** Pimobendan delays CHF and sudden death in occult DCM (Summerfield et al., 2012).
- **Cats (ATE):** Clopidogrel prevents ATE recurrence (Hogan et al., 2015).
- **Horses (Myocarditis):** Rest is the most critical intervention (Mogg, 1999).

When Treatment Has Limited Impact

- Advanced feline RCM often carries a poor prognosis despite optimal therapy (Casu and Prodi, 2014).
- Severe equine cardiomyopathy associated with extensive fibrosis responds poorly to medication (Nath et al., 2024).

Understanding these therapeutic boundaries is essential for compassionate, realistic client counseling.

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Kardiomiopatije u veterinarskoj medicini: Detaljan pregled kod pasa, mačaka i konja

SAŽETAK

Kardiomiopatije predstavljaju glavni uzrok morbiditeta, mortaliteta i ograničenja performansi kod različitih životinjskih vrsta. Iako opći koncept bolesti miokarda obuhvata i veterinarsku i humanu medicinu, njen fenotip, kliničke posljedice, dijagnostički put i terapijske strategije značajno se razlikuju između pasa, mačaka i konja. Kod pasa, dilatativna kardiomiopatija (DCM) i aritmogenetska kardiomiopatija (ACM/ARVC) dominiraju u predisponiranim rasama i odražavaju kombinaciju nasljednih, nutritivnih i metaboličkih faktora. Kardiomiopatije kod mačaka su uglavnom definisane hipertrofičnom kardiomiopatijom (HCM), bolesti oblikovane dijastolnom disfunkcijom, proširenjem lijeve pretkomore i rizikom od arterijske tromboembolije (ATE). Restriktivna kardiomiopatija (RCM), iako rjeđa, ima posebno lošu prognozu zbog ozbiljnog oštećenja dijastolnog punjenja. Kod konja, klinički značajne kardiomiopatije su pretežno stečene, te često nastaju kao posljedica miokarditisa, sistemske bolesti ili maladaptivnog atletskog remodeliranja povezanim sa intenzivnim treninzima. Pad performansi, aritmije izazvane vježbom ili loš oporavak, a ne očigledno zatajenje srca, često nagovještavaju srčanu bolest kod konja. Dijagnoza kod svih vrsta oslanja se na ehokardiografiju, uz podršku elektrokardiografije, Holter monitoringa i srčanih biomarkera kao što su troponin i NT-proBNP. Terapijski pristupi kreću se od intervencija zasnovanih na dokazima – poput pimobendana kod pretkliničke DCM kod pasa i klopidogrela za profilaksu ATE kod mačaka – do uglavnom empirijskog tretmana miokarditisa i aritmija kod konja. Rano prepoznavanje pacijenata visokog rizika, razumijevanje bolesti specifičnih za svaku vrstu i prepoznavanje intervencija koje značajno mijenjaju prognozu ključni su za poboljšanje dugoročnih ishoda. Ovaj pregledni rad objedinjuje trenutno znanje o biologiji, kliničkom ispoljavanju, dijagnostičkoj evaluaciji i terapiji kardiomiopatija kod pasa, mačaka i konja. Poseban naglasak stavljen je na praktično kliničko odlučivanje, ranu detekciju i integraciju specifičnih faktora za svaku vrstu, što je važno za veterinare opće prakse, kardiologe i kliničare u oblasti performansi konja.

Ključne riječi: Mačke, psi, konji, ehokardiografija, ventrikularna disfunkcija