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Acquired heart disease in dogs (Acquired heart disease in dogs)

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Acquired cardiac pathologies are the most frequently diagnosed type of heart disease in dogs.

The 2 main diseases observed in dogs are:

- **Dilated Cardiomyopathy (DCM): A disease that is often silent until clinical signs of heart failure appear, and the prognosis is then bleak.**
- **Myxomatous Mitral Valve Disease (MMVD): Accounts for up to 70% of canine heart diseases observed in general practice.**

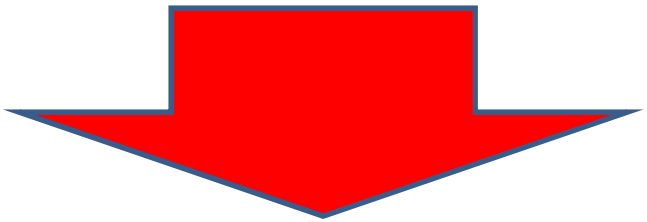
Both DCM and MMVD are diseases that most often affect the left side of the heart:

- The left heart operates under high pressures to supply blood throughout the body and maintain blood pressure.
- If the left heart is compromised, clinical signs of left congestive heart failure (CHF) or forward heart failure may occur.

Dilated cardiomyopathy (DCM)

- **Classification of cardiomyopathies**

- Cardiomyopathy is a term applied to all heart disorders caused by dysfunction of the myocardium.
- The classification of cardiomyopathies is difficult due to the absence, in many cases, of a defined etiology.
- The most commonly used classification system divides cardiomyopathies into primary and secondary:



- ✓ **Primary cardiomyopathies include genetic, non-genetic, and idiopathic diseases. The most common primary cardiomyopathies in dogs are CMD and Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC).**
- ✓ **Secondary cardiomyopathies are diseases of the myocardium that are consequences of congenital or acquired systemic diseases. The most common causes of secondary cardiomyopathies in dogs include medications, tachyarrhythmias, infectious agents, and systemic diseases.**

Classification of cardiomyopathies.

Primary
Dilated cardiomyopathy
Arrhythmogenic right ventricular cardiomyopathy
Secondary
Arrhythmia-induced
Metabolic : Diabetes, Systemic hypertension, Hypothyroidism, Acromegaly, Pheochromocytoma
Drugs and toxins : Chemotherapeutic agents : doxorubicin, Catecholamines, Cobalt, Gossypol, Ionophores
• ***Nutritional :*** Taurine deficiency, L-carnitine deficiency
• ***Myocarditis – Inflammatory:*** ▪ Infectious diseases: Virus : Canine parvovirus 2, Canine distemper virus/Bacterial: *Borrelia burgdorferi*, *Streptococcus* spp./ *Staphylococcus* spp./ Protozoal: *Trypanosoma cruzi*, *Toxoplasma gondii*, *Neospora*/ Fungal: *Cryptococcus*, *Coccidioides*, *Aspergillus* ▪ Noninfectious diseases

Definition of CMD

- Canine dilated cardiomyopathy (DCM) is a primary hereditary and progressive myocardial disease characterized by a decrease in systolic function (decreased contractility) and subsequent ventricular dilation.
- This disease is most often observed in large dog breeds (and giant breeds).
- Significant variations in the presentation, progression, and prognosis of the disease can be observed depending on the dog's breed.

Epidemiology

- Canine DCM is the most common cardiomyopathy in dogs, with a reported prevalence between 0.5% and 1.4% in the general canine population.
- The disease appears to have a breed predisposition, typically affecting large dogs and giant breeds, with a high prevalence in Dobermans, Great Danes, Newfoundlands, and Irish Wolfhounds.

- However, the disease has also been identified in some small and medium-sized dogs such as Portuguese Water Dogs, Cocker Spaniels, and Schnauzers.

Giant breeds	Large breeds	Spaniel breeds	Other
• Irish Wolfhound • Scottish Deerhound • Great Dane • Newfoundland • St Bernard • Leonberger	• Doberman • Boxer • Weimaraner • Dogue de Bordeaux • Golden Retriever • Labrador Retriever • Old English Sheepdog • German Shepherd Dog	• English Cocker Spaniel • English Springer Spaniel • American Cocker Spaniel	• Portuguese Water Dog

Irish Wolfhound

Great Dane

English Cocker
(English Cocker Spaniel)

Labrador Retriever

- The occult form is generally diagnosed in dogs aged 5 to 7 years, but younger dogs can also be affected.
- A juvenile form of CMD has been identified in Portuguese Water Dogs = Affected puppies generally die before the age of 7 months.

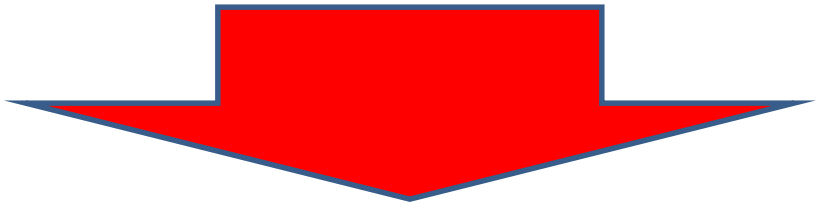
Portuguese Water Dog

- Males appear to be more frequently affected, particularly in Great Danes, while no difference in the sex ratio is currently reported in Dobermans and some breeds.

Doberman Pinscher Dog

○ Etiology

The cause of CMD is not known, although several theories regarding genetic, nutritional, metabolic, inflammatory, infectious, or drug- or toxin-induced myocardial diseases have been suggested.



❑ Genetic origin:

- A genetic (hereditary) cause is strongly suspected in most breeds and is documented in some (Portuguese water dog, boxer, Doberman) with variable modes of inheritance.
- The CMD phenotype may be the result of various myocardial injuries, which are often not identified, making many cases idiopathic.
- In dogs, these injuries are often genetic mutations leading to alteration of the structure and function of cardiac proteins, particularly in purebred breeds predisposed to CMD.

- **Nutritional deficiencies: Taurine (particularly in American cockers), carnitine.**
- **Boutique, exotic or grain-free (BEG) diets: The implications of these diets are not fully understood.**
- **Secondary to sustained tachyarrhythmia (tachycardia induced).**

- **Infectious and toxic causes of myocarditis, characterized by inflammation and myocardial necrosis, can produce a clinical picture resembling CMD (ventricular dilation, systolic dysfunction, arrhythmias) in the chronic phase, and can be considered the underlying injury in certain clinical presentations or geographical areas: Example**

Bacterial agents (Borrelia burgdorferi, Bartonella), viral (parvovirus), protozoan (Trypanosoma cruzi [Chagas disease], Toxoplasma gondii), fungal (Aspergillus, Blastomyces, Coccidioides), and toxic (anthracyclines).

NB:

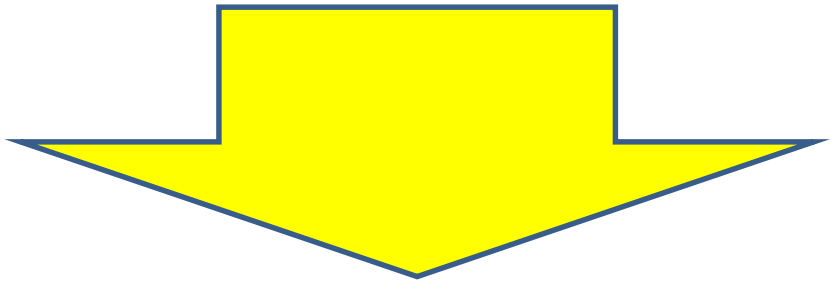
The non-familial and non-idiopathic causes mentioned above represent only a very small percentage of the total number of CMD cases, as idiopathic or familial cases are the most common.

Boxer

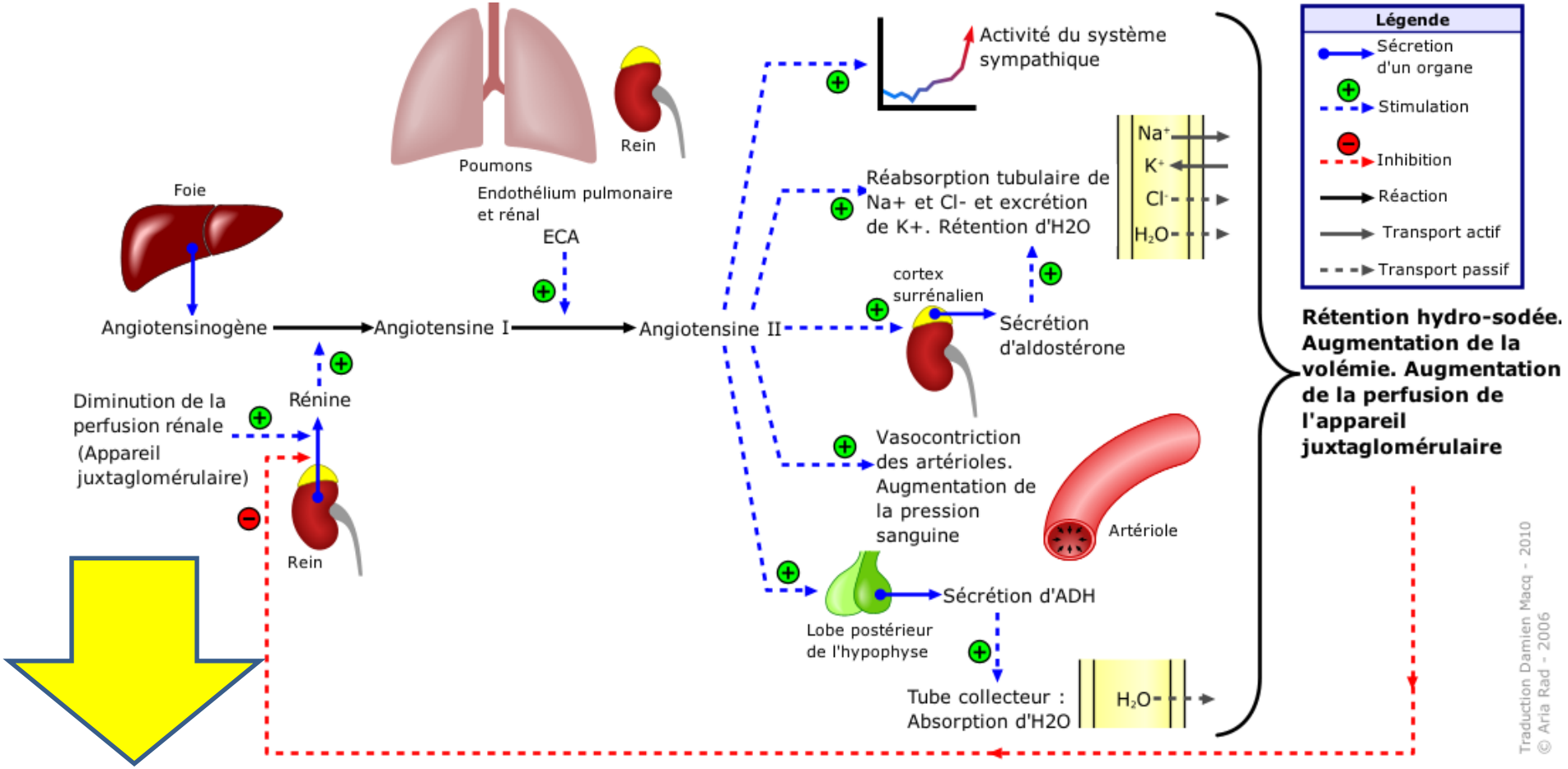
○ Pathophysiology

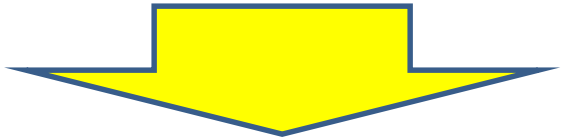
CMD affects the heart's ability to pump blood throughout the body because the myocardium (ventricle) is unable to contract properly (myocardial weakness).

The reduction in cardiac output leads to the activation of compensatory mechanisms:

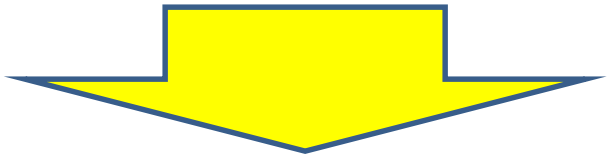


- Sympathetic stimulation causing tachycardia and vasoconstriction.
- Activation of the renin-angiotensin-aldosterone system to retain sodium and water, in order to maintain systemic blood pressure (BP).

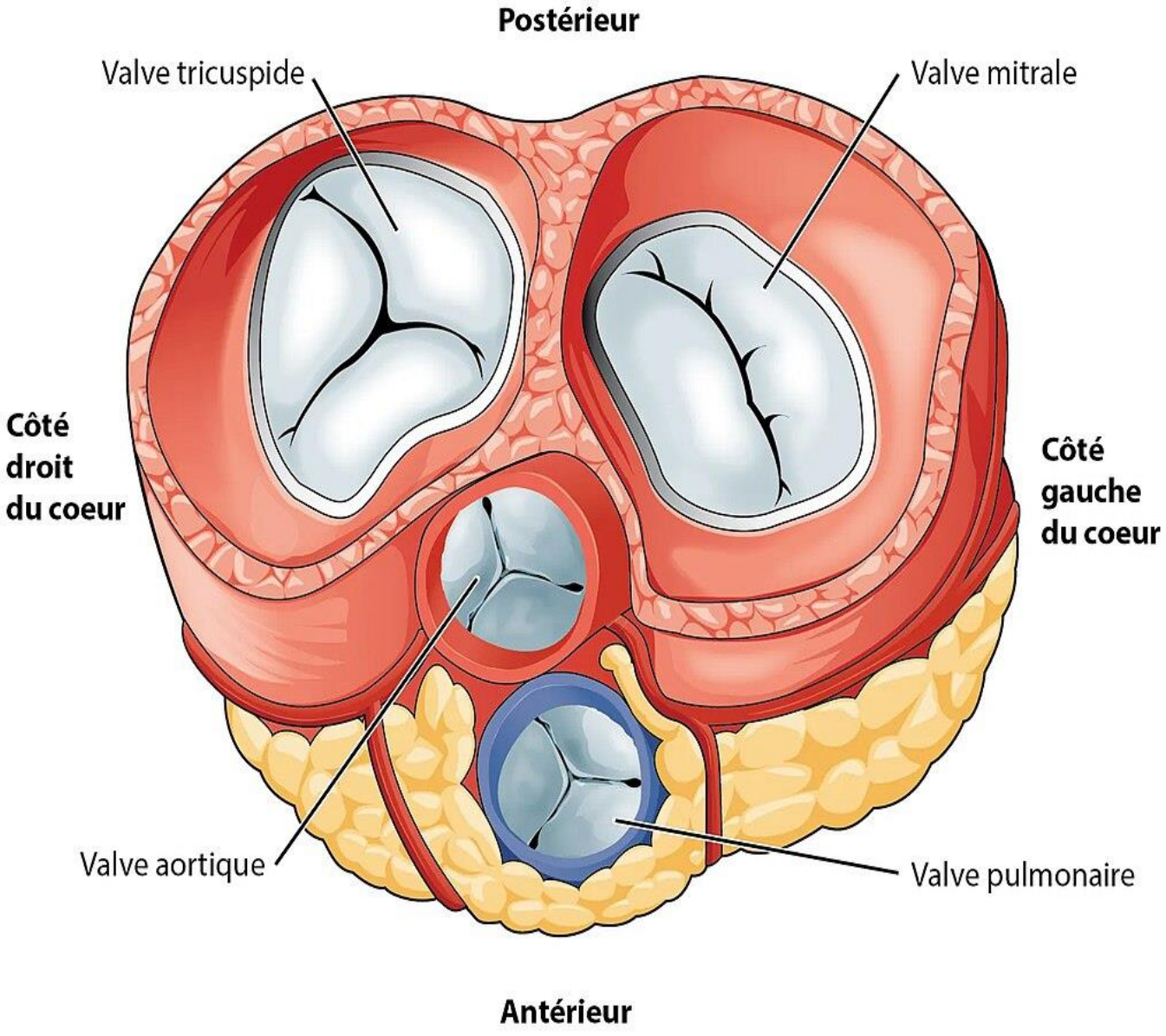




- These processes lead to an increase in circulating volumes.



- This can lead to ventricular dilation, often deforming the mitral valve apparatus (stretching of the mitral ring) and allowing blood to regurgitate into the left atrium each time the heart pumps in systole (mitral regurgitation).


Postérieur
Valve tricuspide
Valve mitrale
Côté droit du coeur
Côté gauche du coeur
Valve aortique
Valve pulmonaire
Antérieur

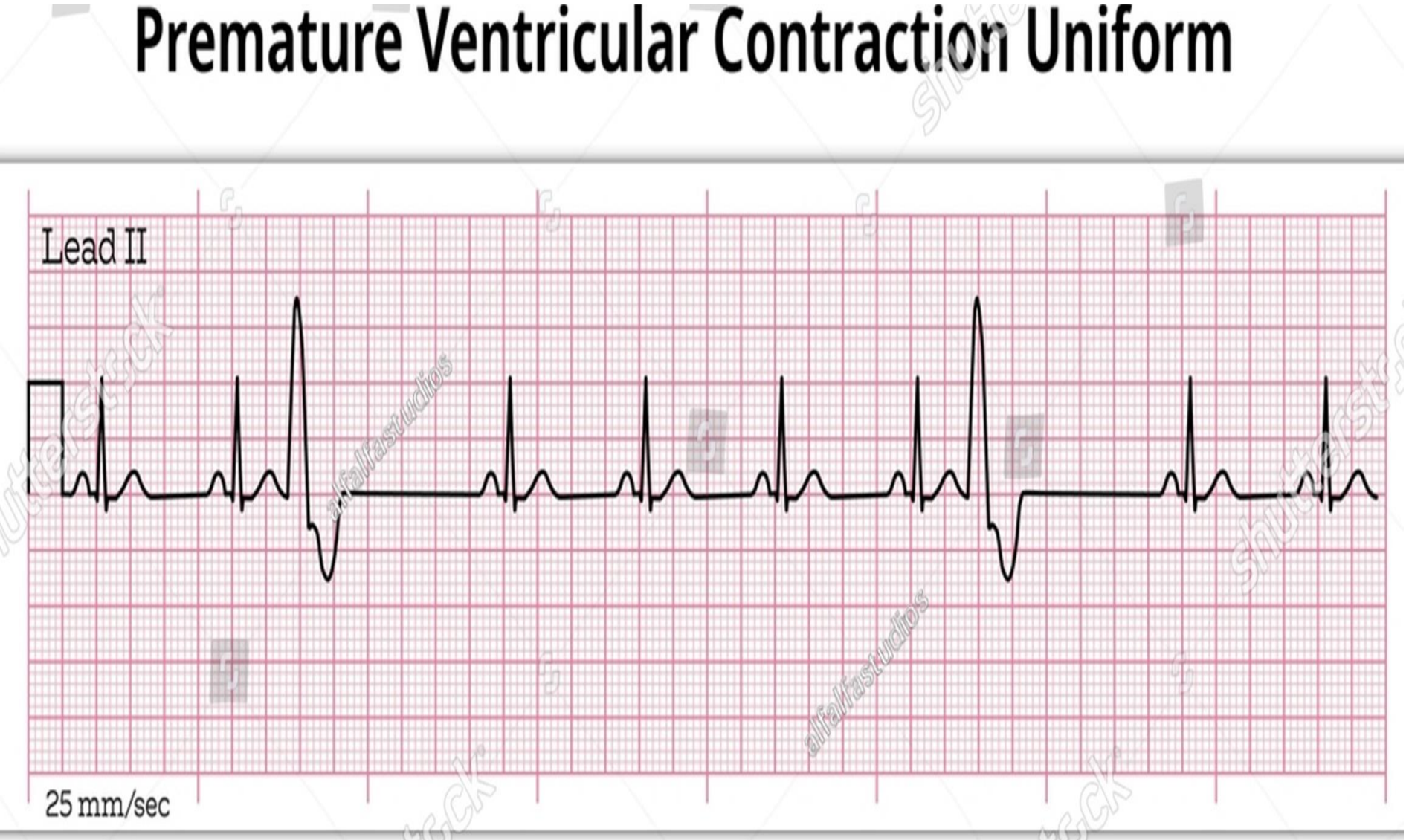
- The more the atria and ventricles dilate, the larger the regurgitation jet becomes, the more compensatory mechanisms are triggered.
- The additional fluid can cause pressure overload (fluid) in the lungs, leading to pulmonary edema and other clinical signs of left heart congestive heart failure (CHF).
- Many patients develop arrhythmias, both ventricular and supraventricular; prolonged arrhythmias can lead to the development of right-sided heart failure.
- Ventricular arrhythmias are common in patients with CMD and can lead to sudden death.

○ Stages of CMD

The stages of CMD are as follows:

- **First stage: Morphologically and electrically normal heart in dogs with a genetic predisposition.**
- **Second stage (occult or preclinical phase): Electrical abnormalities (ventricular extrasystoles for example) and/or myocardial anomalies but absence of clinical signs.**
- **Third stage (clinical phase or manifest phase): Clinical signs of heart failure and/or arrhythmias.**

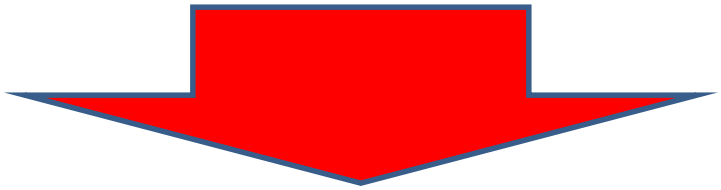
Ventricular extrasystoles



Clinical presentation

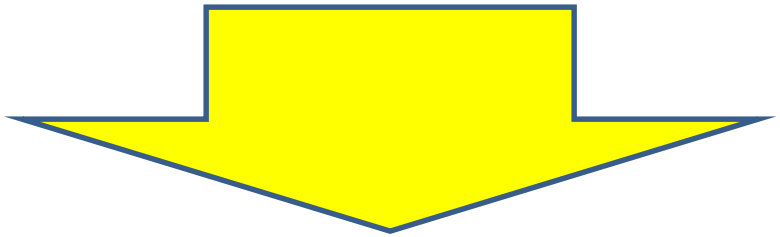
- In general, CMD starts in adulthood.
- Male dogs are more frequently affected.
- However, Portuguese water dogs often develop the disease before the age of 12 weeks.
- Clinical signs can vary from the absence of symptoms (asymptomatic) to heart failure or sudden death.
- The occult stage of the disease can evolve slowly, during which the only manifestations of CMD are echocardiographic and electrocardiographic anomalies.

- In some dogs, sudden death may be the only sign of the disease.



This is especially true for dobermans, where sudden death may be the only sign of the disease in about 30 to 50% of affected dogs.

Historical clues



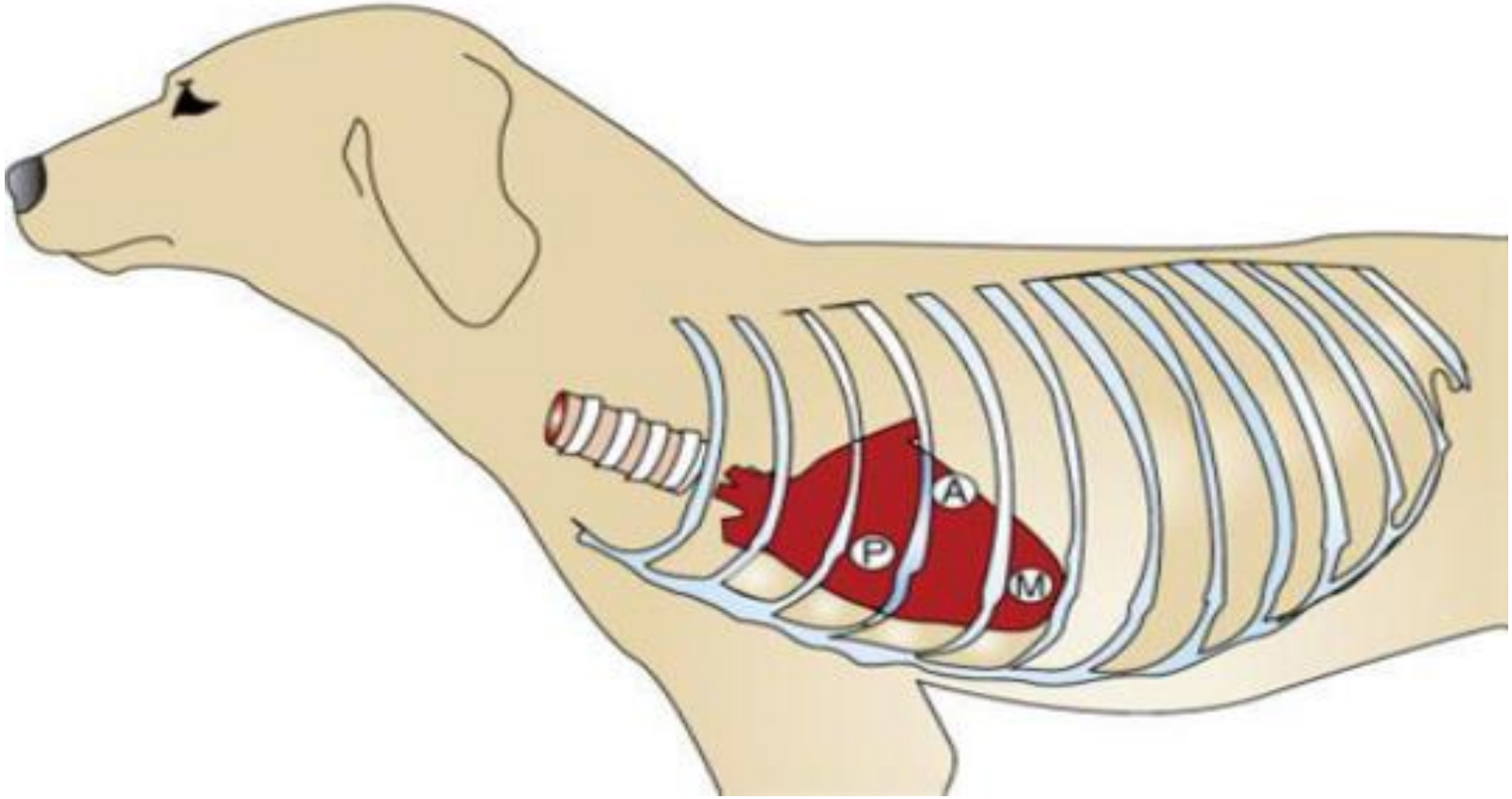
- May be asymptomatic.
- Syncopal episodes.
- Development of a murmur in sensitive breeds.
- Development of CHF in sensitive breeds.

Clinical signs

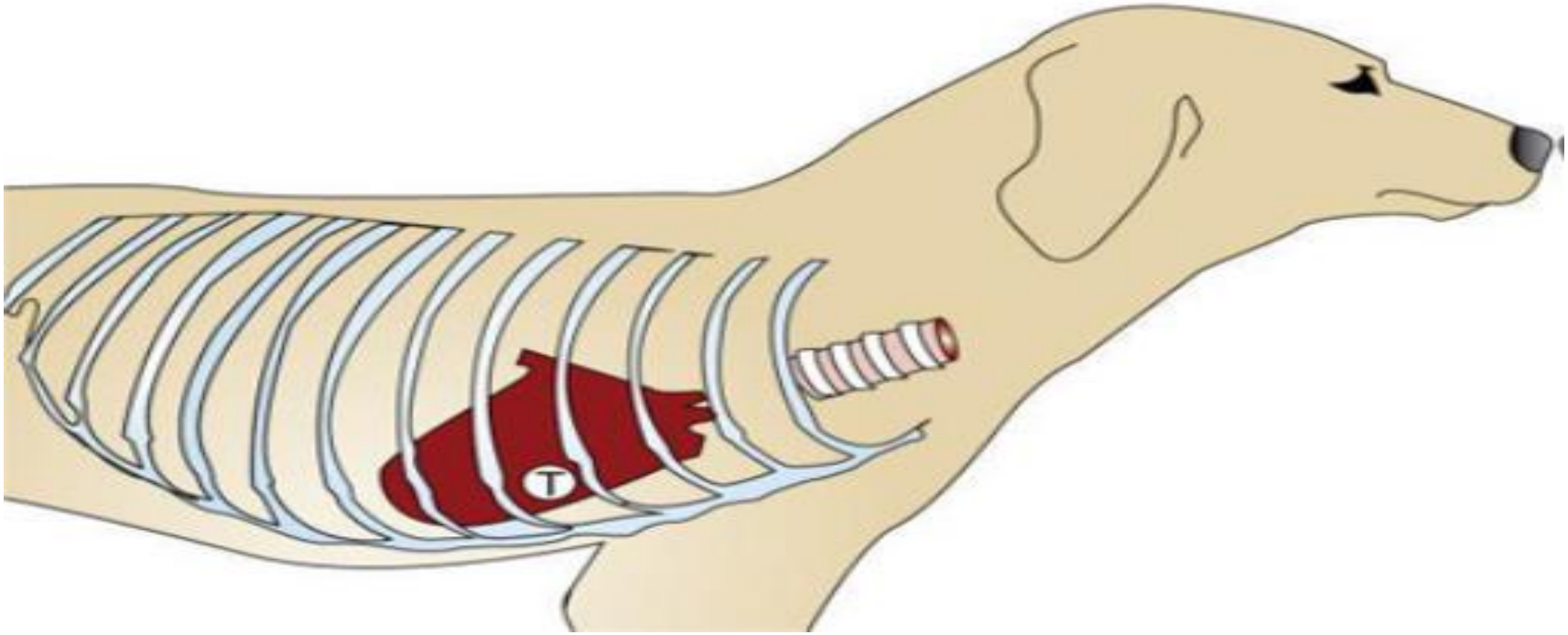
- Dyspnea: Tachypnea, respiratory distress, orthopnea.
- Lethargy/weakness.
- Exercise intolerance.
- Syncope.
- Collapse.
- Loss of appetite.
- Ascites or pleural effusion in the case of right-sided CHF.
- Sudden death.
- Cough – sometimes producing a pink foam.

Physical examination

- Weak systolic heart murmurs (not always present) secondary to dilation of the heart chambers and may be felt during the preclinical stage:
  - Left apical systolic murmur secondary to mitral regurgitation.
  - Right systolic murmur due to tricuspid regurgitation (less common than the first).



Areas of cardiac auscultation of the left hemithorax. Heart valves: Pulmonary (P), aortic (A), and mitral (M).

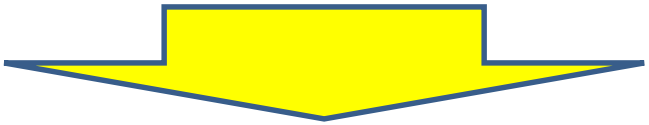


Area of cardiac auscultation of the right hemithorax. Tricuspid valve (T) of the heart.

Physical examination

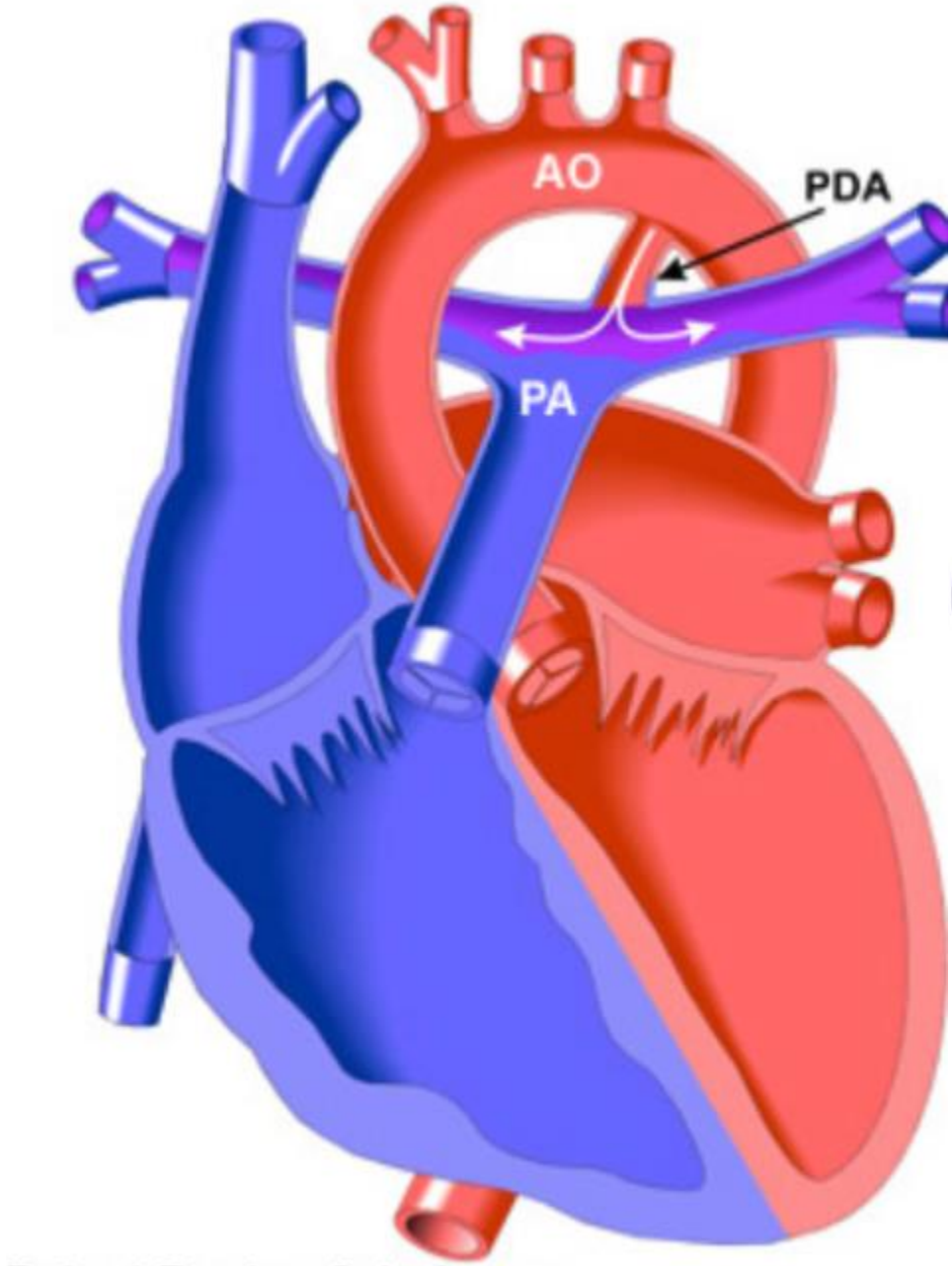
- Arrhythmias: Atrial fibrillation (supraventricular) or ventricular arrhythmias.
- Gallop sounds (S3 = B3) may be detected on auscultation.
- Weak femoral pulses (hypokinetic pulse = low systolic volume) or an alternating pulse.
- Weight loss/cachexia (muscle loss) – in the advanced stages of the disease.

- **Differential diagnosis**
- Left ventricular enlargement in large breed dogs may be due to several causes, such as:

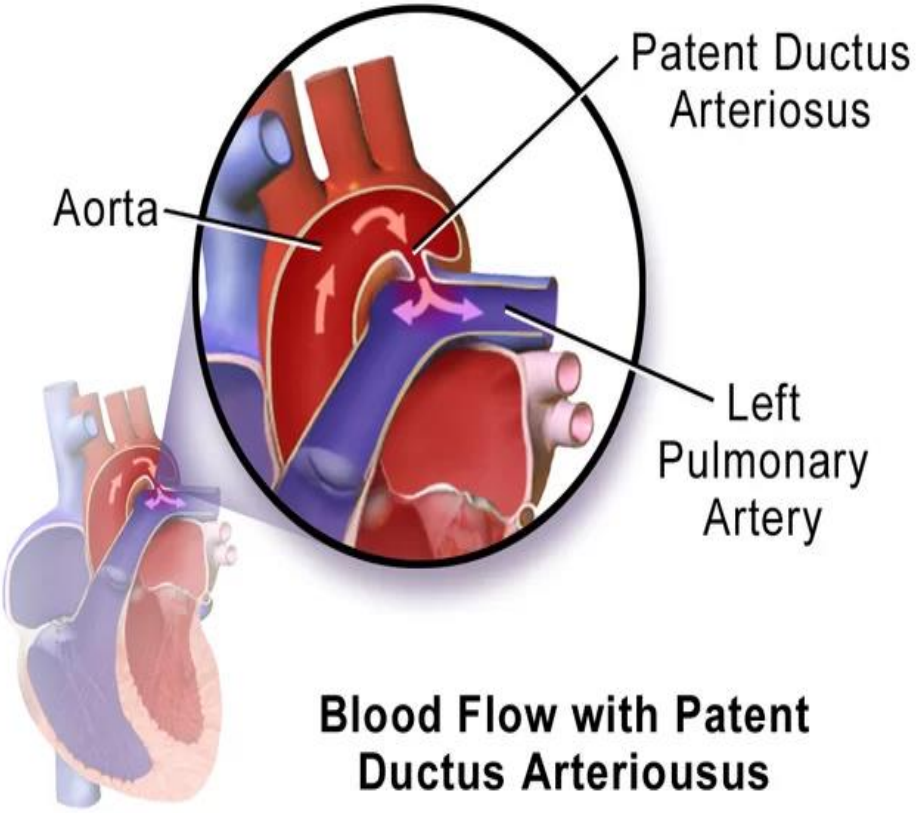


Tachycardiomyopathy, cardiomyopathy due to taurine deficiency or doxorubicin toxicity, and myocarditis.

- Primary mitral insufficiency is associated with left ventricular systolic dysfunction and may mimic the clinical presentation of CMD.
- Chronic cardiac changes leading to left-to-right shunting, particularly Patent Ductus Arteriosus (PDA) with left-to-right shunting, resemble those seen in CMD.



Patent Ductus Arteriosus



Blood Flow with Patent Ductus Arteriousus

○ Diagnosis

- In at-risk dogs, it would be advisable to screen for CMD using electrocardiogram (ECG), chest radiography, echocardiography, urinalysis, and blood chemistry.
- Often, additional tests such as 24-hour ambulatory ECG monitoring (Holter) are performed.

✓Laboratory analyses

The results of laboratory analyses of subjects suffering from CMD may vary:

- May lack specificity.

- Cardiac biomarkers:

- NT-proBNP [N-terminal Pro B-Type Natriuretic Peptide].

and

- Troponin I.

NT-proBNP + Troponin I

- May be used in the screening for CMD in predisposed breeds.
- CMD may be characterized by a combination of increased NT-proBNP and increased troponin I.
- However, care must be taken regarding significant differences between breeds in terms of normal NT-proBNP values.
- A marked increase in NT-proBNP is a sign of acquired or congenital structural heart disease.

- Pre-renal azotemia and increased ALAT (Alanine Aminotransferase) activity are associated with very low cardiac output.
- Marked increase in troponin I in cases caused by myocarditis.
- Reduced taurine concentrations in total blood in some American cockers.

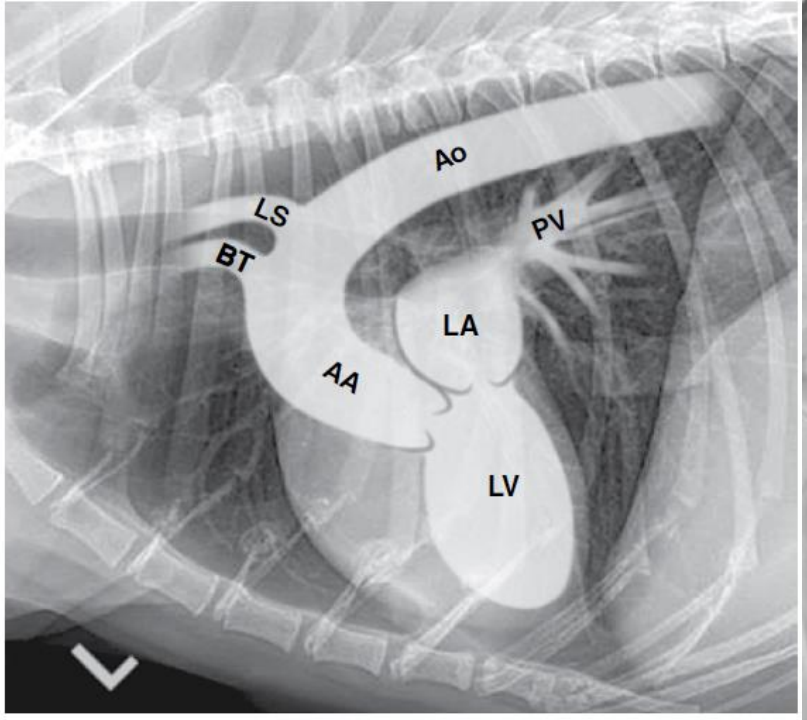
✓**Imaging**

Radiography

- Chest radiographs generally provide limited information in dogs in the occult phase.
- Although generalized cardiomegaly may be identified, chest radiographs are generally relatively insensitive in detecting mild to moderate dilation of the heart chambers.
- However, serial chest radiographs over time may be helpful in assessing disease progression.

➢In the clinical stage, X-rays are more useful for establishing the diagnosis:

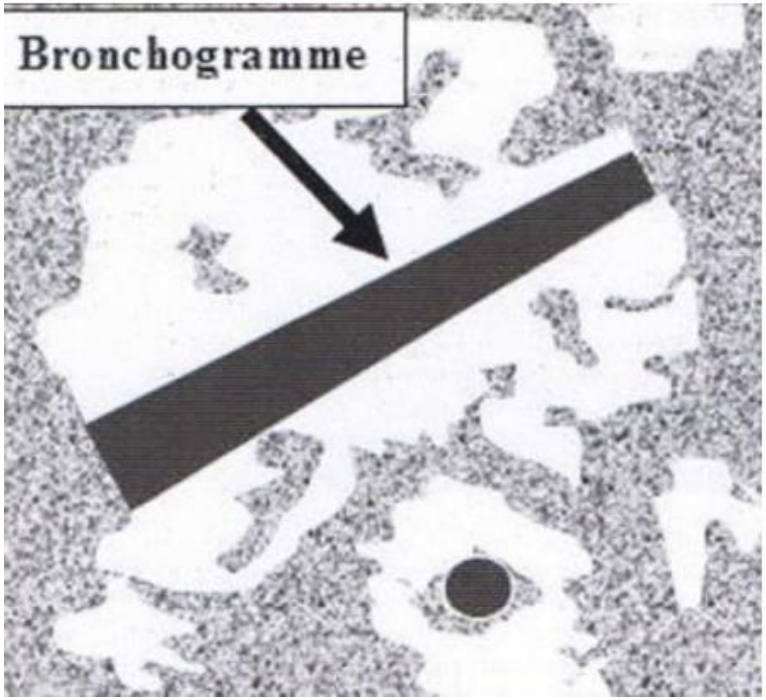
- Interstitial or alveolar pattern in case of left-sided CHF.
- Progressive cardiomegaly on the left side (or global).
- Pulmonary venous congestion in case of left-sided CHF.
- Pleural effusion/ascites in case of right-sided CHF.



AA = ascending aorta; Ao = descending aorta; BT = brachycephalic trunk; PV = pulmonary veins
LA = left atrium; LS = left subclavian artery; LV = left ventricle

Congestive heart failure. Left lateral chest X-ray of a dog suffering from congestive heart failure. A marked interstitial-alveolar pattern with prominent air bronchograms is compatible with severe pulmonary edema, with an air-filled stomach suggesting dyspnea. The pulmonary changes mask many changes in the cardiac silhouette, notably a probable right heart hypertrophy and the division of the caudal main bronchi suggesting left atrial hypertrophy.

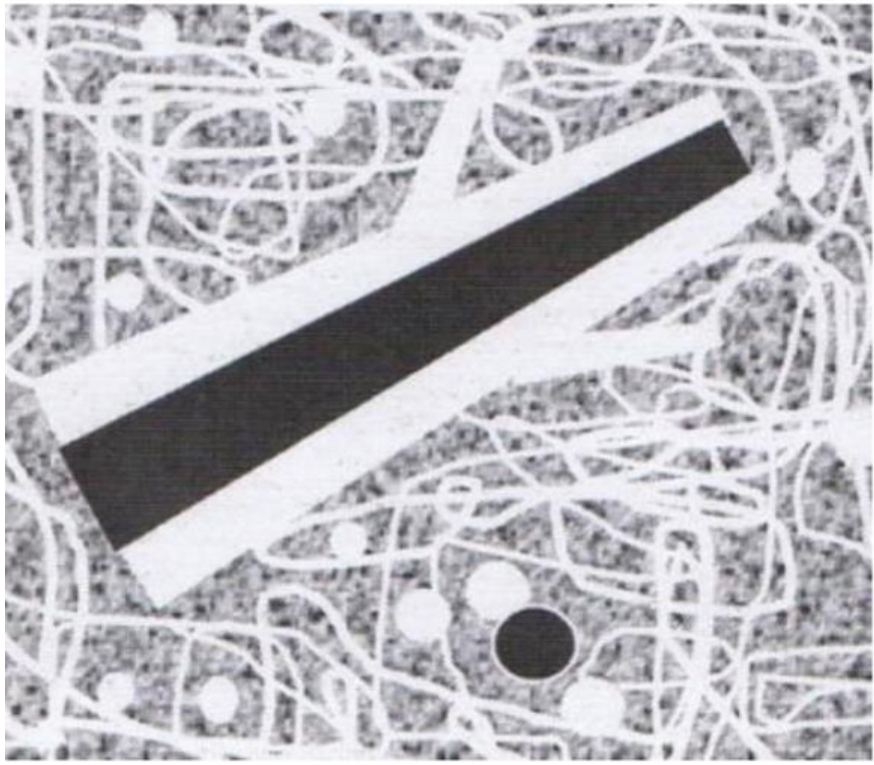
Schematic representation of the radiological aspect of alveolar opacification



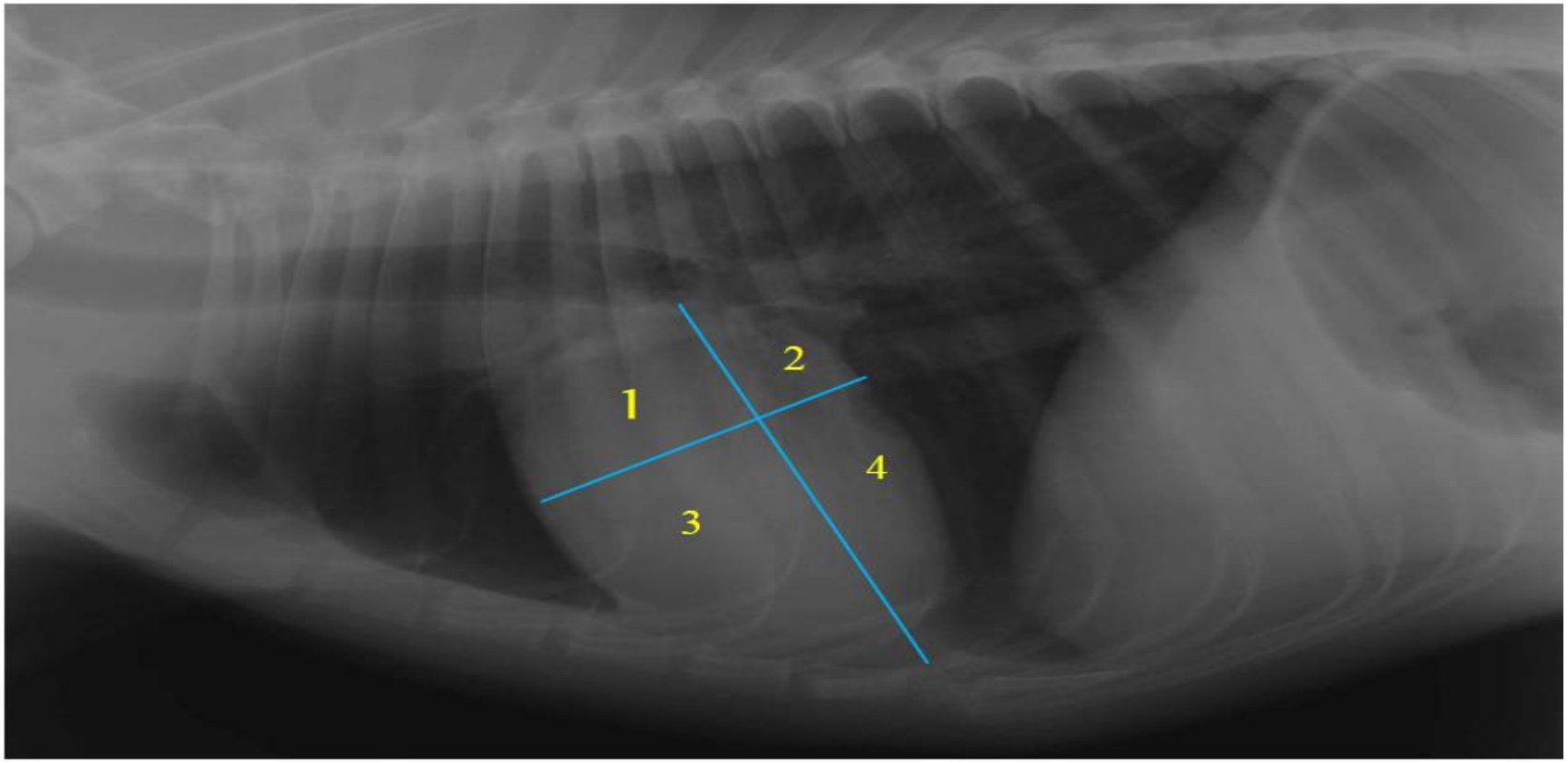
This is observed when the alveoli are filled with liquid, cellular debris, or a neoplastic infiltration, or during alveolar collapse. The liquid or debris displaces the alveolar air and, as a result, the alveoli no longer contribute to the overall contrast.

The bronchi containing air become visible and form tubular or branching radio-transparent structures that contrast with the more opaque lung tissue due to infiltration. This creates air bronchograms.

Schematic representation of the radiological aspect of interstitial opacification.



The pulmonary interstitium may be infiltrated by fibrous tissue, cells (inflammatory or tumor) or fluid. An unstructured opacification is then observed, with a generalized increase in pulmonary opacity related to the increase in opacity of the interstitial tissues, the contour of the vessels becomes less distinct but they remain identifiable, and the wall of the bronchi and bronchioles appears thicker due to the increase in the interstitial component.



1 : Atrium droit
2 : Atrium gauche
3 : Ventricule droit
4 : Ventricule gauche

Schematic representation of the different parts of the dog's heart on a lateral thoracic radiograph.

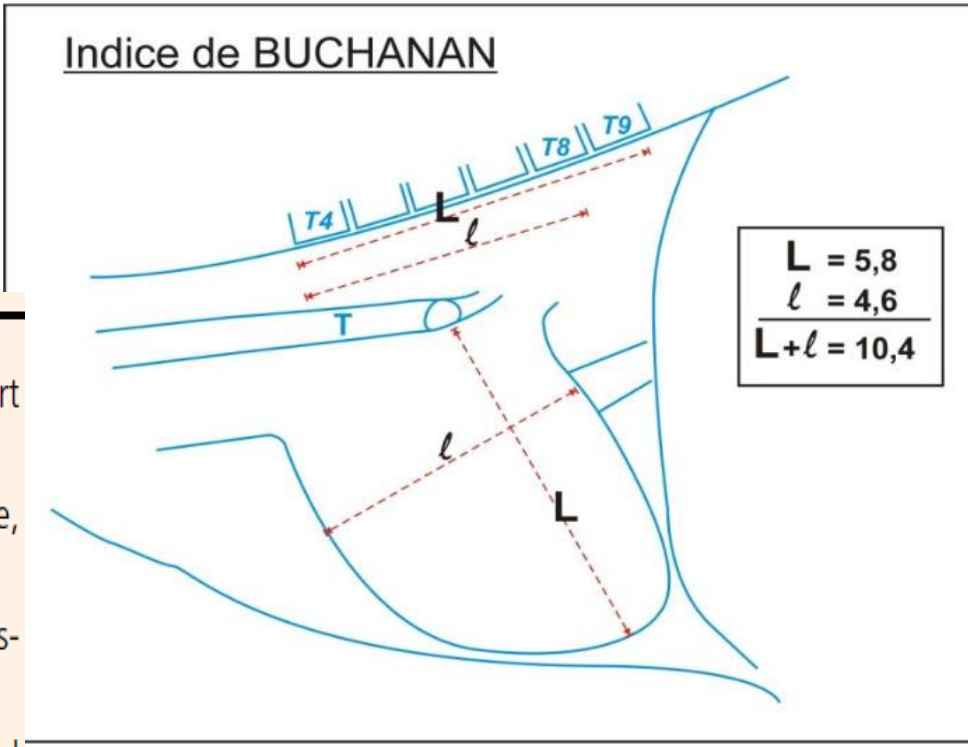
If a line is drawn from the tracheobronchial bifurcation to the apex, two parts are delineated: two-thirds for the right heart, cranial, and one-third, caudal for the left heart.

Heart size

On the profile view:

1. In a lateral view, measure the length (long axis) and width (short axis) of the cardiac shadow.
2. Transpose both of these measurements onto the thoracic spine, each beginning at the cranial edge of T4.
3. Count the number of thoracic vertebrae spanned by each measurement to the nearest 1/10th of a vertebra.
4. Add the total number of vertebrae along the cardiac length and width to get the vertebral heart score.

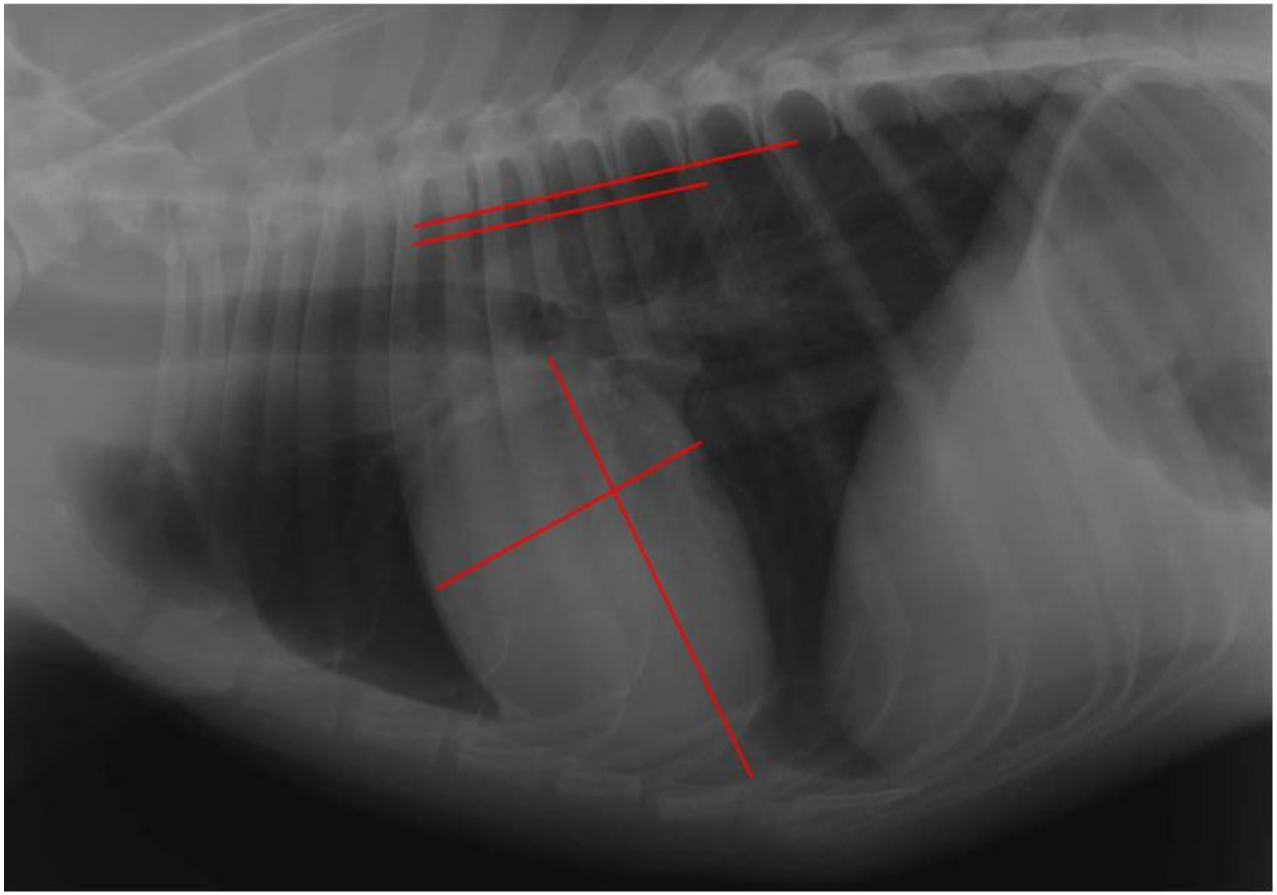
	Dog	Cat
Normal range	8.5 – 10.6	6.8 – 8.1
Mild enlargement	11 – 11.9	8.2 – 8.5
Moderate enlargement	12 – 12.9	8.6 – 8.9
Severe enlargement	13 – 14+	9 – 10+



Vertebral heart score (VHS)

=

Cardiac index or Buchanan index

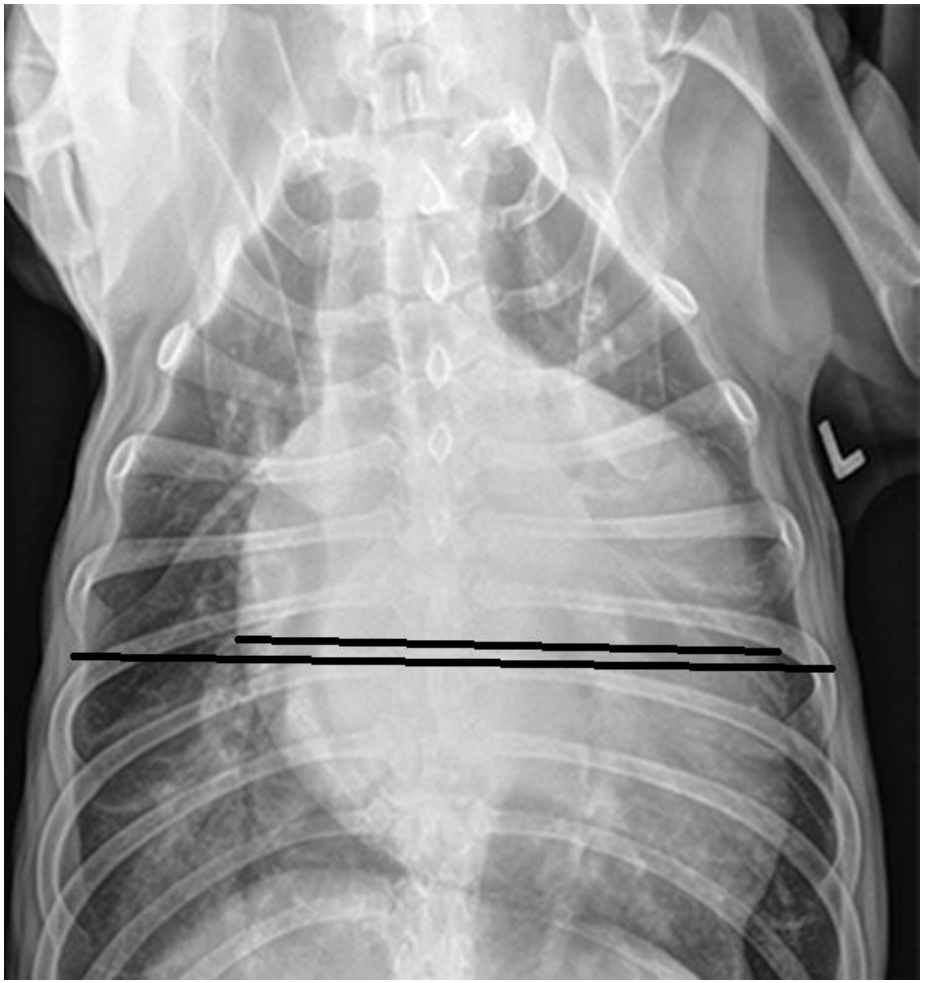


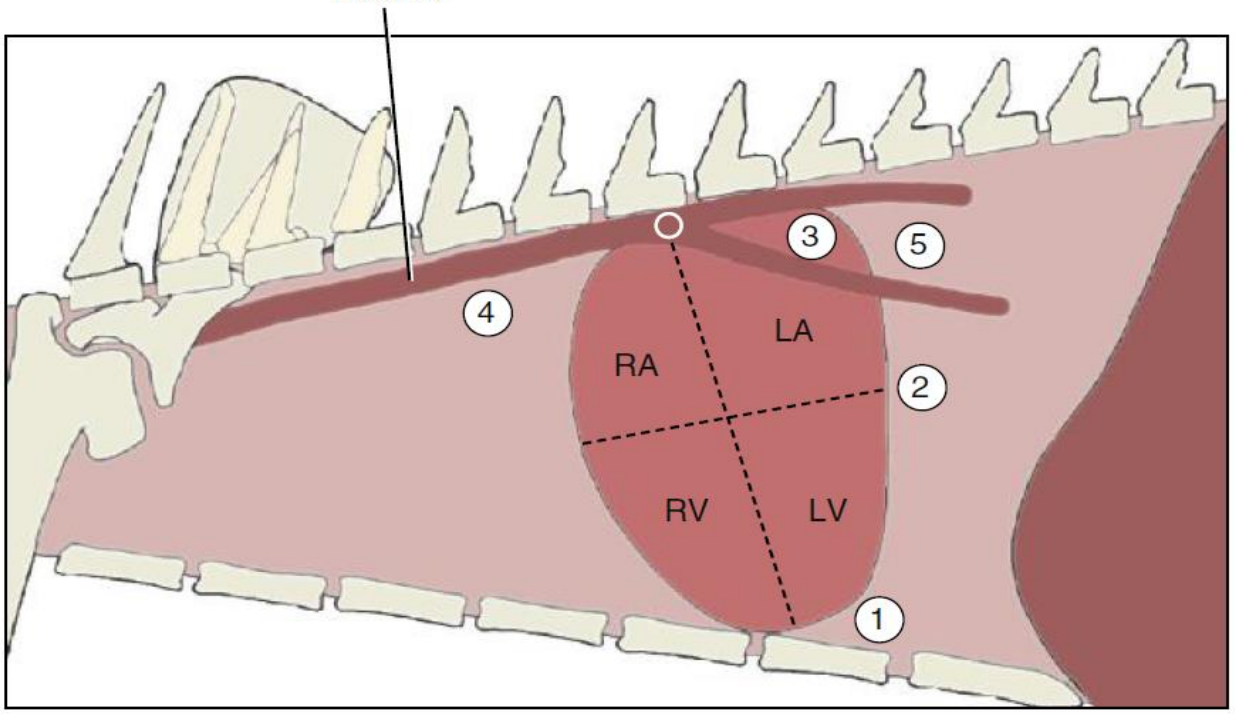
Application of the Buchanan index calculation on a lateral chest X-ray of a dog

On the frontal view (Dorso-ventral incidence):

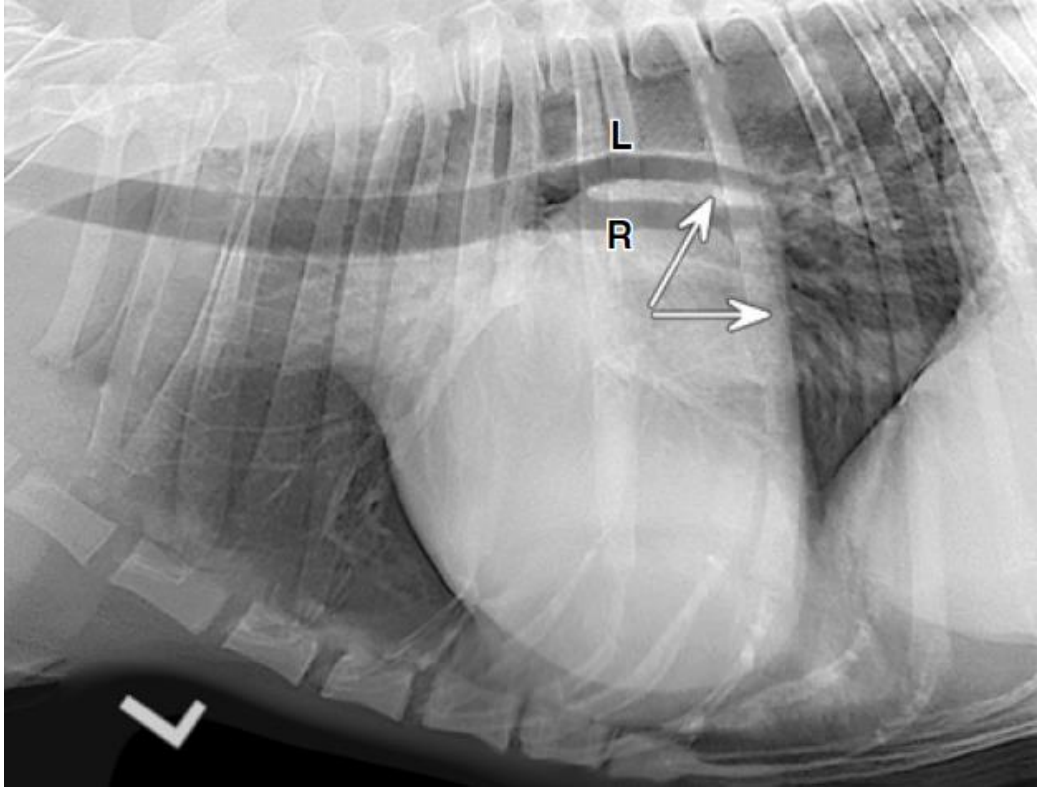
The transverse diameter of the heart at its widest point should not exceed 70% of the transverse diameter of the thoracic cage measured at the same location.

Dog with active congestive heart failure. There is evidence of severe cardiomegaly characterized by left ventricular and left atrial enlargement

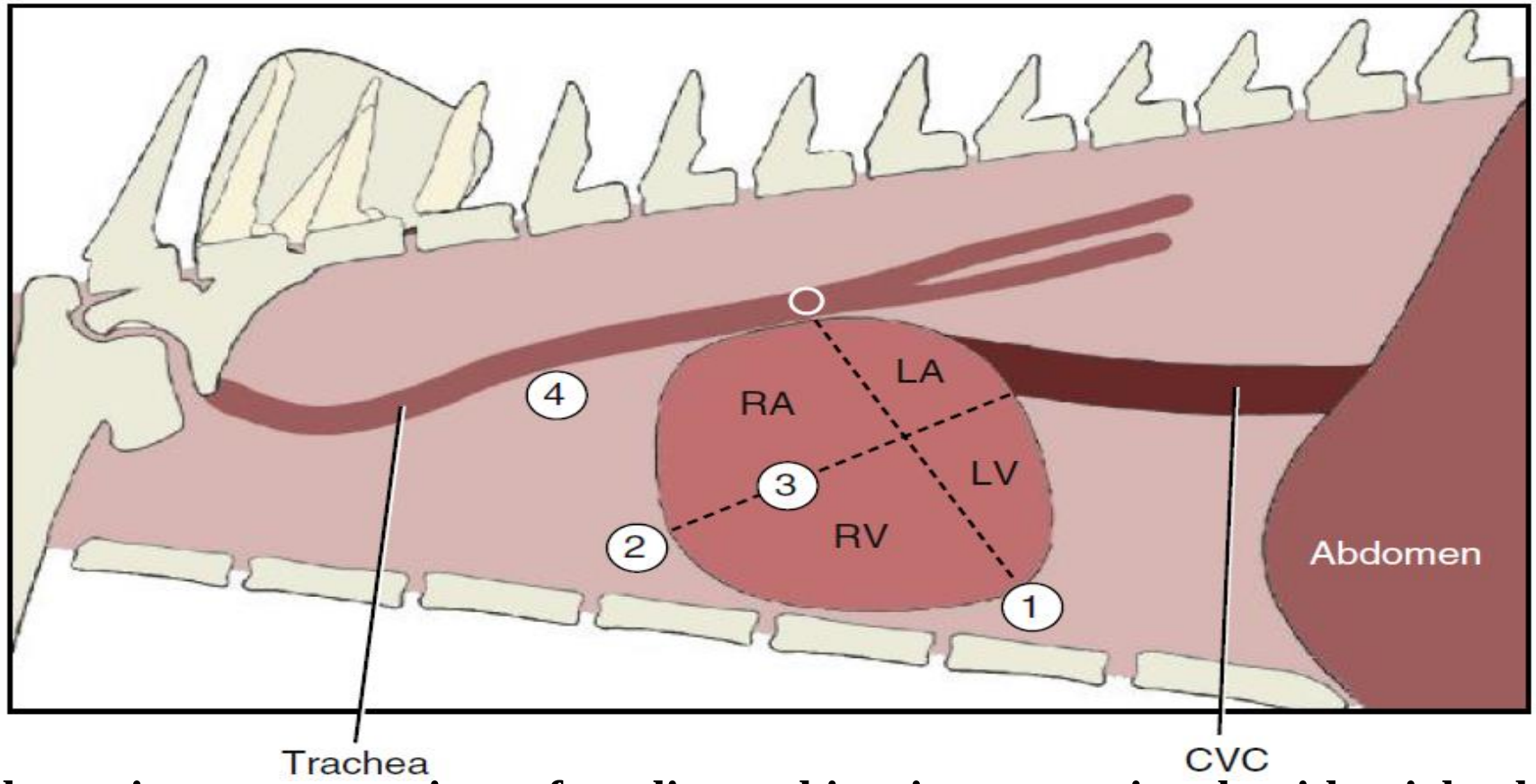




Schematic representation of the radiographic signs associated with left heart hypertrophy in lateral projection. (1) Rounding and widening of the heart apex configuration. (2) Straightening and increase of the vertical axis. (3) Left atrial hypertrophy with characteristic conformation of the caudodorsal margin = Right angle. (4) Dorsal elevation of the intrathoracic portion of the trachea, carina, and main bronchi. The angle between the thoracic column axis and the trachea is decreased to the point of becoming parallel. (5) Separation of the normally superimposed caudal main bronchi. The left side is more dorsal in position than the right side.; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

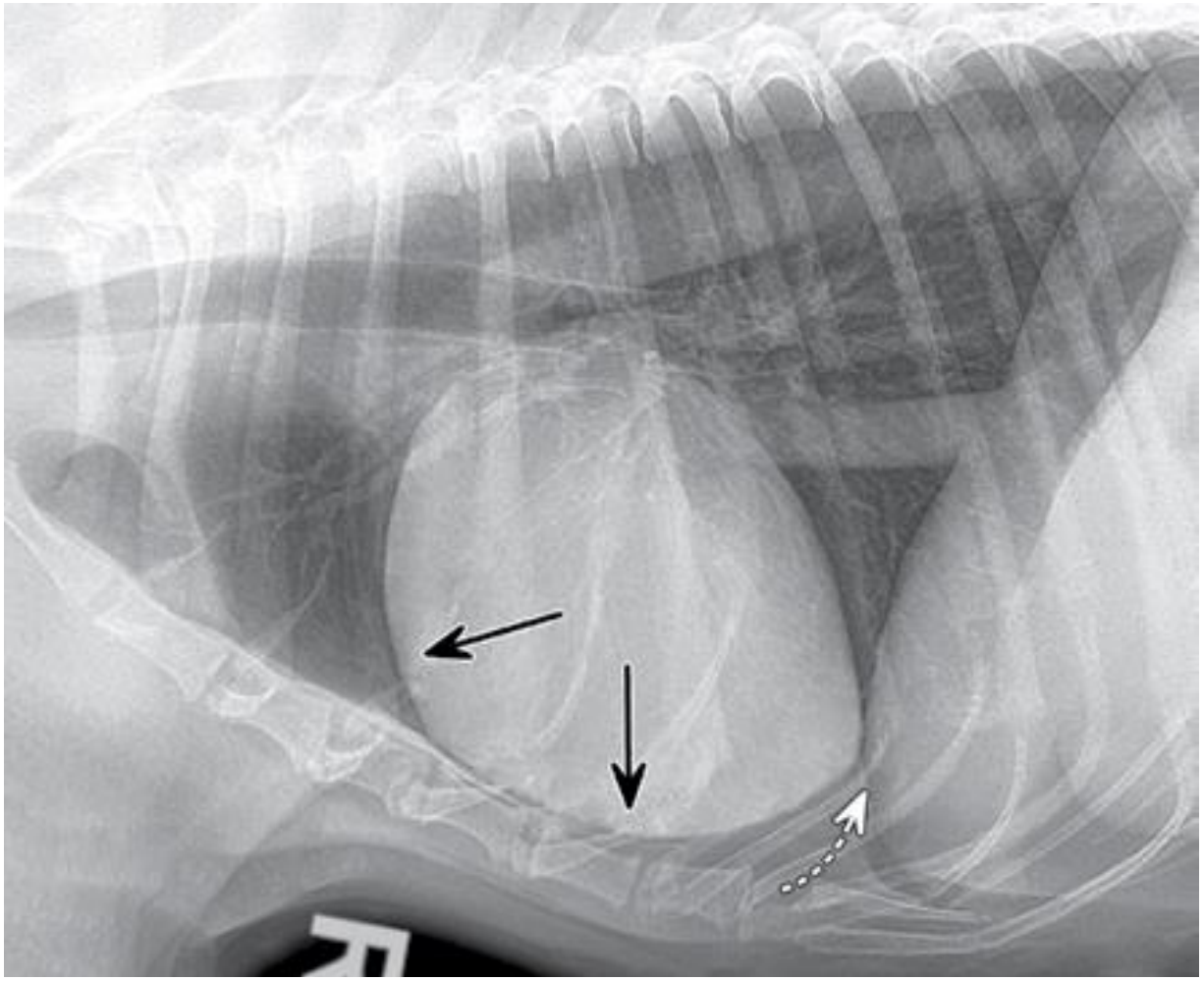


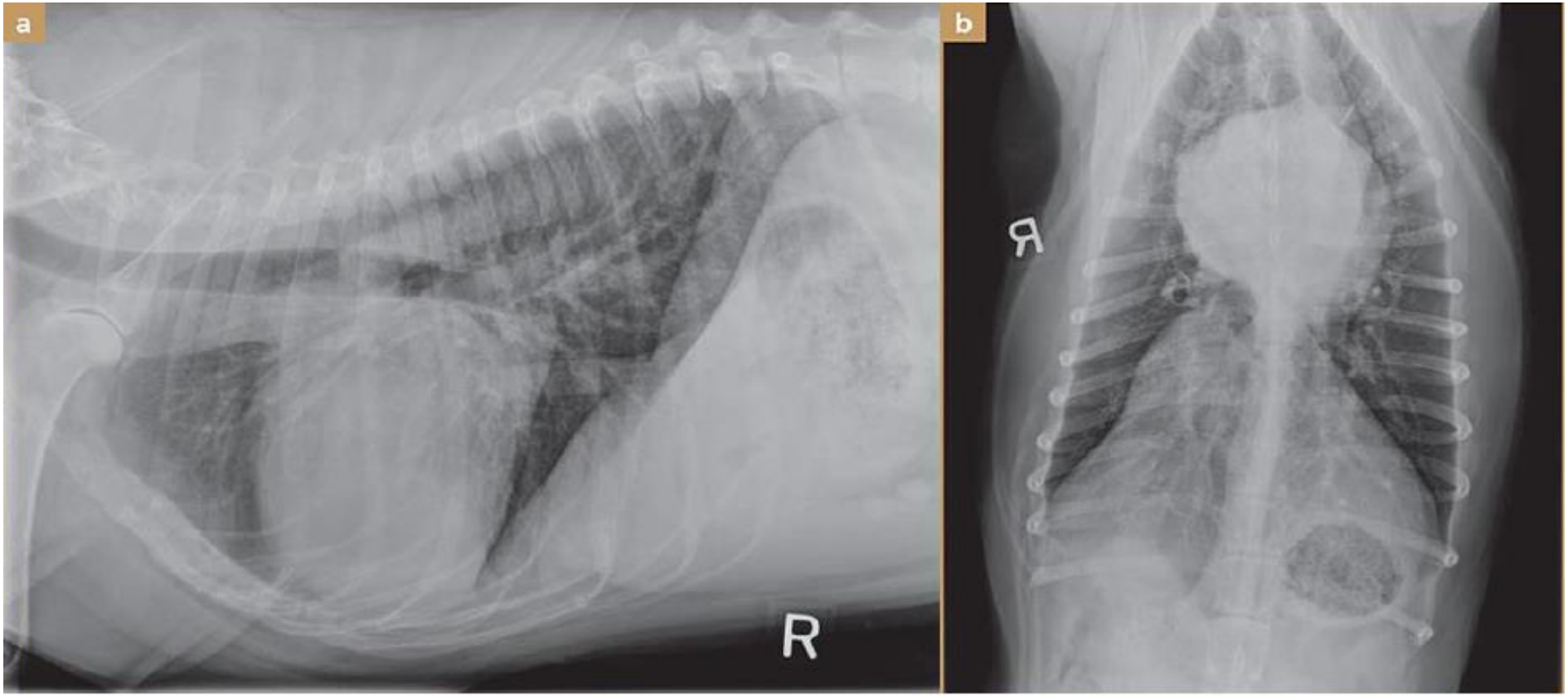
Left atrium markedly dilated. Lateral view of a dog's thorax showing severe left atrial dilatation. The trachea is displaced dorsally and there is a bifurcation of the primary bronchi with the left bronchus (L) positioned dorsally relative to the right bronchus (R).



Schematic representation of radiographic signs associated with right heart hypertrophy in lateral projection. (1) Dorsal elevation of the apex of the sternum. (2) Increased sternal contact with the cranial cardiac margin. (3) Disproportionate widening of the cranial part of the cardiac silhouette when empirically divided into chambers: right and left. (4) Elevation of the trachea when it reaches (dilation) dorsally the right atrium. CVC, caudal vena cava; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

Right ventricular enlargement. Lateral view



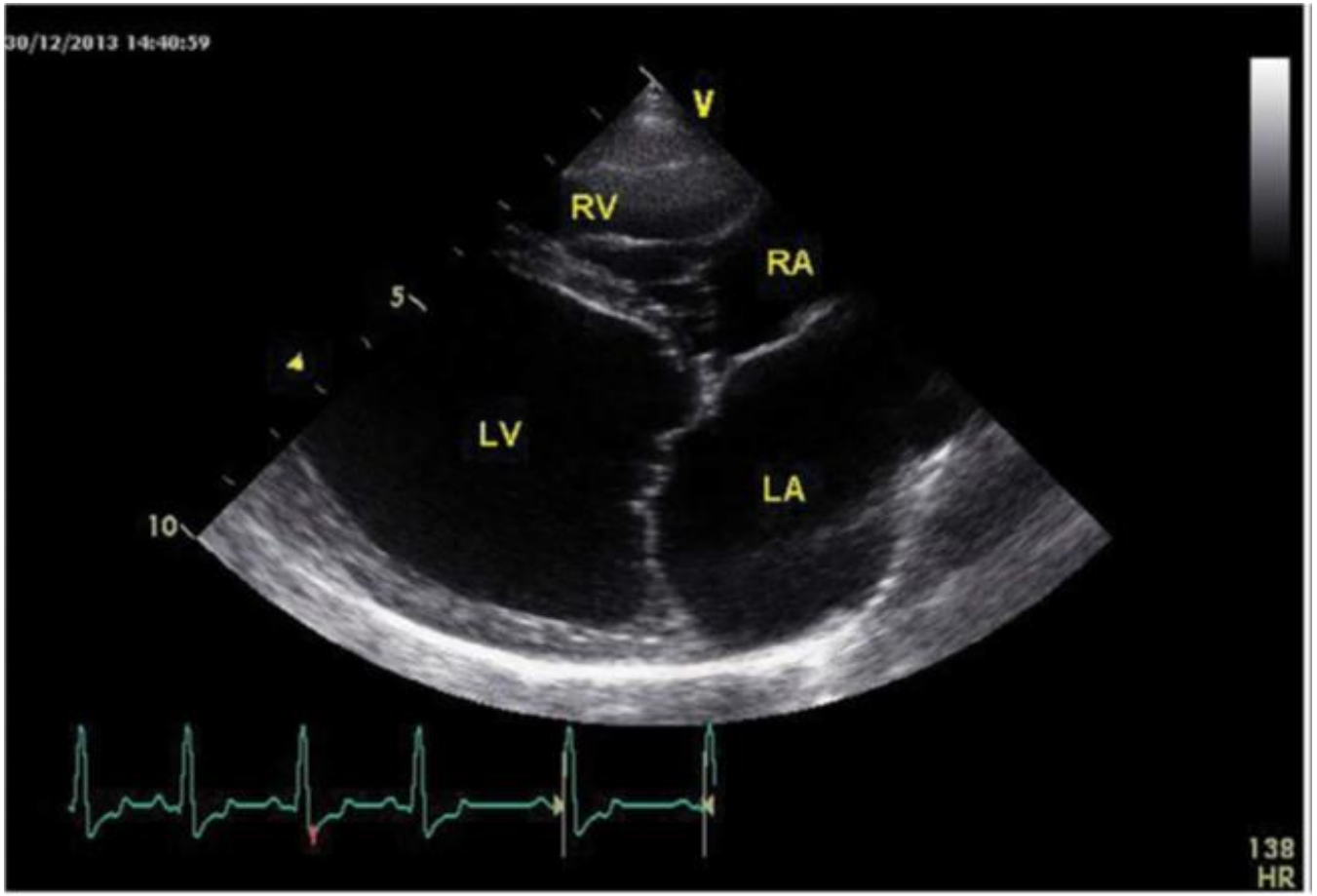


Thoracic radiographs in an 8-year-old female spayed Doberman. Cardiomegaly associated with mild distension of the pulmonary veins. An interstitial to alveolar pattern can be seen in the ventral portion of right cranial, right middle and left cranial lung lobes.

Echocardiography

- Echocardiography is essential for the assessment of cardiac enlargement (cardiomegaly) and cardiac function particularly at the occult stage of the disease.
- Echocardiography (M-mode [motion mode] and 2D [two-dimensional] methods) can reveal:

- Left ventricular dilation with reduced measurements of systolic function (shortening fraction, ejection fraction).

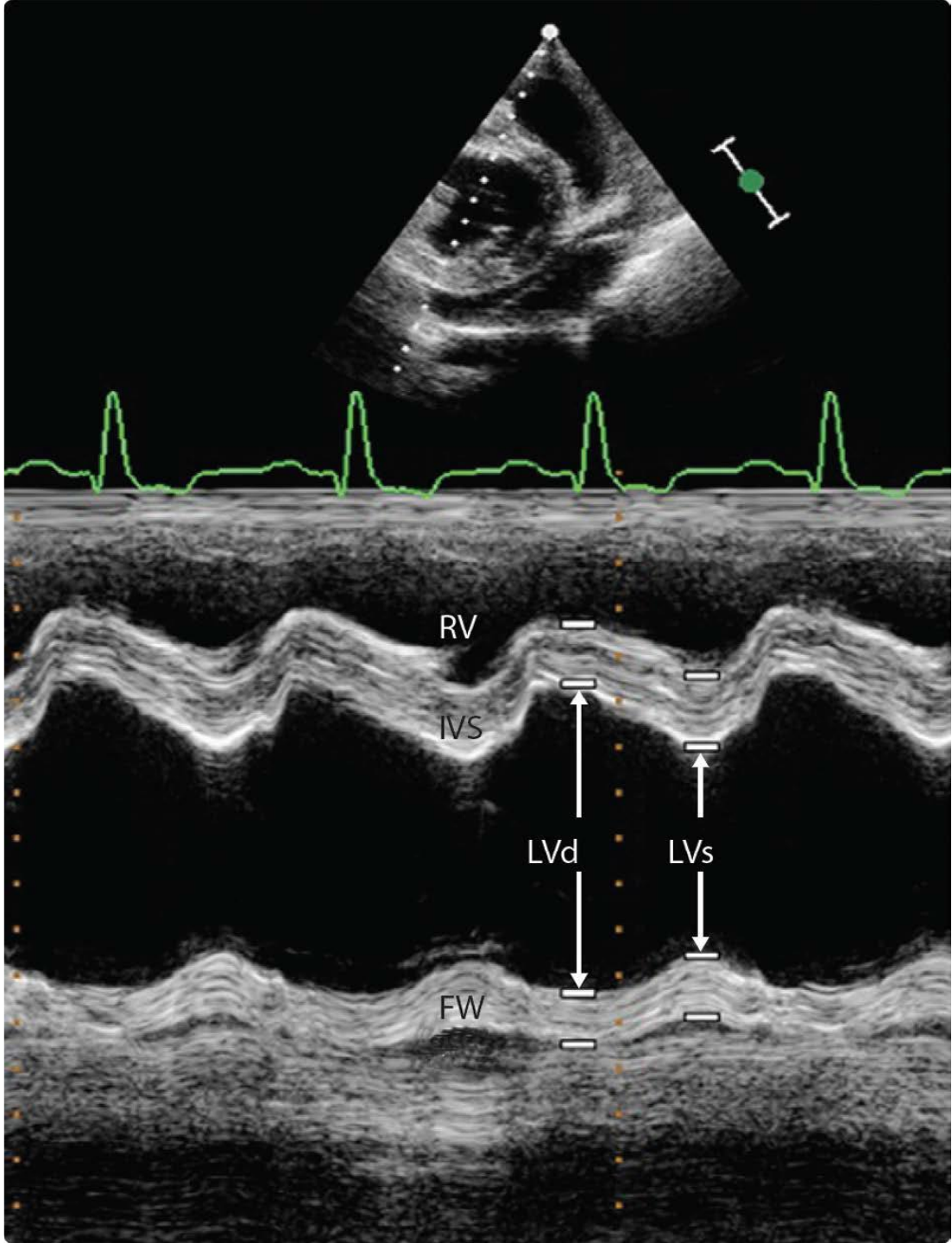


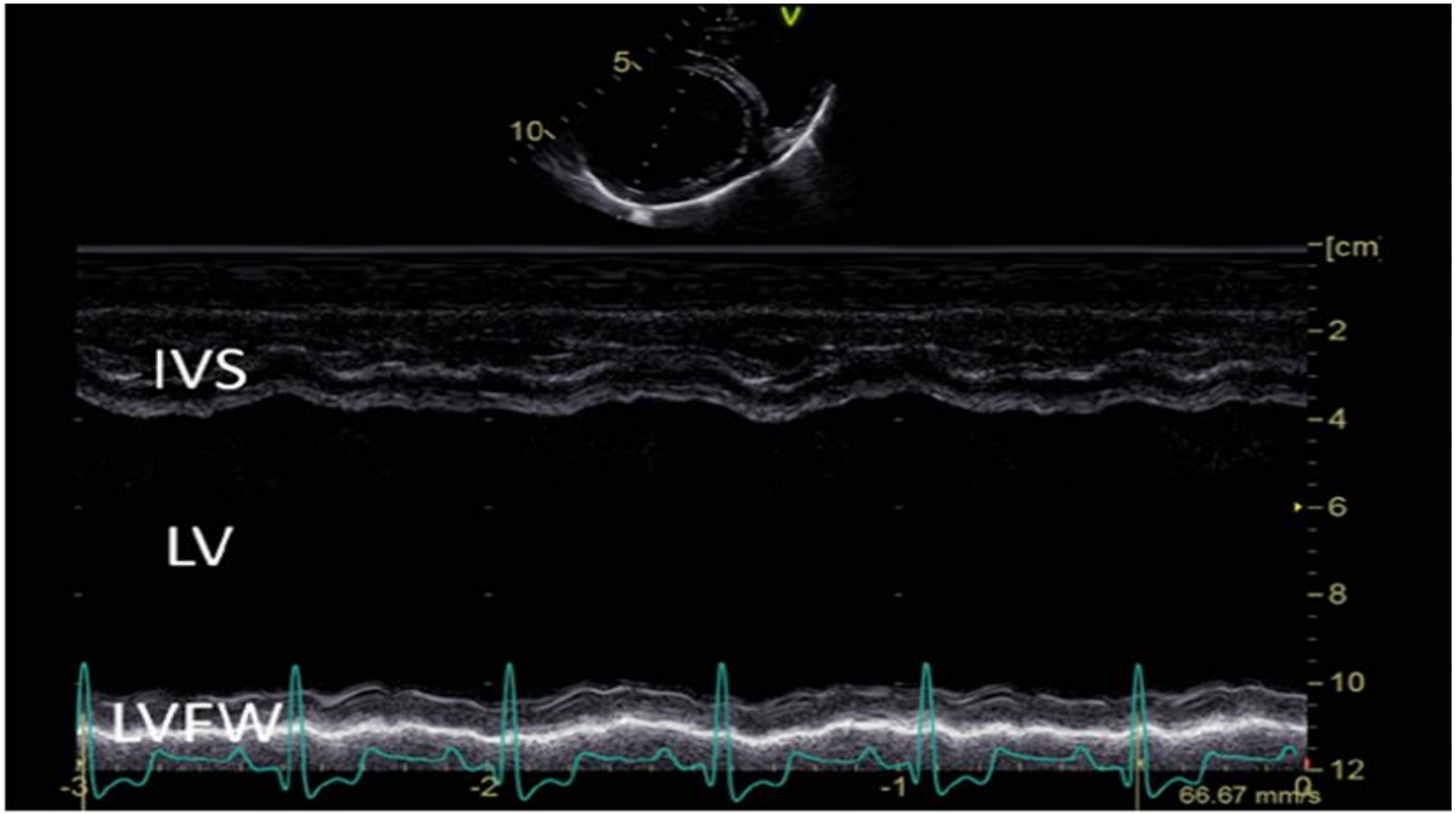
CMD in a dog, observed by echocardiography. Note the enlargement of the left atrium and ventricle, the thin wall of the left ventricle, and the stretching of the mitral valve in this image.

- The classic sign of CMD on an echocardiogram is systolic dysfunction:

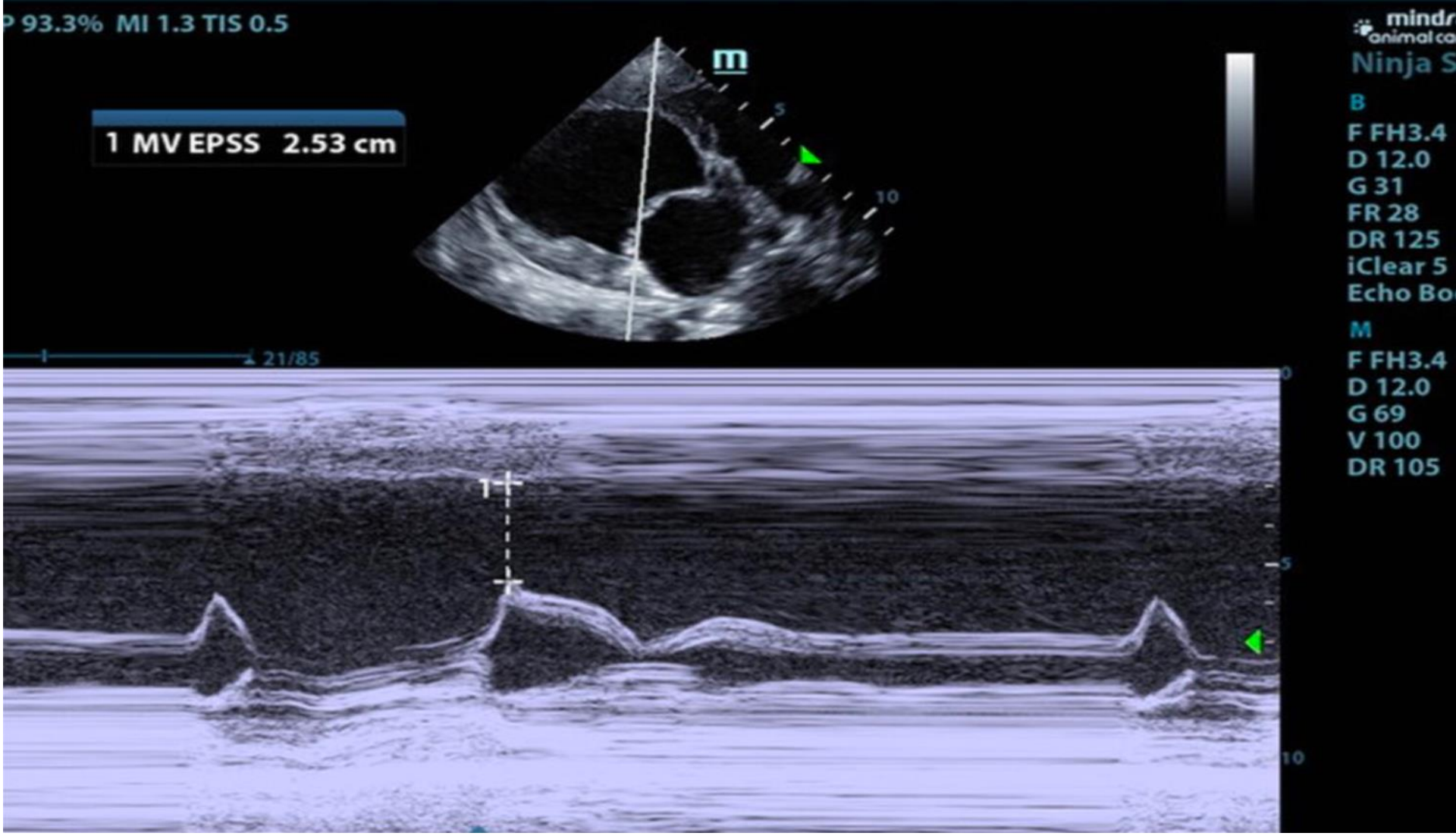
Characterized by a reduction in shortening fraction (normal reference range CN = 25% - 45%) and ejection fraction (normal reference range CN = 55% - 70%) as well as an increase in the value of the 'E point-septal separation' (EPSS = the distance between the anterior leaflet of the mitral valve and the septum) and the left ventricle's telesystolic diameter.

View in M mode of the left ventricle at the level of the chordae tendineae.
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.





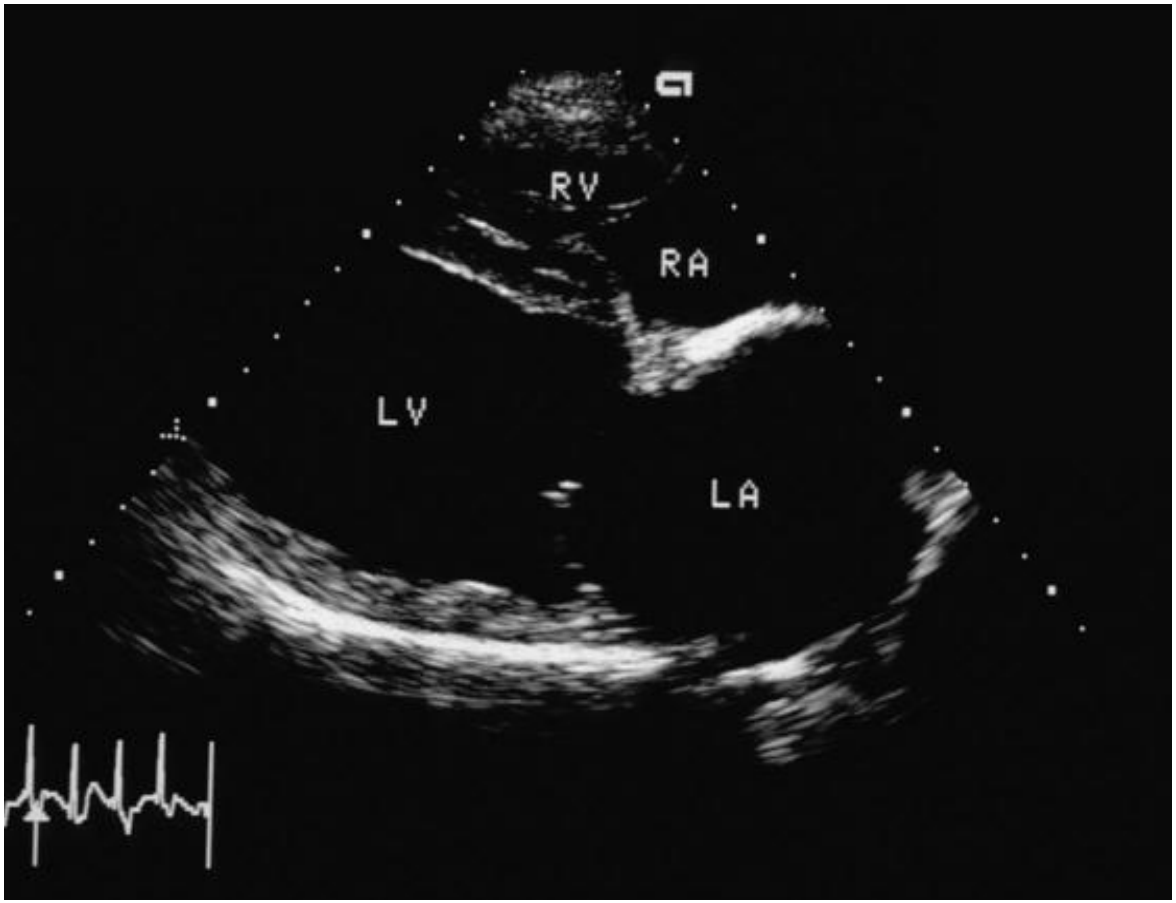
Dilated cardiomyopathy. M-mode echocardiogram of a dog with dilated cardiomyopathy showing a dilated left ventricle and reduced systolic function. IVS - interventricular septum; LV - left ventricle, LVFW - left ventricular free wall.



Increase in EPSS. Dilated cardiomyopathy (CMD) in a 10-year-old mixed breed dog. Normal values in dogs are generally less than 6–7 mm, while an increased distance (>7–13+ mm).

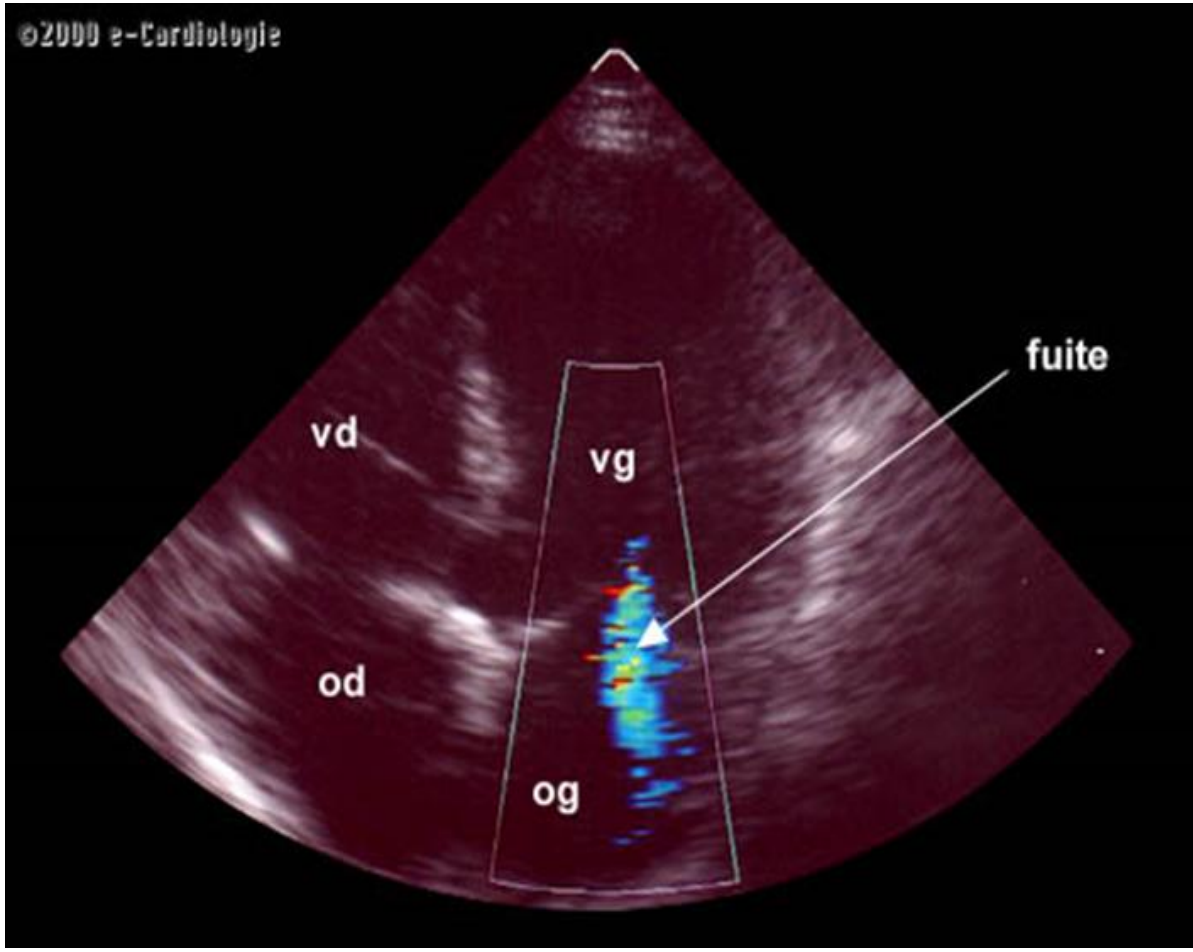
- An increase in the left ventricular telediastolic diameter as well as dilation of the left atrium are observed as the disease progresses (secondary left atrial dilation).

Two-dimensional echocardiogram of the left ventricle (LV) and left atrium (LA) of a Great Dane with dilated cardiomyopathy (DCM). Note the dilated ventricular and atrial chambers. RA, Right atrium; RV, right ventricle.



- Color Doppler can show mitral regurgitation in the left atrium, often with a single jet following a central path.

Visualization of a human heart using ultrasound. Note the existence of a blue jet going from the left ventricle to the left atrium during recording with a color doppler. lv: left ventricle, rv: right ventricle, la: left atrium, ra: right atrium.



Electrocardiography and Holter monitoring

The ECG is the test of choice for detecting arrhythmias and can provide evidence of cardiac dilation (or hypertrophy); however, a normal ECG does not rule out the presence of cardiomyopathy.

Dog wearing a 24-hour Holter ECG monitor.

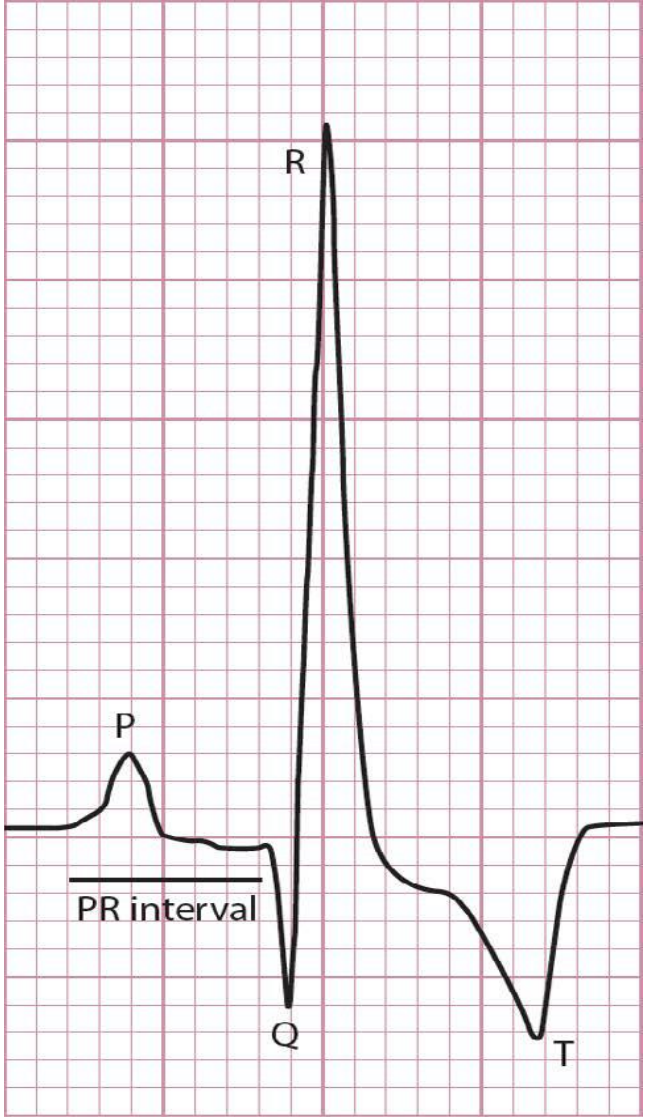
❑ Asymptomatic occult phase

- Arrhythmias are often the first sign of disease and screening is recommended in breeds at high risk of CMD.
- Routine ECG detects frequent arrhythmias but may have limited sensitivity in dogs with infrequent or intermittent arrhythmias.
- The detection of the following ECG signs is associated with a high index of suspicion for occult cardiomyopathy:

- One or more ventricular premature contractions (VPCs) in a Doberman Pinscher or a Boxer.
- In Boxers, VPCs with a left bundle branch block morphology (vertical QRS complex in lead II) are very suggestive of Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC).

Electrocardiogram (ECG) tracing in lead II of a 7-year-old neutered male Boxer with arrhythmogenic right ventricular cardiomyopathy (ARVC). Ventricular premature contractions with left bundle branch block morphology are a common observation in dogs with this disease. 25 mm/sec; 0.5 cm/mV.

- Criteria for left ventricular dilation (QRS duration > 0.06 seconds, R wave amplitude > 3.0 mV) or left atrium (P wave duration > 0.04 seconds).



P-QRS-T illustrating the sequence of activation of the heart. Note that on this ECG there is no clearly defined S wave.

- In Irish Wolfhounds, atrial fibrillation is often an early sign of disease, unlike other breeds, where atrial fibrillation is associated with advanced stages of disease.

ECG from an Irish Setter with atrial fibrillation (AF) with a heart rate of 130/min. This dog had a very dilated left atrium secondary to mitral valve disease (MVD).

- Holter monitoring detects arrhythmias with greater sensitivity and is recommended in high-risk dogs (dogs with family history) or in those who have experienced syncope.
- More than 100 ventricular premature contractions over a 24-hour period strongly suggest CMD or occult ARVC.
- 50 to 100 VPCs over a 24-hour period are suspicious for disease (CMD or ARVC) and should be followed by another Holter examination in 2 to 6 months.

Manifest clinical phase

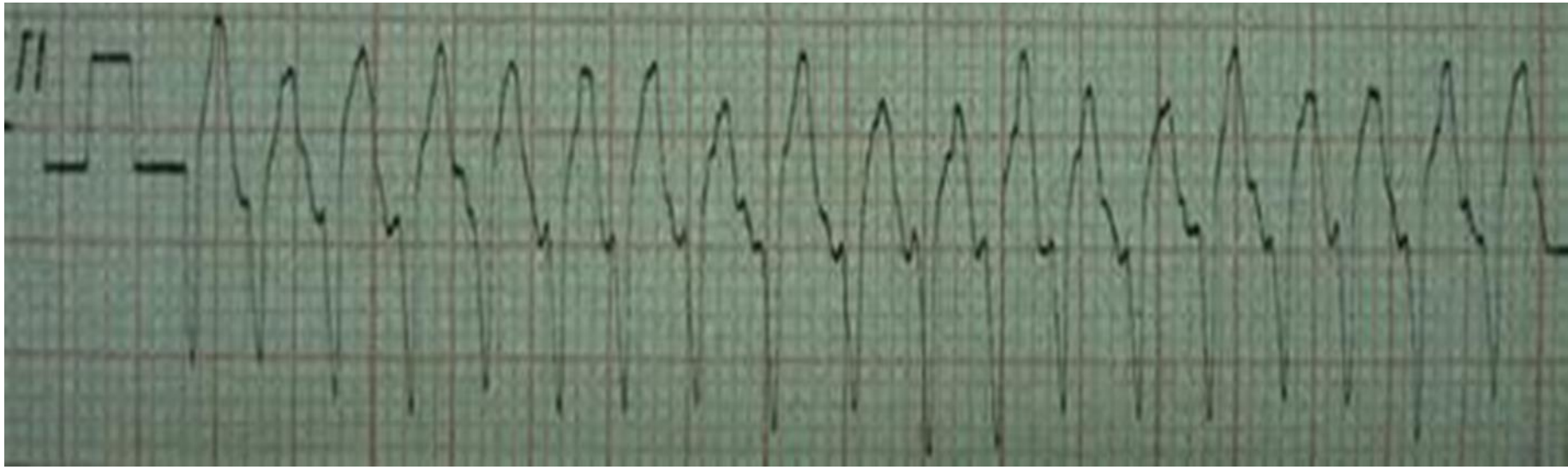
During the manifest clinical phase, the following symptoms may be detected:

- Occasional to frequent ventricular or supraventricular premature beats.
- Ventricular tachycardia.
- Criteria for left ventricular dilation (QRS duration > 0.06 seconds, R wave amplitude > 3.0 mV) or left atrium (P wave duration > 0.04 seconds).
- Left bundle branch block.
- Atrial fibrillation.

ECG showing sinus rhythm with one ventricular premature contraction (VPC) (arrow) = Ventricular extrasystoles.

ECG from a Bull Mastiff with atrial fibrillation (AF) with a heart rate of 150/min. This dog had no underlying heart disease.

ECG of dogs showing supraventricular extrasystoles (arrows). Note that recognition is made by the premature synchronization of QRS and T waves, which have the same morphology as those of normal sinus complexes (25 mm/s and 10 mm/mV).



Ventricular tachycardia in a dog

Blood pressure

- May be normal.
- Some patients present a flow insufficiency and are therefore systemically hypotensive.

Measurement of systolic blood pressure in a cat using the Doppler system.

○ Prognosis

• The prognosis varies according to the breed and the severity of the disease at the time of presentation.

• Cocker spaniels appear to have a long progression of the disease.

• Dobermans have a high incidence of sudden death with a short clinical course.

- Giant breed dogs affected by an arrhythmia may remain in a subclinical phase for years:

Can survive for several years with careful management.

- The outlook is poor once CHF develops.

- **Treatment**

Preclinical

- It has been shown that pimobendan (CARDISURE® 5 mg Tablets for dogs) delays the onset of CHF.

Management of CHF

- Diuretics (furosemide, spironolactone).
- Pimobendan.
- Angiotensin-converting enzyme (ACE) inhibitors (Ramipril, Enalapril); to be avoided in cases of systemic hypotension.

Management of arrhythmias

Anti-arrhythmics – depends on ECG diagnosis, clinical signs, and echocardiographic appearance:

- Atrial fibrillation: may require diltiazem, digoxin.
- Ventricular arrhythmias: may require lidocaine, mexiletine, sotalol, amiodarone.

Other

- Dietary change for subjects following a BEG diet.
- Taurine supplementation for subjects with confirmed deficiency.

Monitoring

- Echocardiography.
- Holter ECG monitoring.

Myxomatous Mitral Valve Disease (MMVD)

Definition of MMVD

- Degenerative Mitral Valve Disease (MVDM; Myxomatous Mitral Valve Disease [MMVD]) is also referred to as Degenerative Valve Disease (MVD) or chronic valve disease.
- MMVD is the most common heart disease in dogs = responsible for more than 75% of all acquired heart diseases in this species.

- The term 'myxomatous = mucoid material' describes a histological characteristic of the degenerative process that occurs in the valvular apparatus of affected dogs.
- Myxomatous degeneration most often affects the mitral valve apparatus; however, the other 3 cardiac valves may also be involved.

Alternative names to define mitral valve disease

- Maladie de la valve mitrale (Mitral valve disease)
- Maladie Valvulaire Degenerative Mitrale (Degenerative mitral valve disease)
- Maladie myxomateuse de la valve mitrale (Myxomatous mitral valve disease)
- Fibrose chronique de la valve mitrale (Chronic mitral valve fibrosis)
- Maladie valvulaire dégénérative chronique* (Chronic degenerative valvular disease)
- Maladie valvulaire chronique* (Chronic valvular disease)
- Endocardiose*(Endocardiosis)

*Names including both the mitral and tricuspid valves.

○ Predisposition

- Dogs with MMVD show no signs of valvular abnormality at birth (normal mitral valve apparatus), but develop the disease at later stages of their lives.
- MVDM mainly affects small and medium-sized dogs, but some large breed dogs can also be affected.
- The reason MVDM occurs in some dogs is unknown.

- The disease is more common in middle-aged or older dogs:
  - Up to 85% of dogs over 13 years old are affected by MVDM.
  - The disease can also be diagnosed in younger dogs (1 to 2 years) belonging to breeds that are susceptible to predisposition.
- Males seem to be predisposed to MVDM and are more likely to develop it at a younger age than females.

Breeds predisposed to MMVD

- Cavalier King Charles Spaniel*
- Teckel (Dachshund) *
- Caniche nain (Miniature poodle)
- Chihuahua
- Lévrier nain (Whippet)
- Pinscher nain (Miniature Pinscher)
- Épagneul nain continental variété papillon (Papillon)

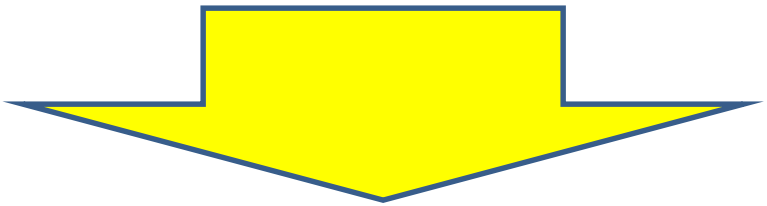
* A genetic link has been identified in Cavalier King Charles Spaniels and Dachshunds.

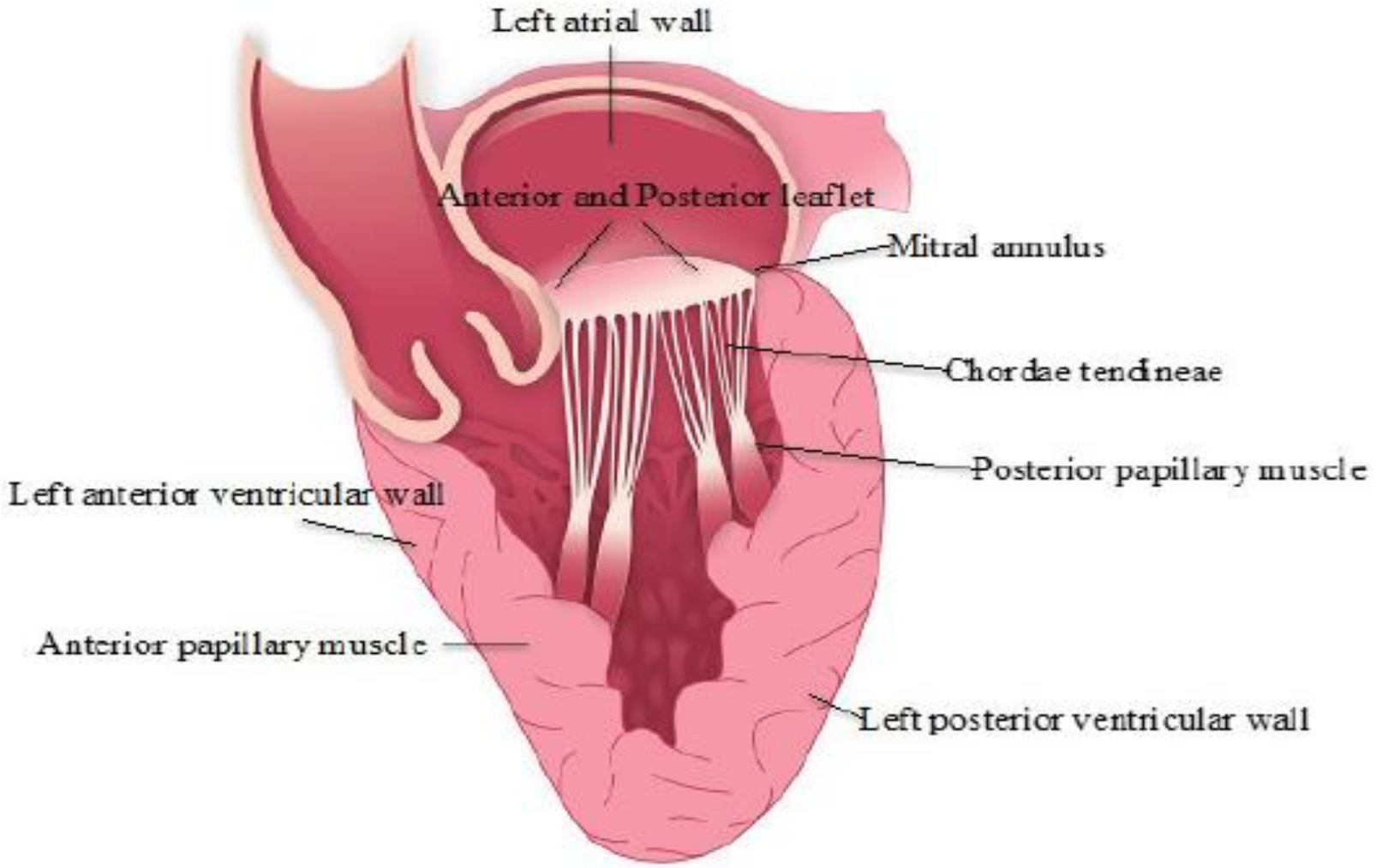
Continental Toy Spaniel variety Papillon

Cavalier King Charles Spaniel

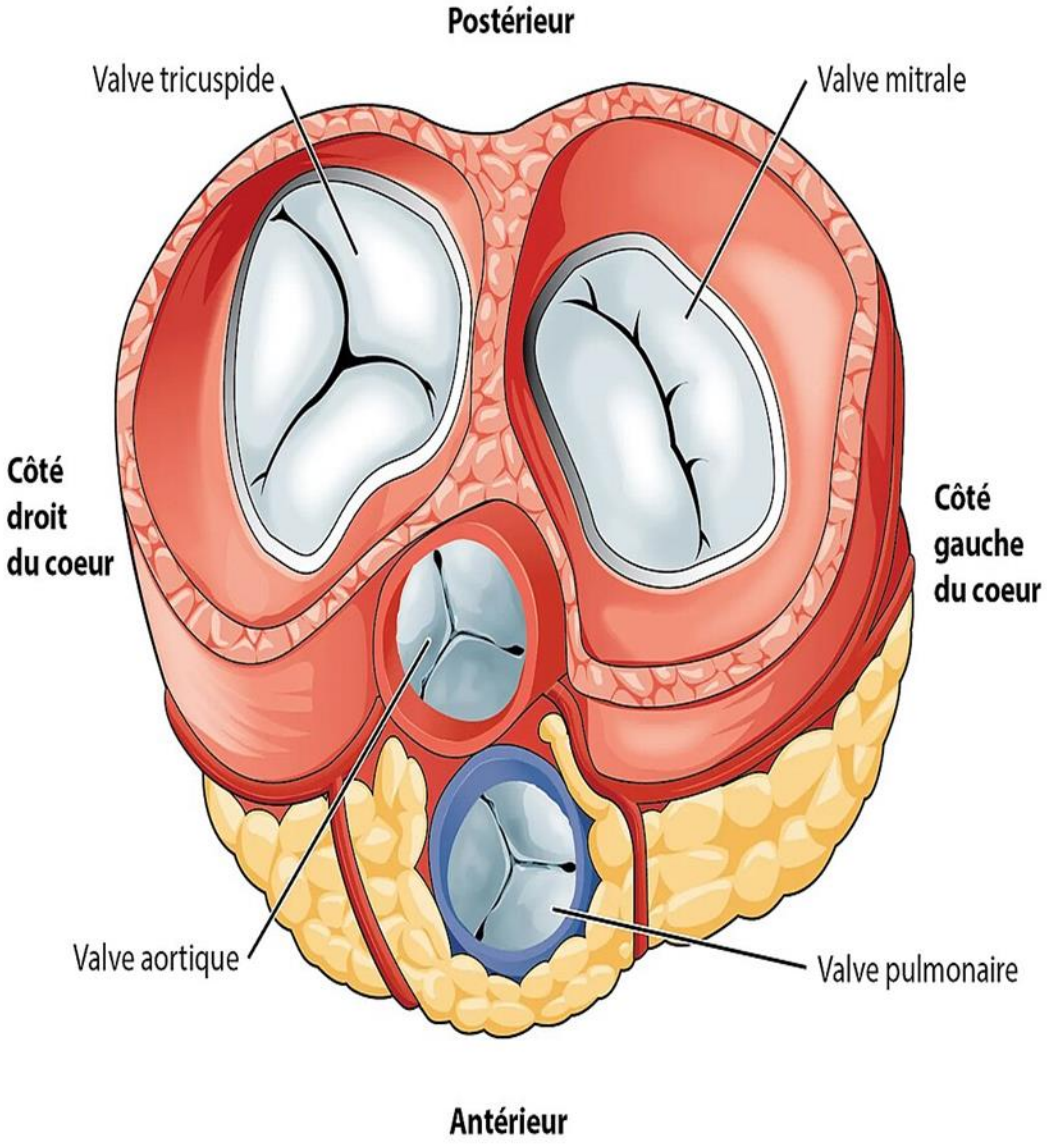
○ Etiology/pathogenesis

- The mitral valve is located between the left atrium and the left ventricle.
- This is a combination of structures known as the mitral valve apparatus, consisting of 6 elements that work together to optimize the systolic function of the left ventricle:



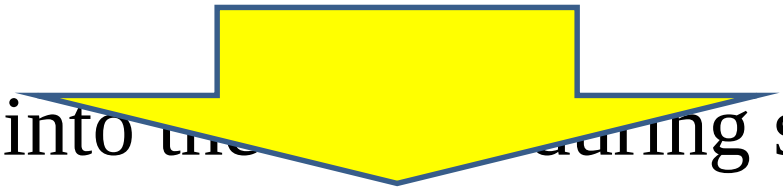


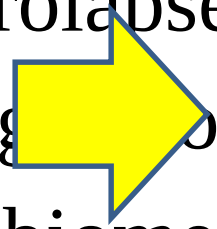
1- Posterior wall of the left atrium; 2 - Mitral ring: The ring of tissue to which the mitral leaflets are suspended; 3- Leaflets of the mitral valve; 4- Chordae tendineae; 5- Papillary muscles of the left ventricle; 6- Myocardium of the left ventricle.

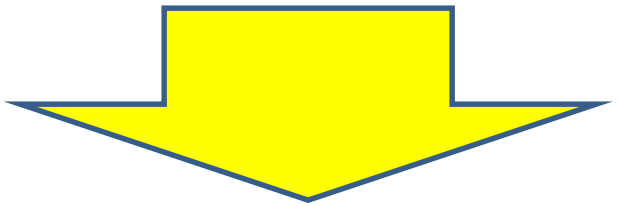

Postérieur
Valve tricuspide
Valve mitrale
Côté droit du coeur
Côté gauche du coeur
Valve aortique
Valve pulmonaire
Antérieur

- MVDM is a progressive myxomatous degeneration of the leaflets of the mitral valve and the chordae tendineae, leading to thickening of the valve leaflets, stretching, and later, rupture of the chordae tendineae, resulting

in prolapse of the valve into the [illegible] during systole (bulging of the valve into the atrium during systole) and biomechanical dysfunction.

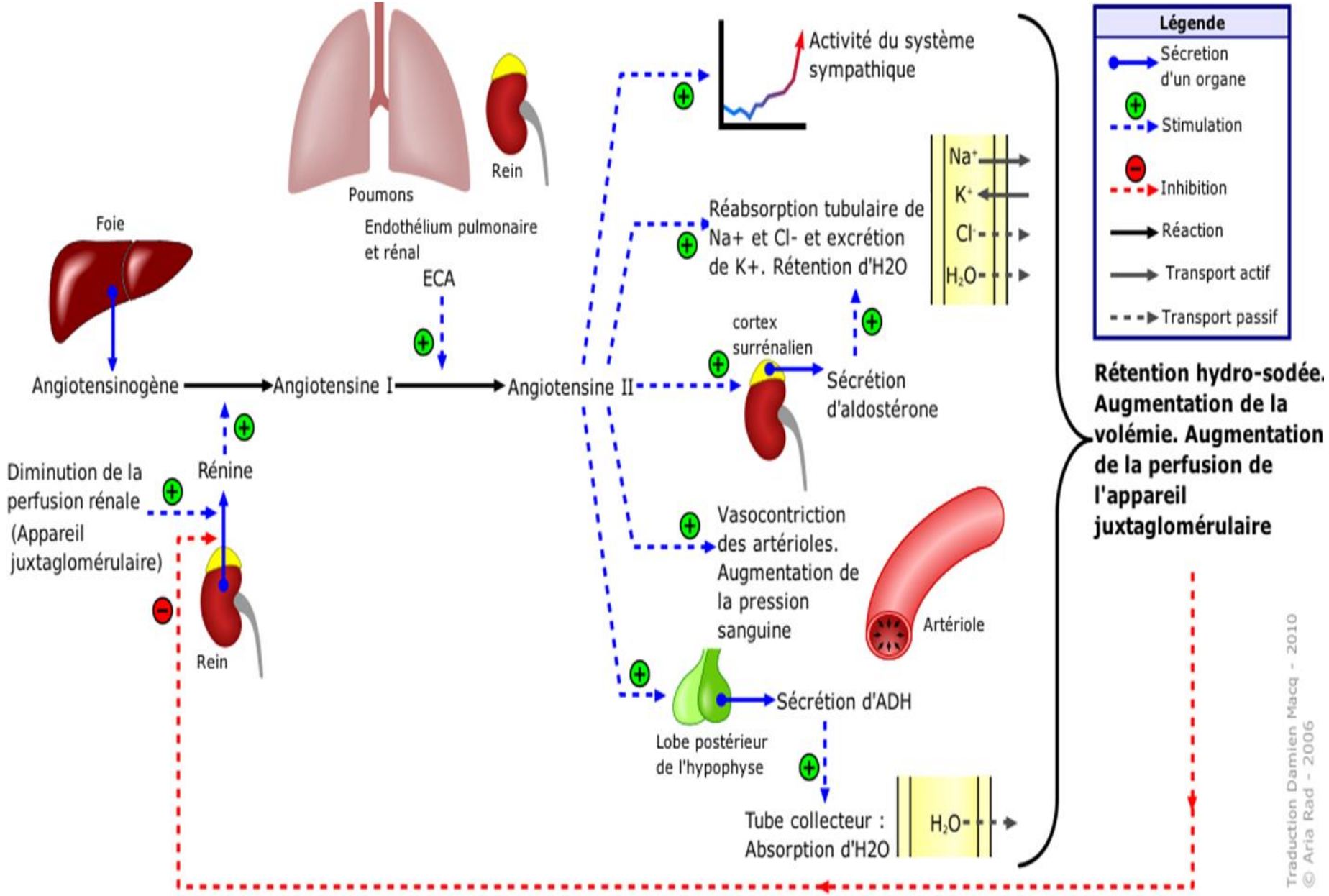






- As the apparatus degenerates, the leaflets of the mitral valve become unable to close properly. Consequently, when blood is ejected from the left ventricle into the aorta during systole, a portion of the blood flows back into the left atrium.
- This pattern of blood flow is called mitral regurgitation and is the most common finding in MVDM. This regurgitant jet causes a heart murmur.
- The greater the degenerative changes, the greater the volume of regurgitation, the louder the heart murmur.

- Large volumes of mitral regurgitation lead to a reduction in cardiac output and, consequently, compensatory mechanisms are activated, notably sympathetic stimulation of vasoconstriction and activation of the renin-angiotensin-aldosterone system (RAAS) to retain sodium and water, in order to maintain systemic blood pressure (BP); these processes lead to an increase in circulating volumes.



- As the disease progresses, left heart volume overload (increased intracardiac pressures) occurs, leading to remodeling of the atria and left ventricle = Elevated pressures and end-diastolic volumes lead to ventricular dilation and hypertrophy).
- When the left atrium and ventricle dilate to accommodate the additional volume, the mitral valve apparatus is forced apart, which increases the volume of regurgitation.

- The greater the regurgitation jet, the more compensatory mechanisms are activated to increase the volume of fluid (blood).
- In advanced cases of the disease, congestive heart failure and arrhythmias may occur, and the chordae tendineae may rupture due to additional mechanical stress.
- Forward heart failure (clinical signs: syncope) may occur when cardiac output is insufficient despite the activation of compensatory mechanisms.

- Congestive heart failure (CHF) is a syndrome caused by elevations in venous pressure resulting from cardiac dysfunction:
  - Left CHF is defined by the presence of cardiogenic pulmonary edema (clinical signs: exercise intolerance or dyspnea). This occurs partly due to increased pressures in the left atrium, and also due to hypoxic pulmonary vasoconstriction (HPV).
  - In advanced cases of MVDM, right congestive heart failure (rare) may also occur (systemic congestion; in dogs, ascites is the most common manifestation of right CHF).

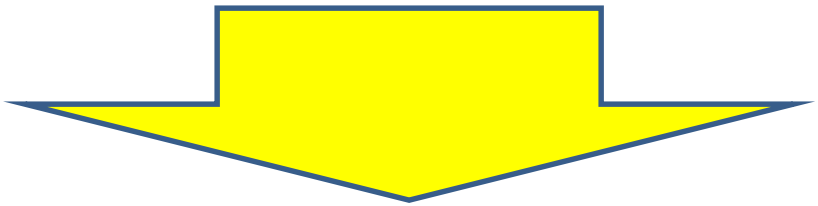
Compensatory mechanisms activated when cardiac output is compromised

Système adrénergique	Système neurohormonal ou système rénine angiotensine-aldostérone (SRAA)
• Augmentation du rythme cardiaque • Vasoconstriction • Augmentation de la contractilité	• Vasoconstriction • Rétention de sodium • Rétention d'eau

- The mitral valve is the most frequently and severely affected; many cases also show changes in the tricuspid valve, although right-sided enlargement and right CHF are rare consequences.

○ Stages of MVDM

The classification of the different stages of the disease adopted by the American College of Veterinary Internal Medicine (ACVIM) is presented in the table below (widely recognized):



Stade	Critères de classification	Description
A	À risque	Les races de chiens présentant un risque élevé de développer une MVDM, mais ne présentent aucun signe de maladie cardiaque.
B	Maladie cardiaque présente, mais pas d'insuffisance cardiaque	Deux catégories distinctes à ce stade : • **B1 –** Souffle cardiaque audible à l'auscultation, mais pas de modifications échocardiographiques ou radiographiques (aucune indication d'hypertrophie du cœur). • **B2 –** Souffle cardiaque audible à l'auscultation, avec présence de modifications échocardiographiques et/ou radiographiques liées à la maladie de la valve mitrale, comme l'hypertrophie du côté gauche.
C	Insuffisance cardiaque congestive (ICC)	Insuffisance cardiaque congestive (ICC) : • Chiens pris en charge par médicaments et traités en ambulatoire. • Patients souffrant d'insuffisance cardiaque aiguë mettant la vie en danger.
D	ICC réfractaire	ICC réfractaire : • Les médicaments de routine contre l'insuffisance cardiaque ne sont plus efficaces

- **Clinical study**

Anamnesis elements

- It is possible for MVDM to remain asymptomatic for long periods, but it may also never develop clinical signs.
- The disappearance of sinus arrhythmia and the appearance of tachycardias over time indicate likely progressive cardiac remodeling.
- It should be noted that the heart murmur is often present for several years, gradually becoming louder before the appearance of clinical signs.

Clinical signs

There is a great diversity of clinical signs that can be divided into major and minor signs.

- **Major clinical signs: The disease can be asymptomatic. However, the main symptoms that may appear are cough, dyspnea, exercise intolerance, and lethargy.**
- **Minor clinical signs: Minor signs such as syncope and weight loss may occur.**

- **Potential complications may also occur, including the onset of left heart failure, pulmonary hypertension secondary to high left atrial pressure that may lead to right heart failure, rupture of the chordae tendineae (which can lead to acute decompensation), and a tear of the left atrium (which can lead to pericardial effusion that is often fatal).**

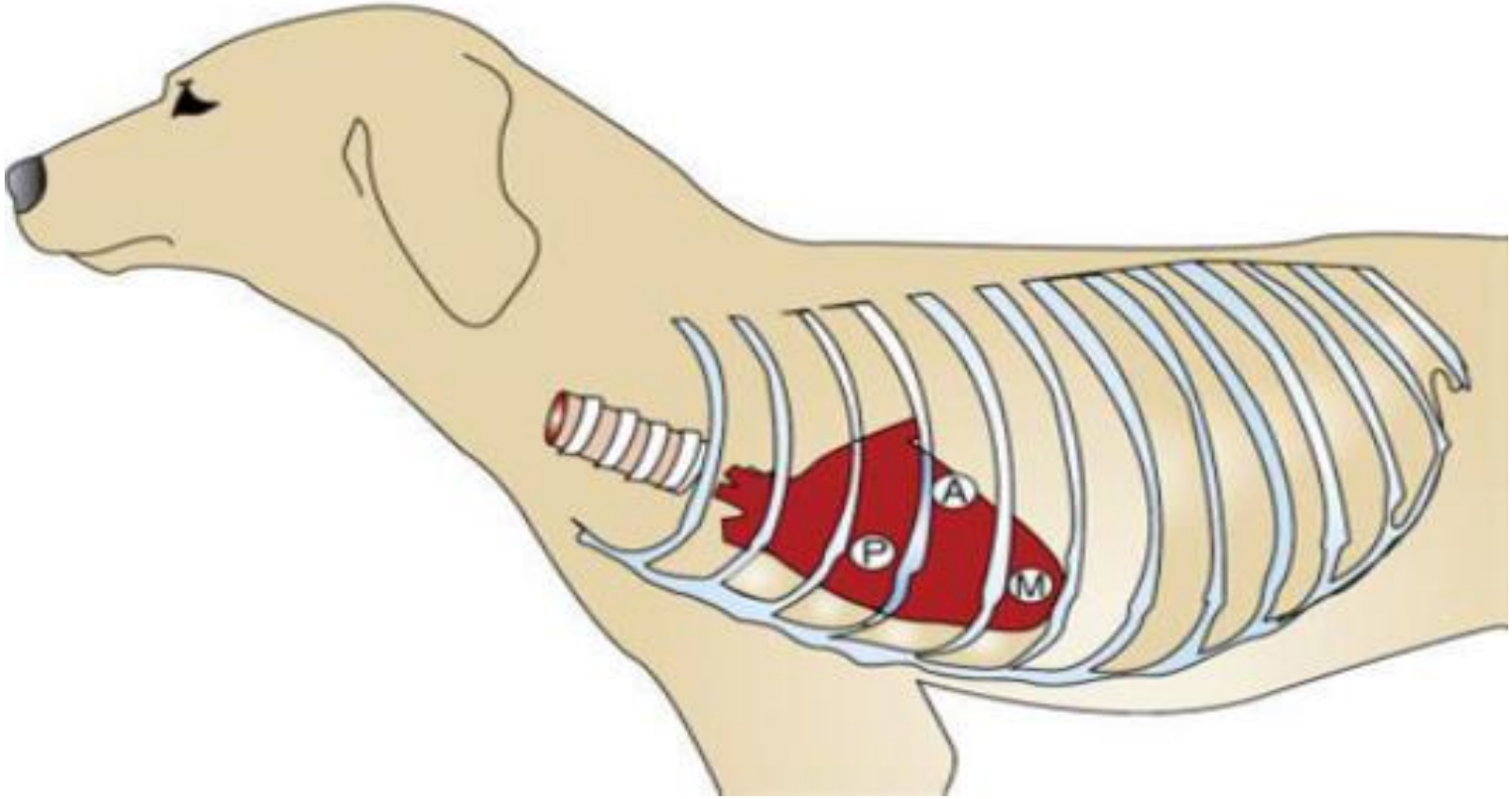
Clinical signs of heart failure resulting from a MVDM

- Souffle cardiaque : Généralement de grade IV à VI ; le grade VI en cas d'insuffisance cardiaque
- Toux
- Tachypnée, détresse respiratoire, orthopnée
- Léthargie
- Anorexie
- Intolérance à l'effort
- Syncope
- Perte de poids et de masse musculaire – Aux stades avancés de la maladie
- Arythmies : Le plus souvent, des complexes d’extrasystoles ventriculaires et la fibrillation auriculaire
- Ascite : Dans le cas du développement d’une insuffisance cardiaque droite
- Signes insuffisance cardiaque antérograde dans les cas extrêmes : Faiblesse, collapsus, pâleur, pouls faible ou absent

Clinical examination

Cardiac auscultation reveals a heart murmur characterized by the following description:

- Left apical systolic murmur: Band-shaped systolic murmur with a Point of Maximum Intensity (PMI) at the left apex.
- The grade of the murmur intensifies as the disease progresses.
- In severe forms of the disease, the breath can widely radiate (extend).



Cardiac auscultation zones of the left hemithorax. Heart valves: Pulmonary (P), aortic (A), and mitral (M).

- The presence of sinus arrhythmia (parasympathetic vagal tone) suggests that the risk of contracting CHF is unlikely to be high in the near future.
- Signs of CHF on the left side, such as dyspnea, pulmonary crackles, and tachycardia, could be detected through a clinical examination.

○ Paraclinical examination

Laboratory tests

- Laboratory test results are generally not characteristic.
- However, the degree of increase in cardiac biomarkers (NT-proBNP [N-terminal Pro B-Type Natriuretic Peptide] and troponin I) correlates with the severity of the disease.

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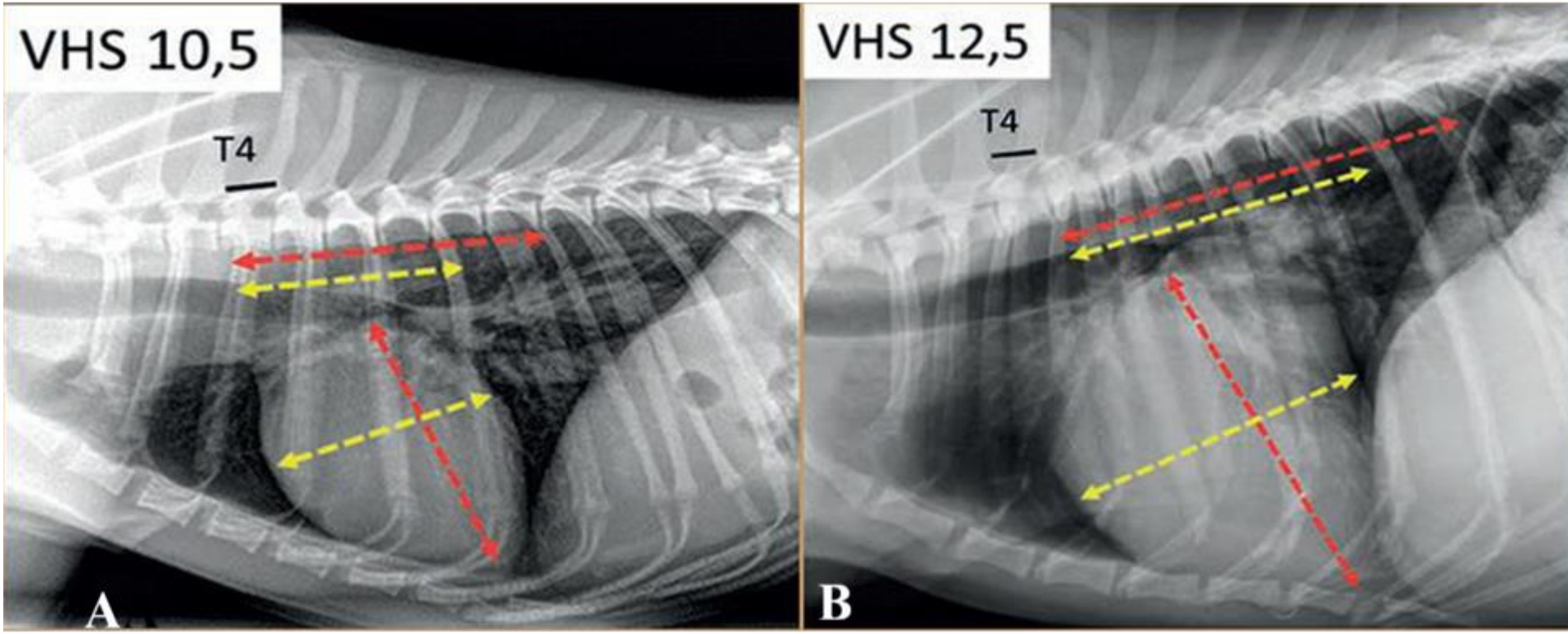
Imaging

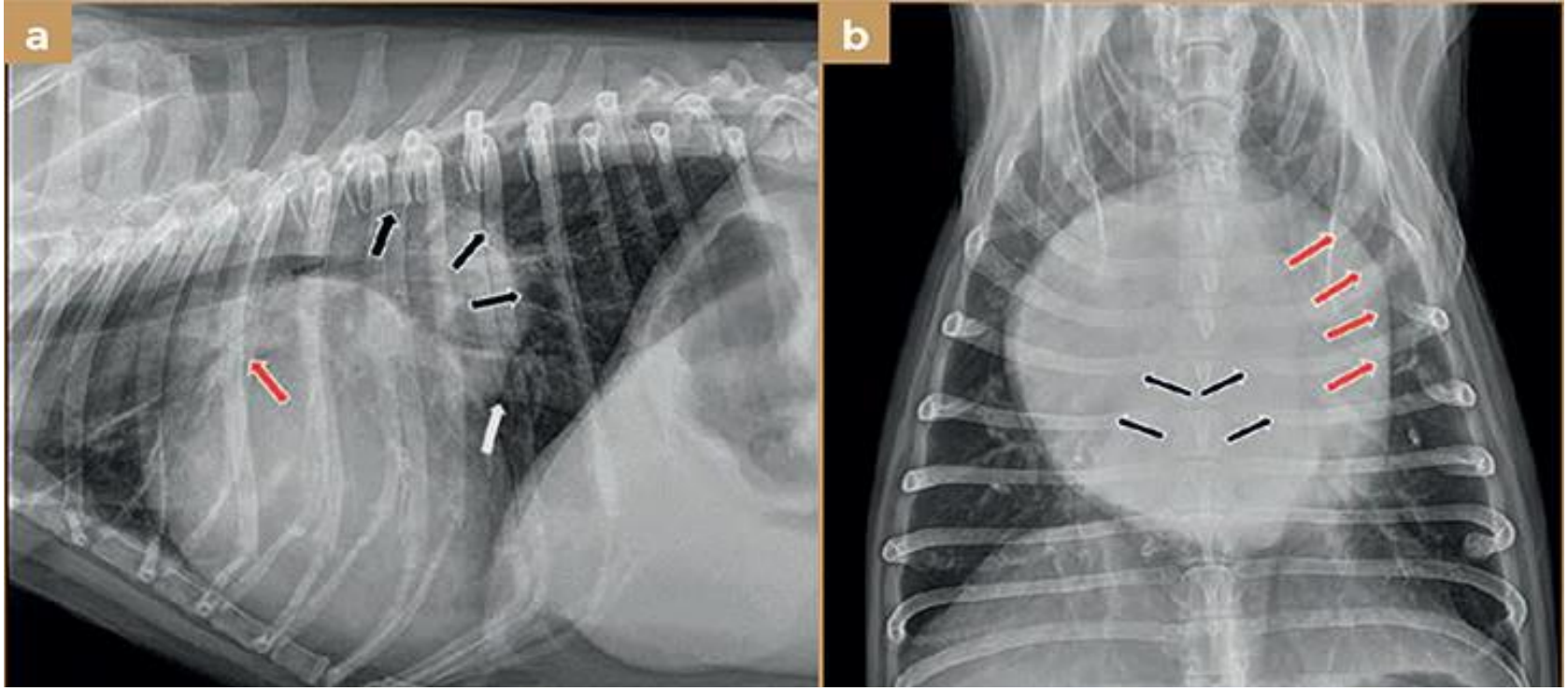
X-ray

A simple X-ray (frontal and lateral) of the thorax may reveal the following elements:

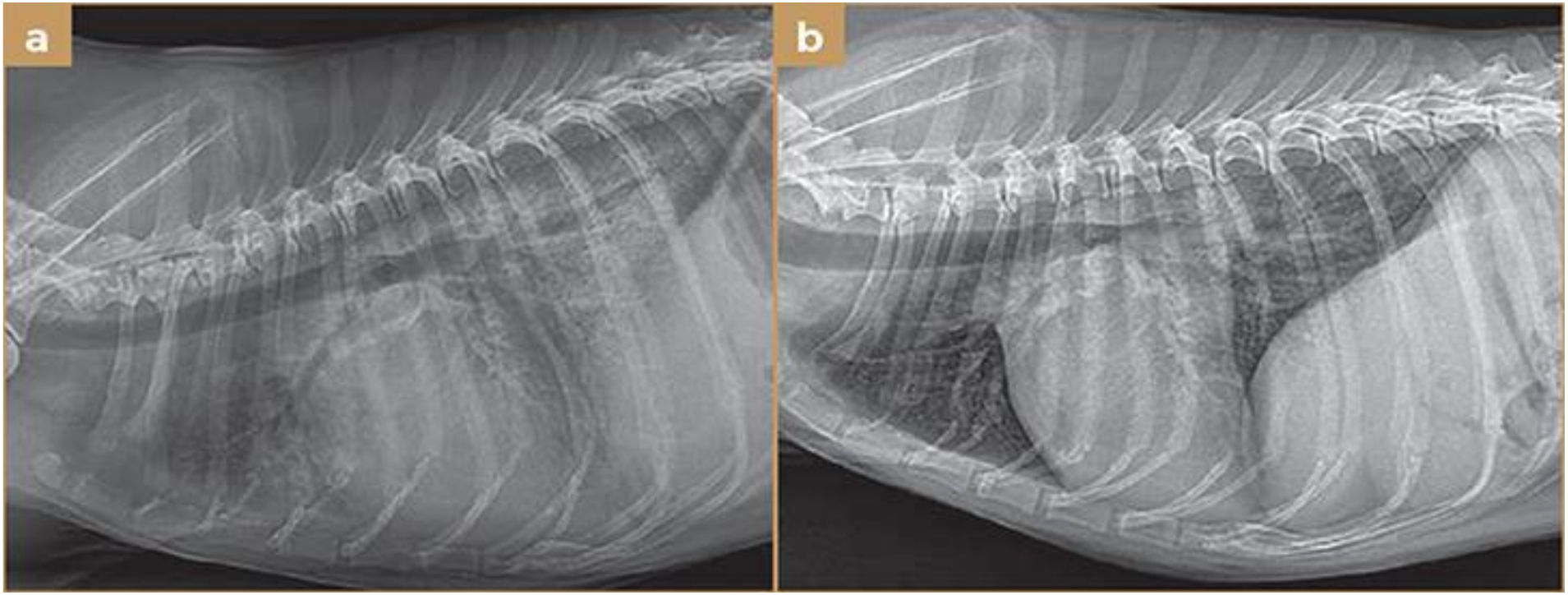
- May be normal at the onset of the disease.
- Progressive left (or global) cardiomegaly.
- Pulmonary venous congestion.
- Interstitial or alveolar pattern related to left CHF.



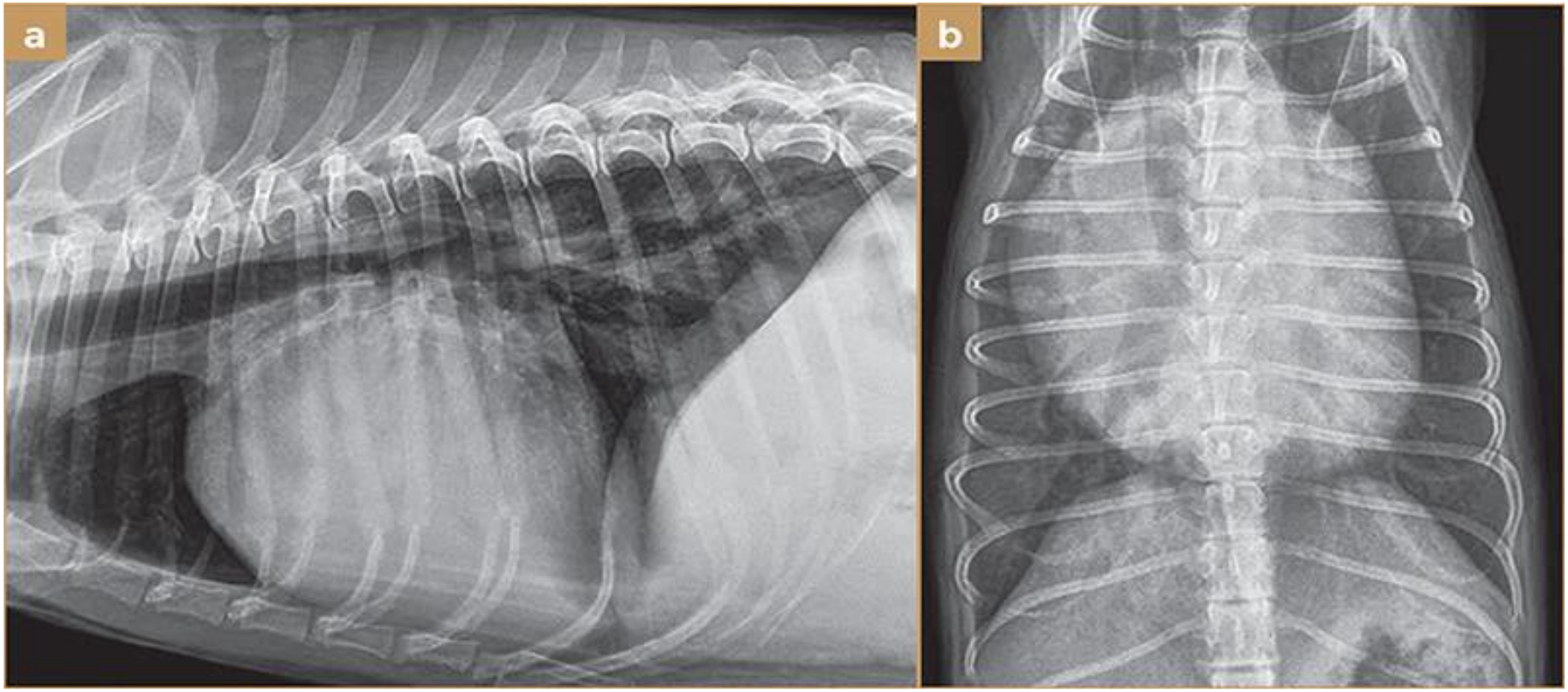
Radiographic measurement of the cardiac size of a normal dog (a) and of a dog with moderate cardiac enlargement (b) using the vertebral heart score (VHS) method. VHS >10.5 is considered suggestive of cardiac enlargement in many dogs, but breed variation exists.



Extreme cardiac hypertrophy in a dog with severe MVD. a) In the lateral view, the caudal margin is straight and extends dorsally into an enlarged left atrium (black arrows). The dilated left ventricle raises the cranial part of the caudal vena cava (white arrow). There is dilation of the pulmonary veins (red arrow). b) In the ventro-dorsal view, the dilated left atrium laterally displaces the caudal main bronchi (black arrows). The significantly dilated left atrium outlines the cardiac silhouette (red arrows).



Lateral views of a dog with severe myxomatous mitral valve disease. a) The first radiograph shows cardiomegaly and acute, interstitial, and alveolar pulmonary edema. b) After one week of aggressive diuretic therapy, a residual interstitial infiltrate and a remarkable reduction in the width of the cardiac silhouette could be observed.

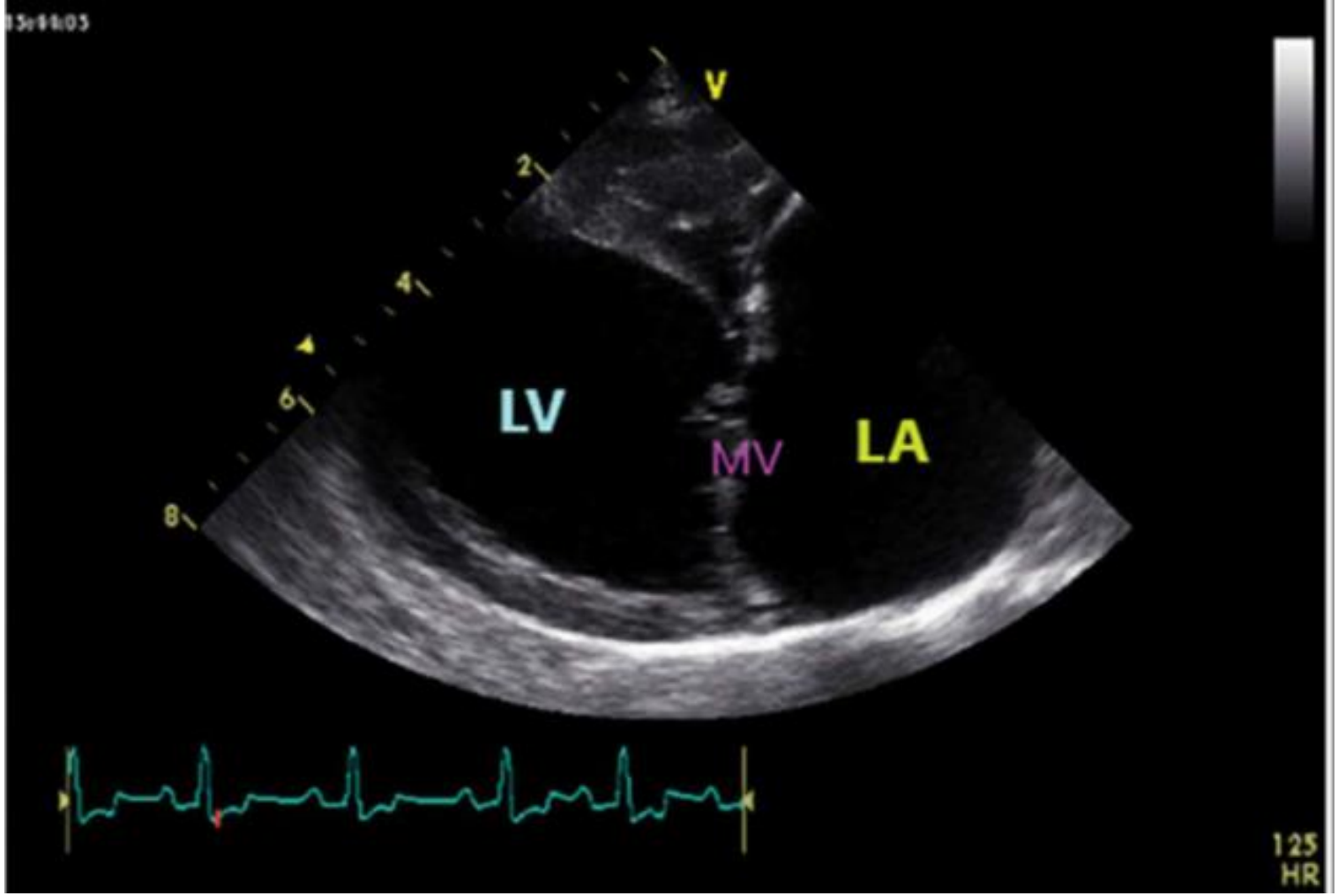


Radiographs of a dog with severe MVD and severe cardiomegaly due to right ventricular hypertrophy from type 2 pulmonary hypertension. a) The lateral view shows an enlarged right ventricle with increased contact between the right ventricular outflow tract and the sternum. b) The cardiac silhouette has an inverted "D" shape on the ventrodorsal view.

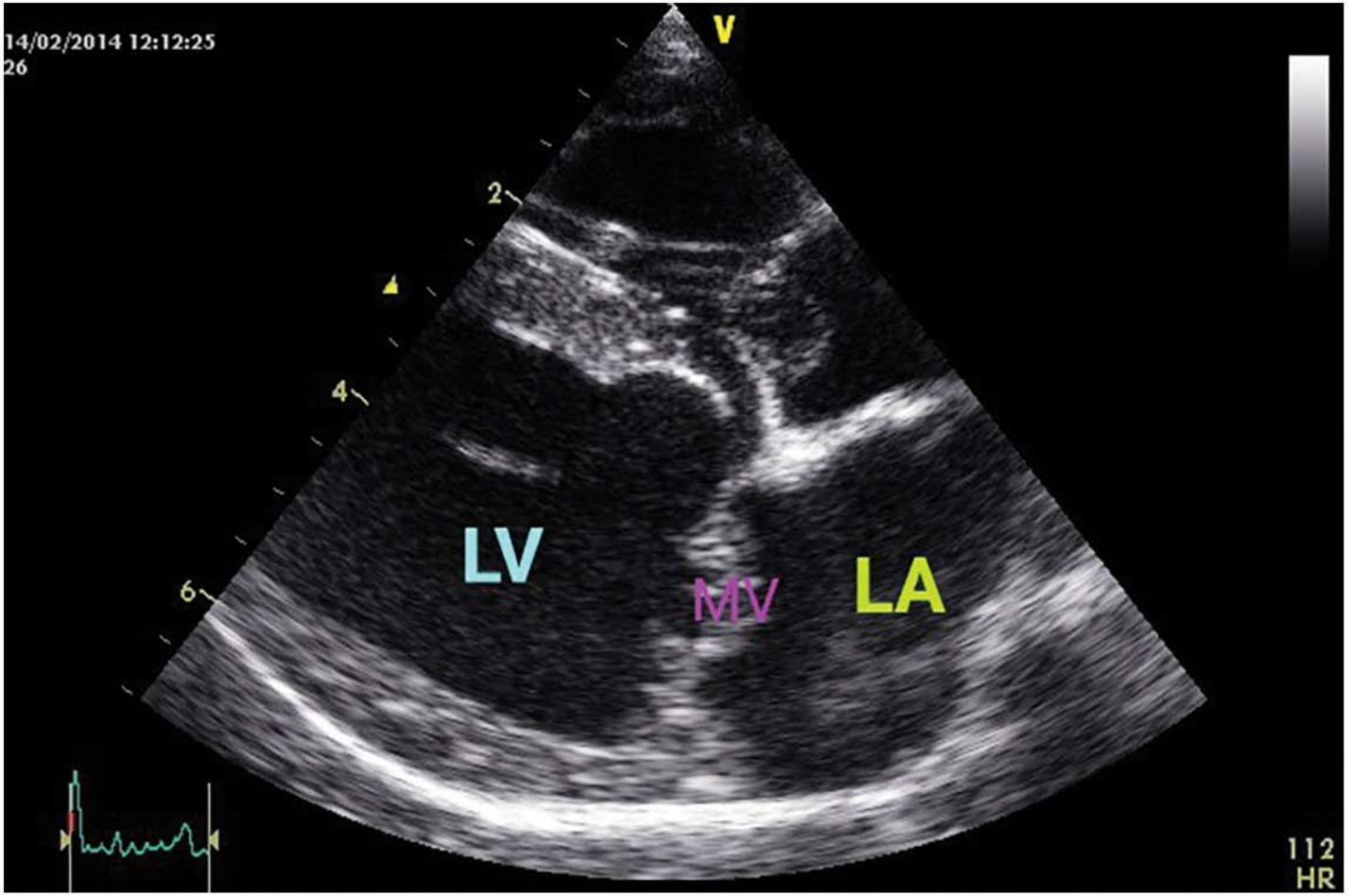
Echocardiography

The M mode (M-mode [motion mode] and 2D [two-dimensional]) of echocardiography can demonstrate:

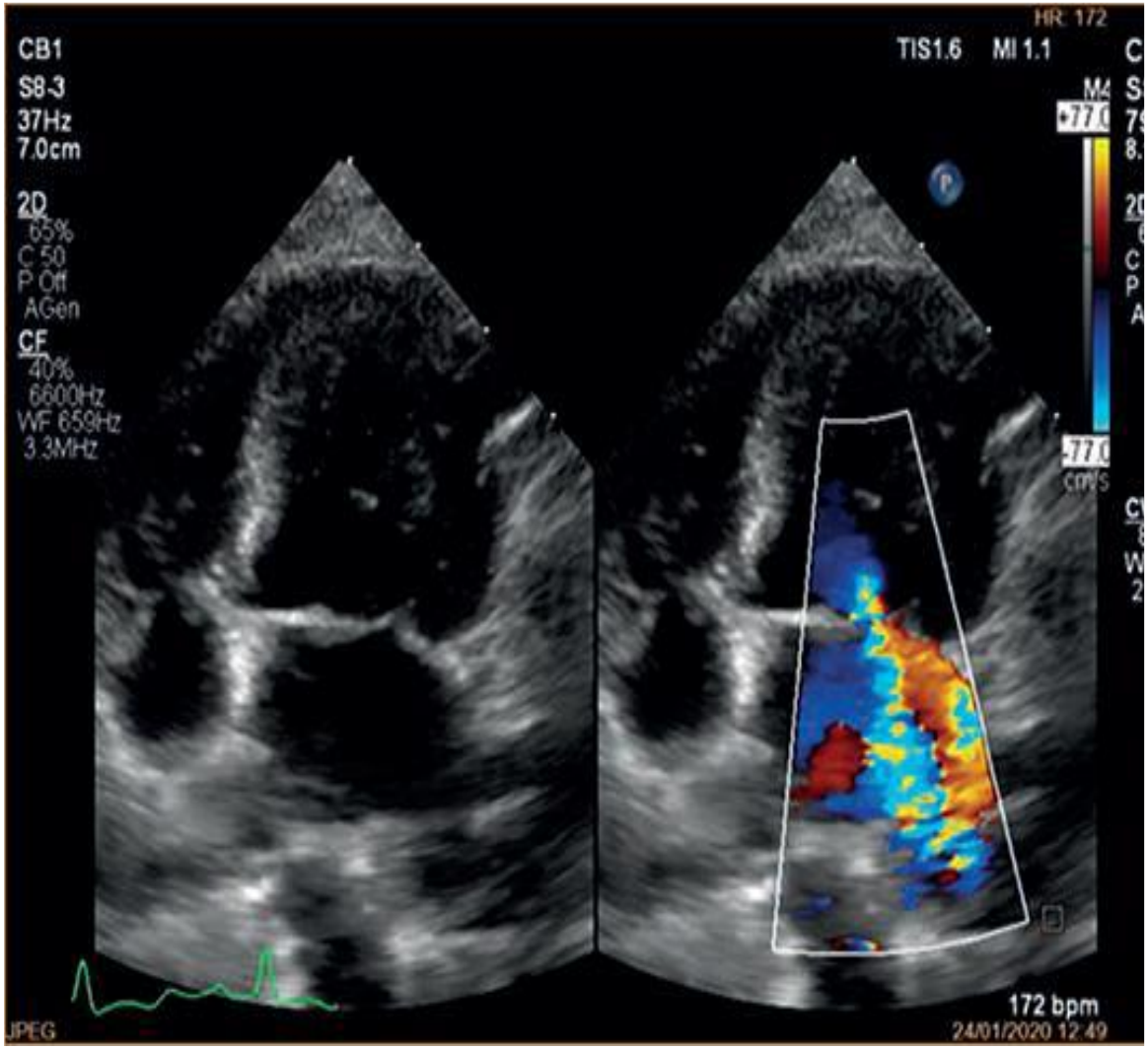
- Thickening and prolapse of the mitral valve: the degree varies according to the severity of the disease (and the breed).
- Secondary dilation of the left ventricle and left atrium in advanced cases (stage B2 and beyond).
- Color Doppler reveals mitral regurgitation in the left atrium, often with several jets taking eccentric trajectories.



Echocardiographic image of MMVD at stage B2. Note the increase in size of the left atrium (LA) and the left ventricle (LV), and the visible thickening of the mitral valve (MV).



Echocardiographic image of MMVD at stage C. Note that the left atrium (LA) and left ventricle (LV) are dilated, and the mitral valve (MV) is stretched and bowing into the left atrium.



Myxomatous mitral valve disease (mild) and mild mitral regurgitation (MR) in a 10-year-old female chihuahua with Cushing's syndrome and systemic hypertension.

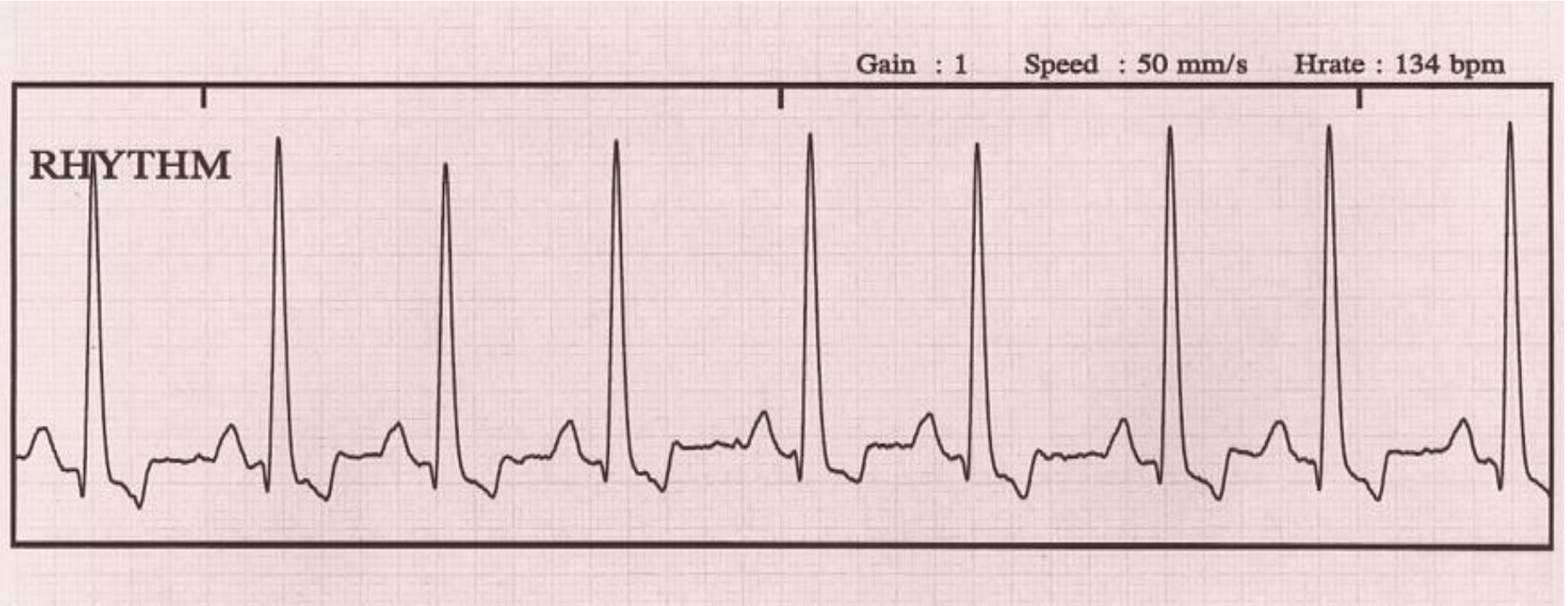
Electrocardiography (ECG)

- The ECG of a dog with MMVD is often normal (particularly in the early stages).
- However, the abnormalities indicated below may manifest:

➢ Widening of the P wave (increased duration of the P wave = wide P waves).

➢ Increase in the amplitude of the R wave (tall R waves).

➢ Arrhythmias (generally supraventricular) in advanced cases.



Electrocardiogram recorded in a 13-year-old Beagle showing physical signs of mitral regurgitation (MR). A wide P wave and an R amplitude greater than 3 mV are observed, suggesting left atrial dilation and left ventricular hypertrophy, respectively. (Lead II, 50 mm/sec, 1 mV = 1 cm.).

Salvos of supraventricular tachycardia recorded by 24-hour Holter monitoring in a dog with myxomatous mitral valve disease.

○ Treatment and monitoring

Treatment depends on the stage of disease progression:

- ➢ Stage B2: Pimobendan has been shown to delay the onset of CHF.
- ➢ Stage C: Treatment focuses on management of left-sided CHF.
  - ❖ Diuretics (furosemide, spironolactone).
  - ❖ Pimobendan.
  - ❖ Inhibitors of the angiotensin-converting enzyme (ACE; example: benazepril, ramipril).
  - ❖ Antiarrhythmic treatment based on ECG results and clinical signs.

- Surgical valve repair or replacement: The availability of surgical treatment is limited.
- It should be noted that monitoring at-risk dogs (certain breeds, older dogs) is necessary:

➢ Auscultation: Searching for a systolic murmur on the left side.

➢ Clinical signs: Coughing, dyspnea, exercise intolerance, etc.

➢ Imaging (radiography or echocardiography): Particularly in patients without clinical signs to identify the progression from stage B1 to stage B2.

Prognosis

- The progression and prognosis of the disease are variable, with many dogs remaining asymptomatic throughout their lives.
- However, starting from the beginning of the ICC, the average survival duration is 12 to 18 months with medical