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Feline Hypertrophic Cardiomyopathy (HCM)

Dr. Djemai Samir

Introduction

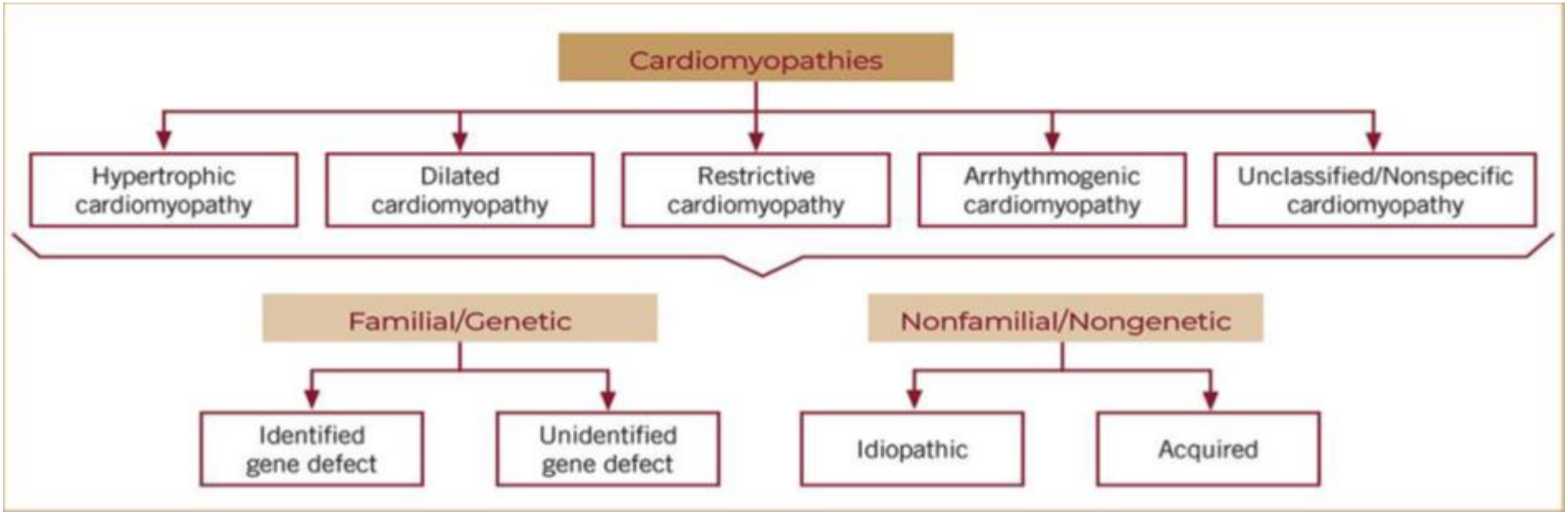
- Cardiomyopathies represent a significant heterogeneous group of myocardial diseases in humans, dogs, and cats, and are associated with failure of mechanical function (systolic or diastolic dysfunction) or cardiac electrical function (predisposition to arrhythmias).
- The European Society of Cardiology proposed the following definition of cardiomyopathy in 2008: "A myocardial disorder in which the heart muscle is structurally and functionally abnormal, in the absence of coronary artery disease, hypertension, valvular disease, and congenital heart disease, which may cause an observed myocardial abnormality."

Cardiomyopathies are classified into distinct morphological and functional phenotypes (a phenotype is an observable and thus measurable trait):

- Hypertrophic cardiomyopathy (HCM; Hypertrophic cardiomyopathy [HCM]).
- Dilated cardiomyopathy (DCM; Dilated cardiomyopathy [DCM]).
- Restrictive cardiomyopathy (RCM; Restrictive cardiomyopathy [RCM]).
- Arrhythmogenic cardiomyopathy (ACM).
- Unclassified/nonspecific cardiomyopathy (NSCM).

Each phenotype is subdivided into familial and non-familial forms:

- The term 'familial cardiomyopathies' refers to the presence of the same disorder or phenotype in several members of the same family, which is (or could be) caused by the same genetic mutation, rather than by an acquired cardiac or systemic disease.
- Non-familial cardiomyopathies are clinically defined by the appearance of cardiomyopathy in an individual, while other family members do not show it (based on genealogical analysis and clinical evaluation).



Classification system for cardiomyopathies proposed (in 2008) by the European Society of Cardiology based on morphological and functional phenotypes.

- Non-familial cardiomyopathies are subdivided into idiopathic cardiomyopathies (cause not identifiable) and acquired, in which ventricular dysfunction is a complication rather than an intrinsic characteristic of the disease.

- It is worth noting that HCM is the most frequently described form of cardiomyopathy in cats (57.5%), followed by DCM (20.7%), CMD (10.4%), and ACM (1%).
- It should also be noted that the percentage of unclassified/nonspecific cardiomyopathies is around 10.4%.

Table. Description of the phenotypic groups of feline cardiomyopathies.

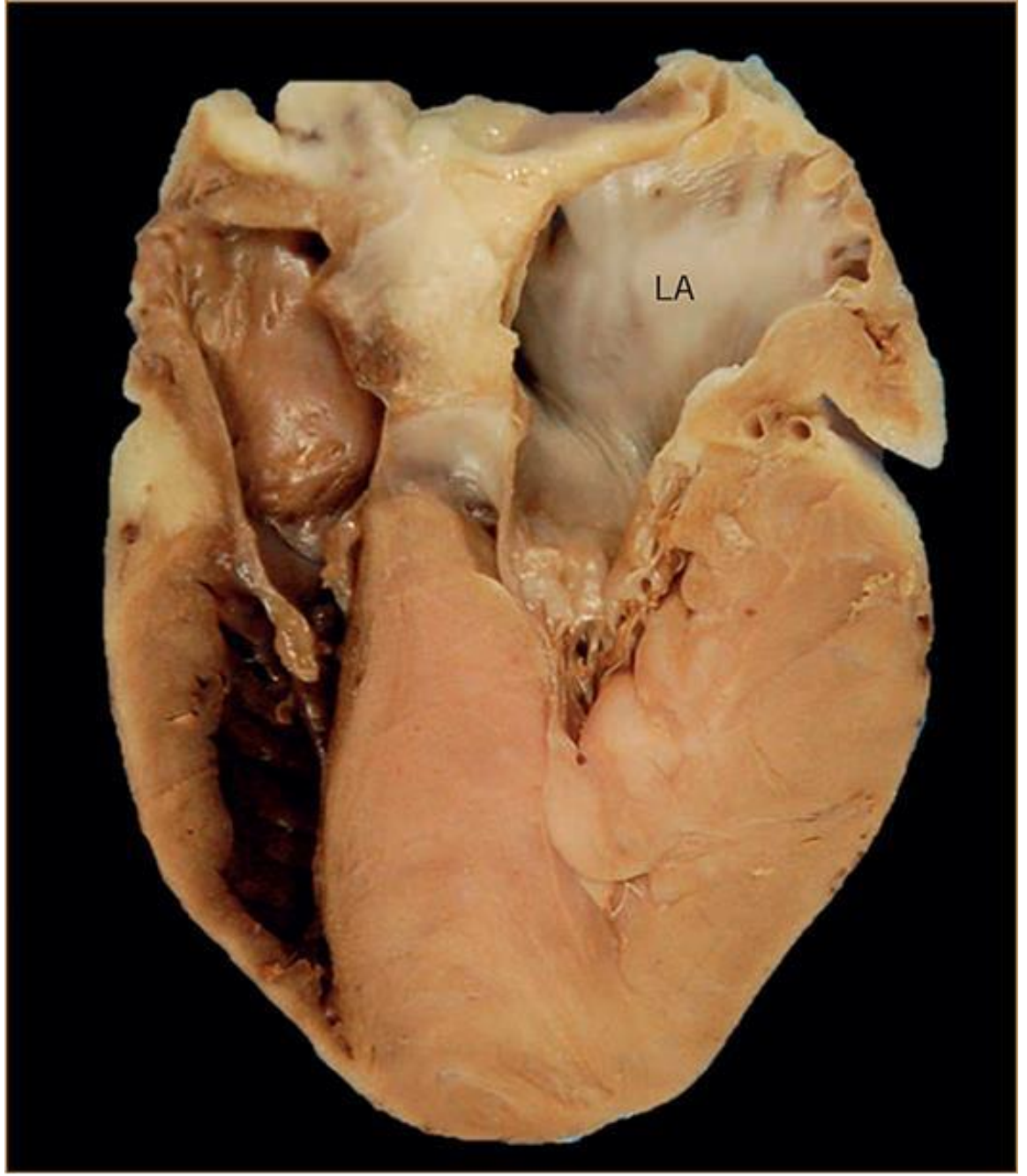
Phénotype	Définition
Cardiomyopathie hypertrophique (CMH)	• Augmentation diffuse ou régionale de l'épaisseur de la paroi du ventricule gauche.
Cardiomyopathie restrictive (CMR)	• La CMR est définie comme un remplissage restreint et un volume diastolique réduit d'un ou des deux ventricules avec une fonction systolique normale ou réduite et une épaisseur de paroi normale. • Mesures ventriculaires gauches normales avec dilatation auriculaire gauche (ou dilatation biauriculaire).
Cardiomyopathie dilatée (CMD)	• Dysfonctionnement systolique du ventricule gauche. • Épaisseur de la paroi ventriculaire gauche normale ou hypertrophiée ; dilatation de la chambre ventriculaire gauche et dilatation auriculaire.
Cardiomyopathie arythmogène	• Dilatation sévère de l'oreillette droite et du ventricule droit. • Arythmies et insuffisance cardiaque congestive droite fréquentes.
Phénotype non spécifique	• Les phénotypes cardiomyopathiques qui ne sont pas bien expliqués.

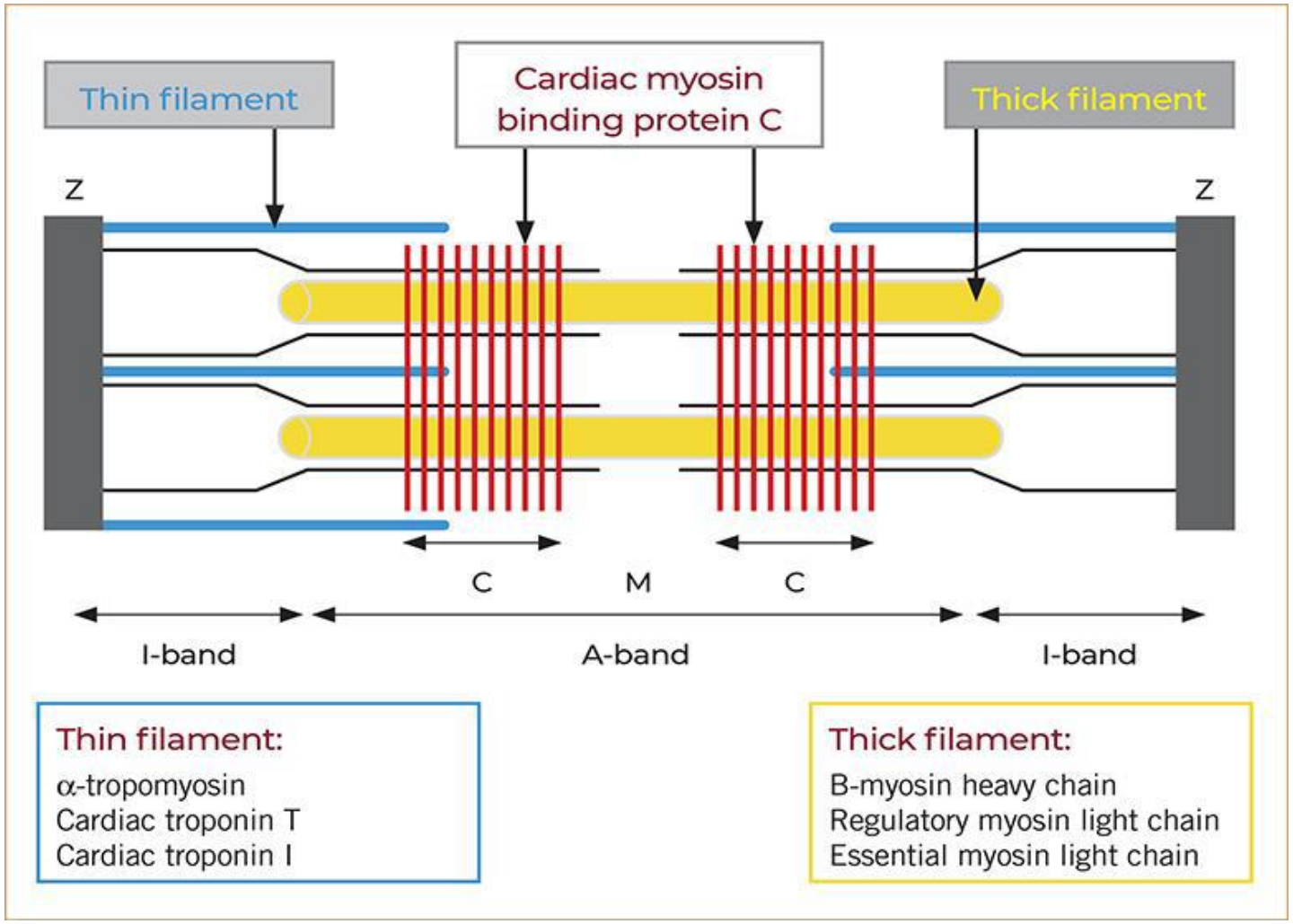
These descriptions are based on echocardiographic results.

Definition of Hypertrophic Cardiomyopathy (HCM)

- Hypertrophic Cardiomyopathy (HCM) is the most common form of myocardial diseases in cats.
- It is a sarcomeric disease that results in an increase in cardiac mass (diffuse or regional increase) characterized by hypertrophy of the left ventricular wall (the ventricle is not dilated) and of the interventricular septum in the absence of hemodynamic loading conditions or metabolic cause (aortic stenosis, systemic hypertension, hyperthyroidism, and acromegaly, etc.).

Post-mortem specimen of a 4-year-old male Siamese cat with hypertrophic cardiomyopathy. Longitudinal section showing severe concentric hypertrophy of the interventricular septum and the free wall of the left ventricle and a very reduced left ventricular chamber. LA, left atrium.





Organization of sarcomeric proteins associated with hypertrophic cardiomyopathy.

Epidemiology

- HCM is the most frequent form (phenotype) of cardiomyopathy in felines (60% of feline cardiomyopathy cases), and a genetic link is believed to exist.
- Such a link has been demonstrated in Maine Coon and Ragdoll breeds, where the incidence is particularly high.
- European cats (domestic shorthairs) are frequently affected.

Breeds predisposed to HCM.

- Domestic Shorthair (89,1%)
- Domestic Longhair (2,2 %)
- Maine Coon (2,2 %)
- Ragdoll
- Bengal
- British Shorthair
- American Shorthair
- Sphynx
- Sibérien (Siberian)
- Persian (6,5 %)
- Chat des forêts norvégiennes (Norwegian Forest)

Maine Coon

Ragdoll

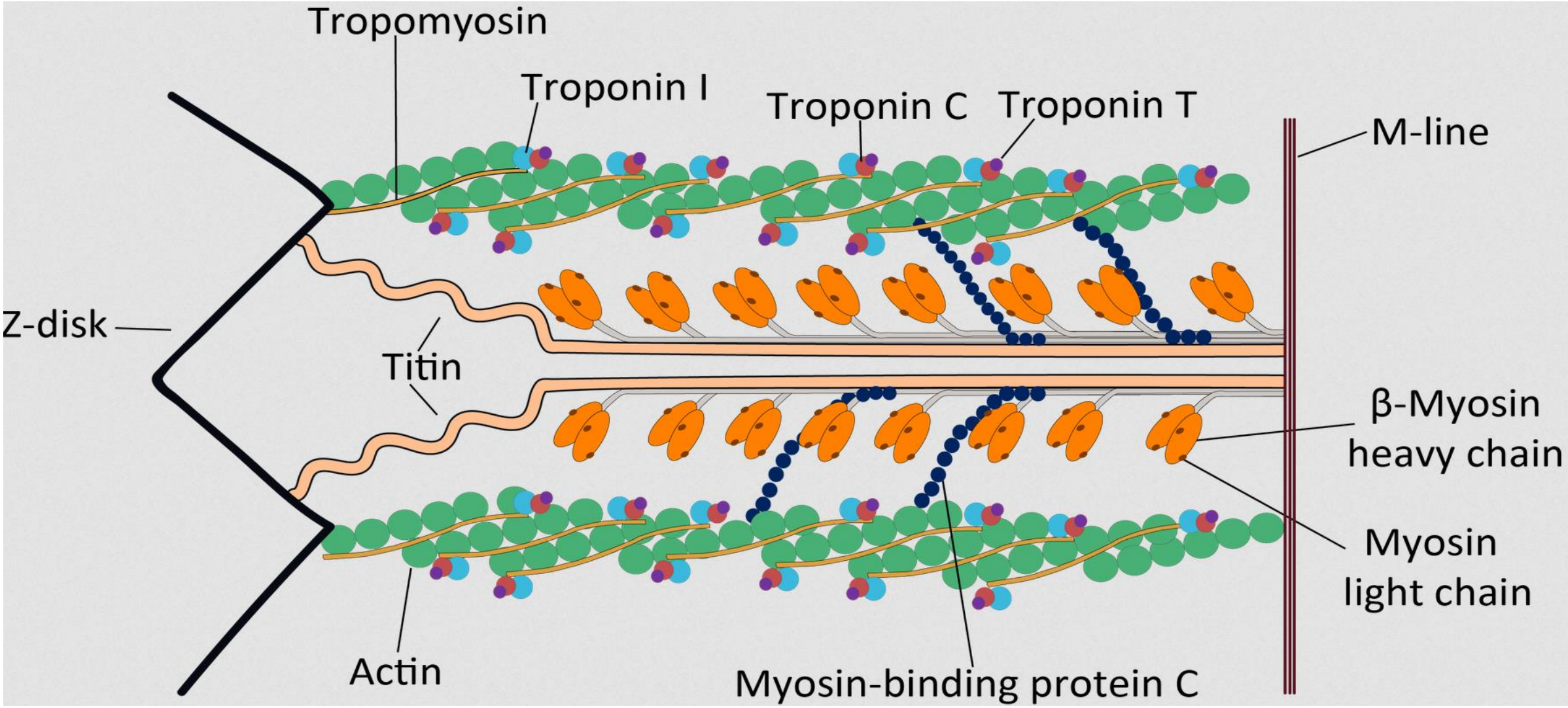
- It is important to note that males are more likely to contract the disease (65 to 70% of cases) than females; this may be linked to genetic or hormonal influence.
- The prevalence within the feline population has been reported between 8% and 15%, although diagnostic criteria vary between studies and prevalence may be higher in certain breeds.

Etiology

- Like in human medicine, feline HCM is a genetic disease with autosomal dominant transmission, incomplete penetrance, and variable expressivity.
- **Example = Two missense sarcomeric mutations have been identified in the gene coding for myosin-binding protein C (MYBPC3):**

❑HCM associated with the mutation 'A31P' in Maine Coon cats: DNA sequencing revealed a change of a single base pair; from guanine (G) to cytosine (C) in codon 31 (exon 3) in affected cats but in none of the unaffected control cats. This changed the amino acid from alanine to proline (A31P).

❑HCM associated with the mutation 'R820W' in Ragdoll cats: DNA sequencing revealed a change of a single base pair from cytosine (C) to thymine (T) in codon 820 in all affected cats and in none of the unaffected control cats. This change modified the amino acid from arginine to tryptophan.



An illustration showing various components of the cardiac sarcomere, including the myosin-binding protein C

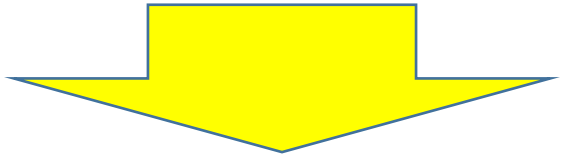
- Since these 2 induced mutations haveonly been identified in 34 to 40% of Maine Coon cats and 17 to 23% of Ragdoll cats, other mutations or unknown factors are considered to contribute to the HCM phenotype and are still under research (epigenetic factors, sex, obesity, systemic arterial hypertension, etc.).
- More than 1,400 mutations in 27 genes coding for sarcomeric proteins or other structural proteins have been identified in humans with HCM.
- In humans, there is a correlation (for certain genes) between the location of the mutation, the severity of hypertrophy, and the incidence of sudden death.

Pathophysiology

- In HCM, it is assumed that the production of abnormal sarcomeric proteins creates a functional defect in the muscle contraction process, leading to increased stress on myocytes with subsequent activation of trophic factors (angiotensin II, aldosterone, Insulin-like Growth Factor [IGF-1]) inducing hypertrophy of myocytes:

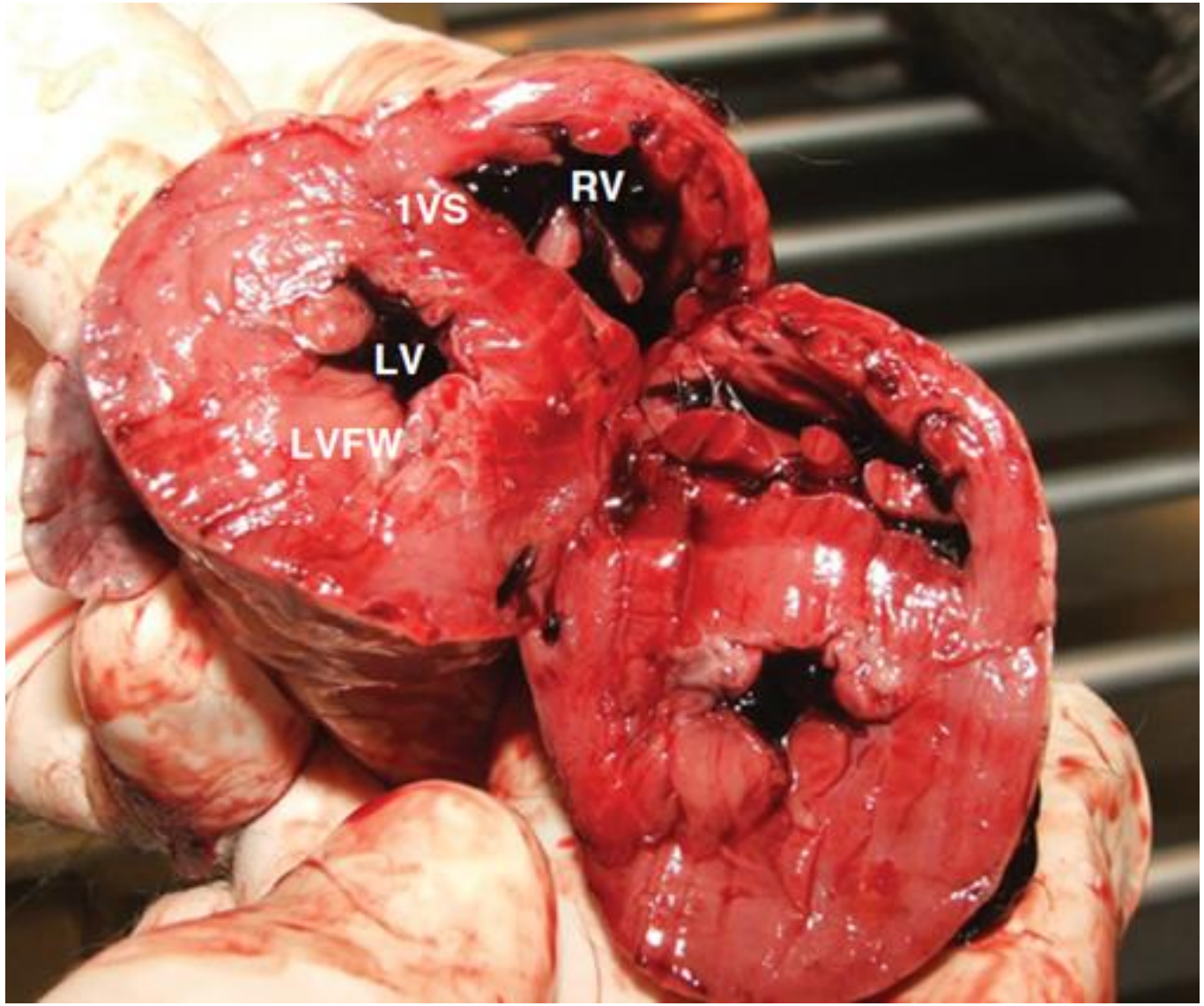
❑Diastolic dysfunction is the main mechanism responsible for the clinical manifestations of HCM and RCM.

❑Left ventricular wall hypertrophy (HCM) results in impaired or delayed relaxation and interstitial/endomyocardial fibrosis (HCM and RCM) increases myocardial stiffness and decreases distensibility (compliance).



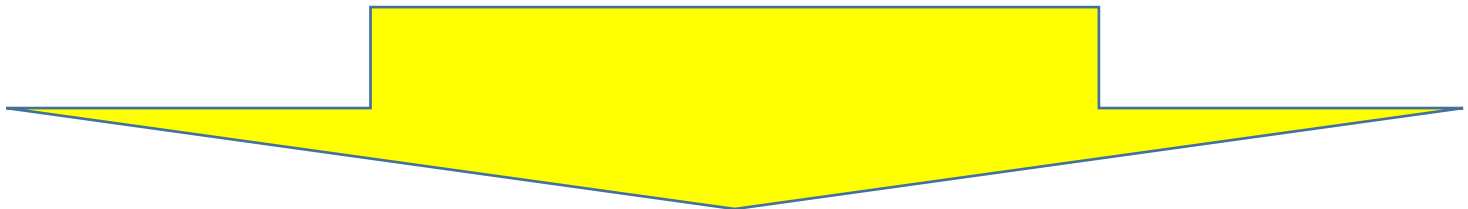
- These changes alter the filling of the left ventricle and result in filling only at higher pressures.
- High filling pressures inevitably lead to dilation of the left atrium, pulmonary venous hypertension, and congestive heart failure (CHF).

Left ventricular wall thickening is the primary lesion of hypertrophic cardiomyopathy. In this cat, the left ventricular chamber (LV) is smaller than the right ventricular chamber (RV) because the thickening of the left ventricular wall is directed inward. IVS, interventricular septum; LVFW, left ventricular free wall.



The main mechanisms that influence the clinical phenotypes of HCM are as follows:

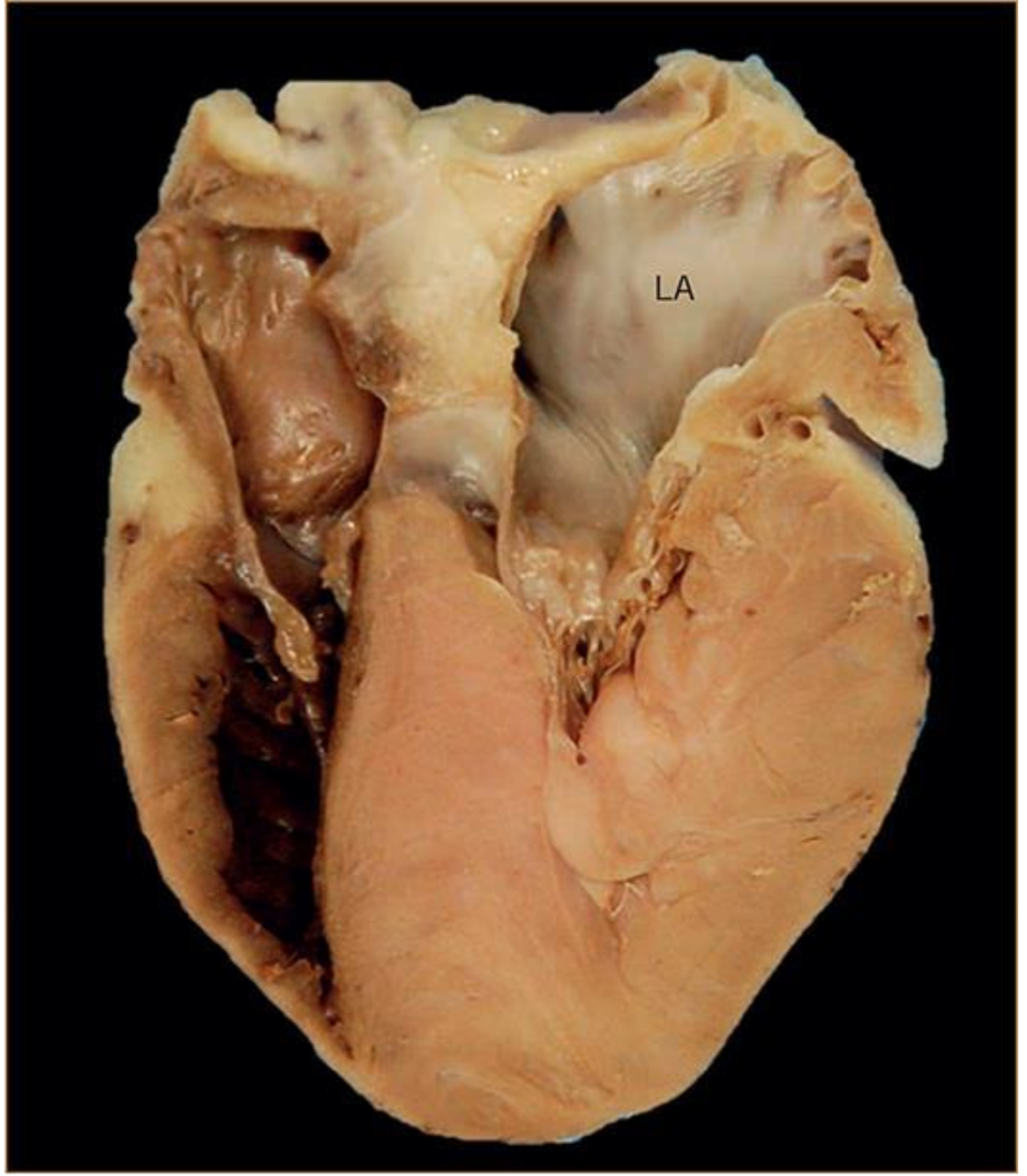
- Diastolic dysfunction.
- Obstruction of the left ventricular outflow tract (left ventricular outflow tract [LVOT]).
- Complications = heart failure + pulmonary edema + pleural effusion.
- Complication = arterial or aortic thromboembolism (TEA; Aortic thromboembolism [ATE]).



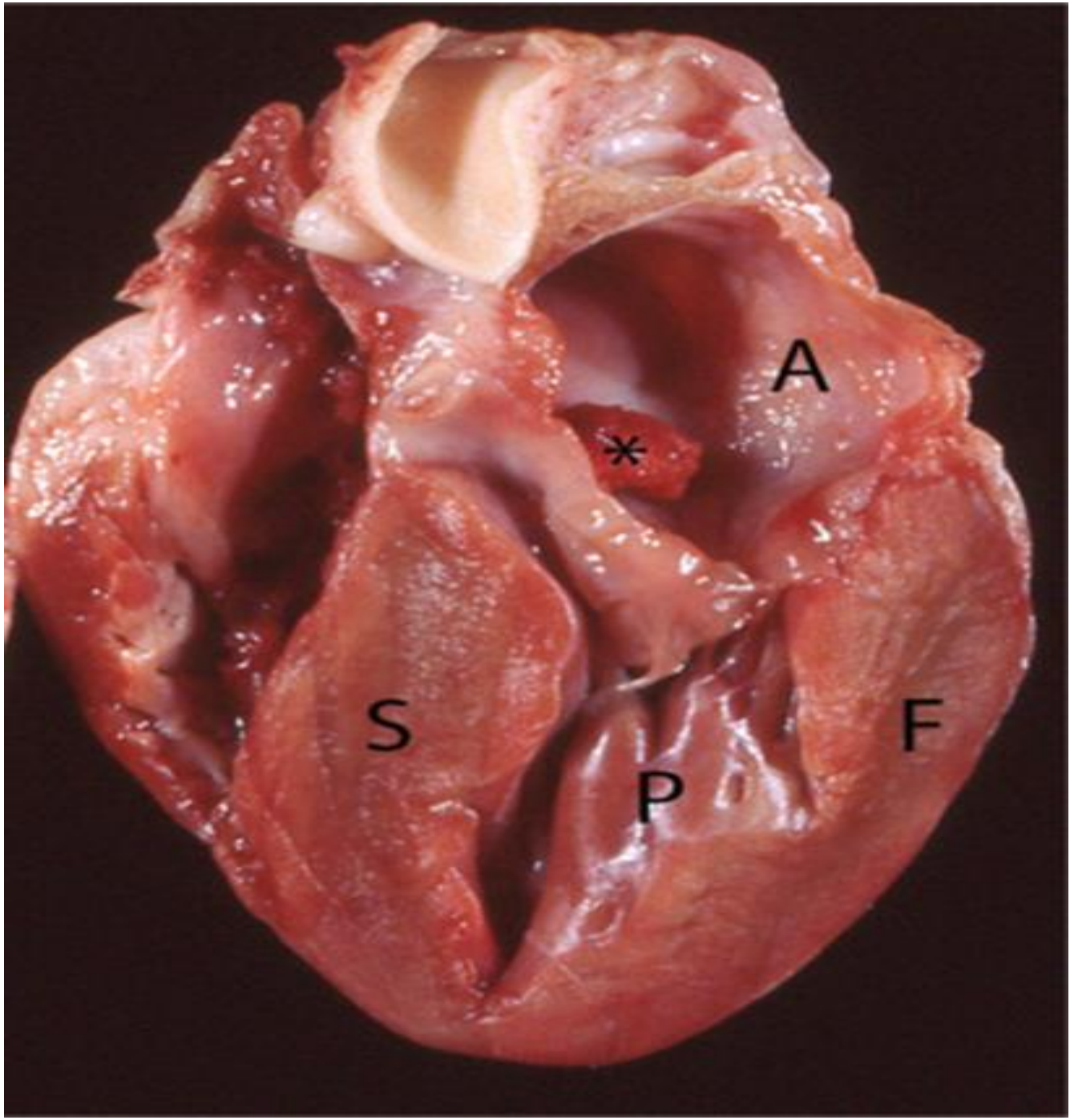
❑ ***Diastolic dysfunction:***

- Diastolic dysfunction is one of the most visible and early hemodynamic alterations during HCM.
- It is characterized by a slowing of relaxation and increased stiffness of the left ventricle.
- The result is the reduction: of the systolic volume, the chamber size, and left ventricular compliance.
- The left atrial volume, an indirect indicator of left ventricular diastolic pressure, is often increased (dilatation) in patients with HCM, heralding the development of heart failure.

Post-mortem specimen of a 4-year-old male Siamese cat with hypertrophic cardiomyopathy. Longitudinal section showing severe concentric hypertrophy of the interventricular septum and the free wall of the left ventricle and a very reduced left ventricle chamber. LA, left atrium.



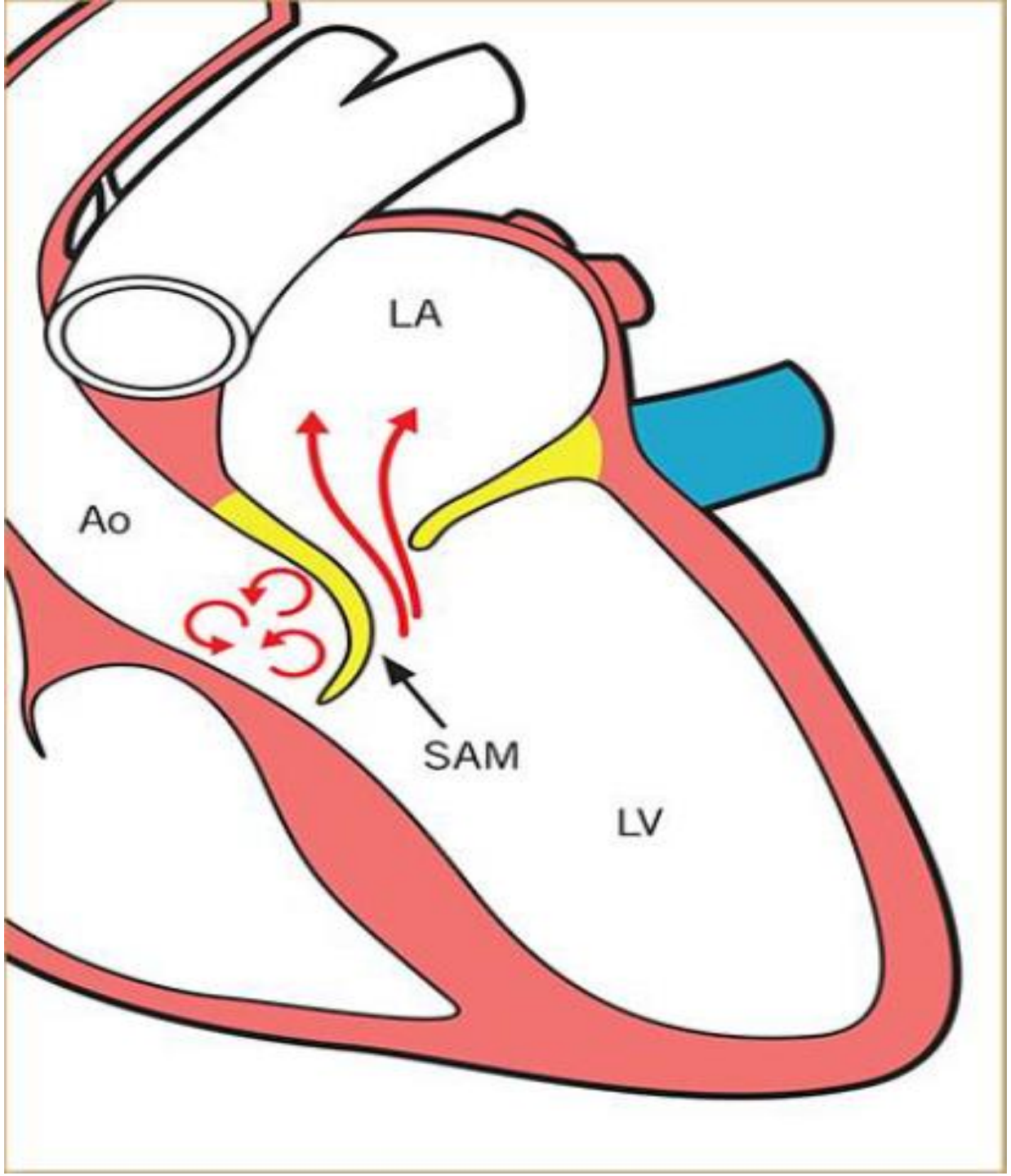
A heart affected by HCM showing markedly thickened left ventricular walls and papillary muscles and an enlarged left atrium. A thrombus is present in the body of the left atrium (black asterisk). S = interventricular septum; F = free wall of the left ventricle; P = base of the papillary muscles; A = body of the left atrium.



❑***Obstruction of the left ventricular outflow tract (LVOT):***

- Dynamic LVOT obstruction is very common in cats with HCM (67%).
- It results from the systolic anterior motion (SAM) of the anterior leaflet of the mitral valve towards the interventricular septum, leading to an increase in pressure in the left ventricular chamber and a high flow velocity through the LVOT.
- A high velocity through the LVOT can further exacerbate the SAM and lead to the development of mitral regurgitation and may contribute to increased left atrial pressure.

Diagram of left ventricular outflow tract (LVOT) obstruction resulting from the systolic anterior motion (SAM) of the anterior leaflet of the mitral valve (arrow). LA, left atrium; LV, left ventricle; Ao, aorta.



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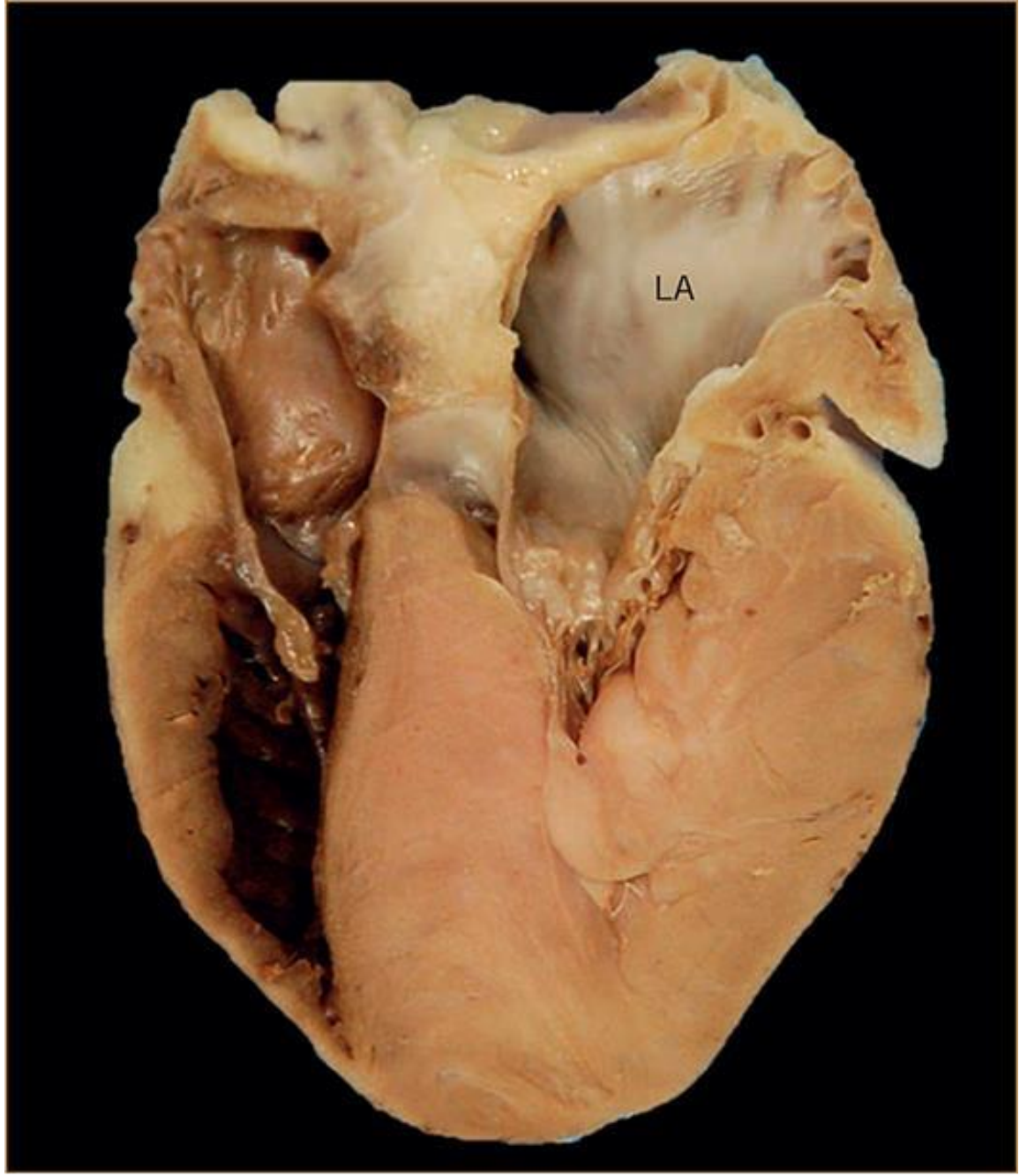
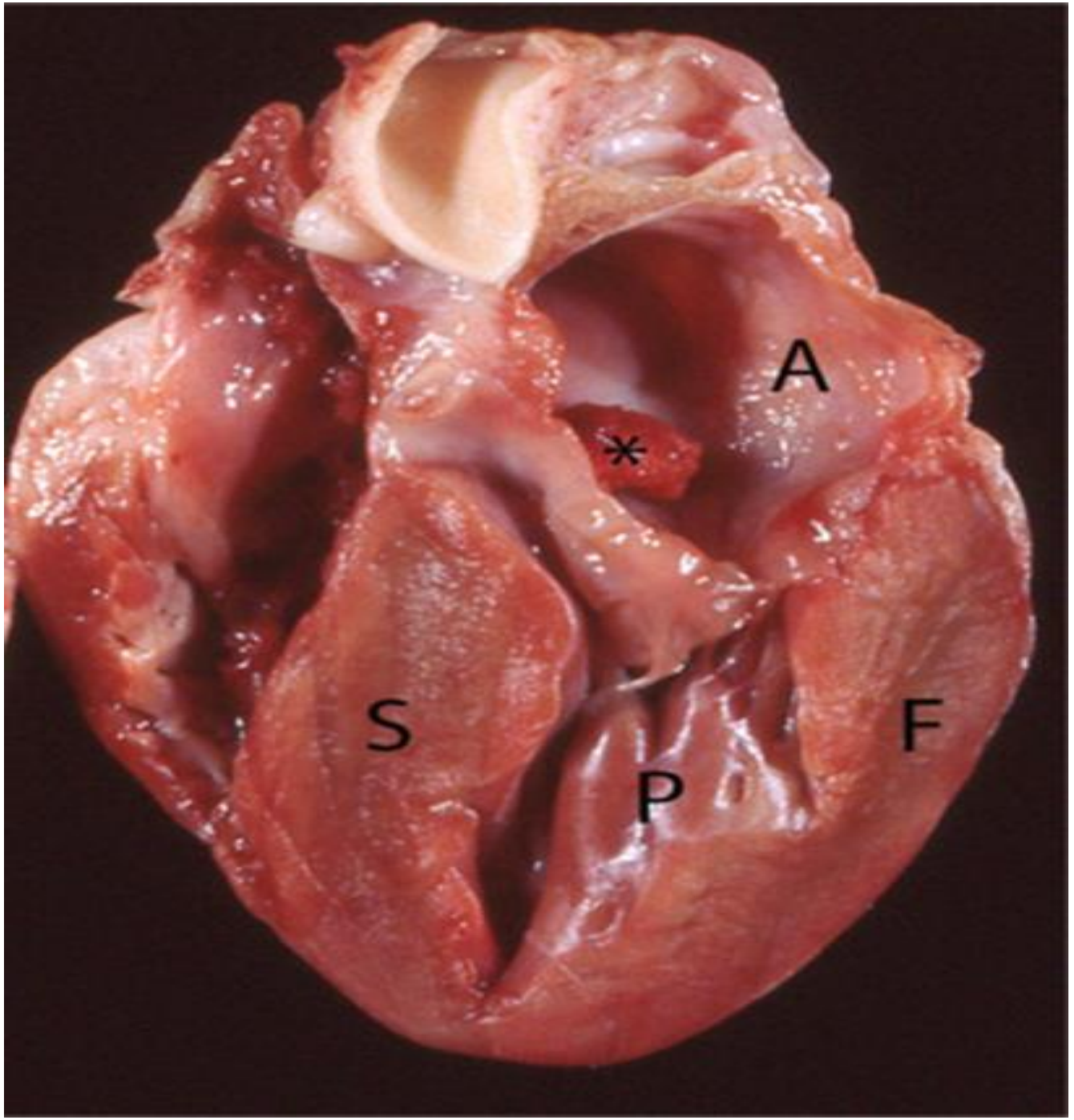
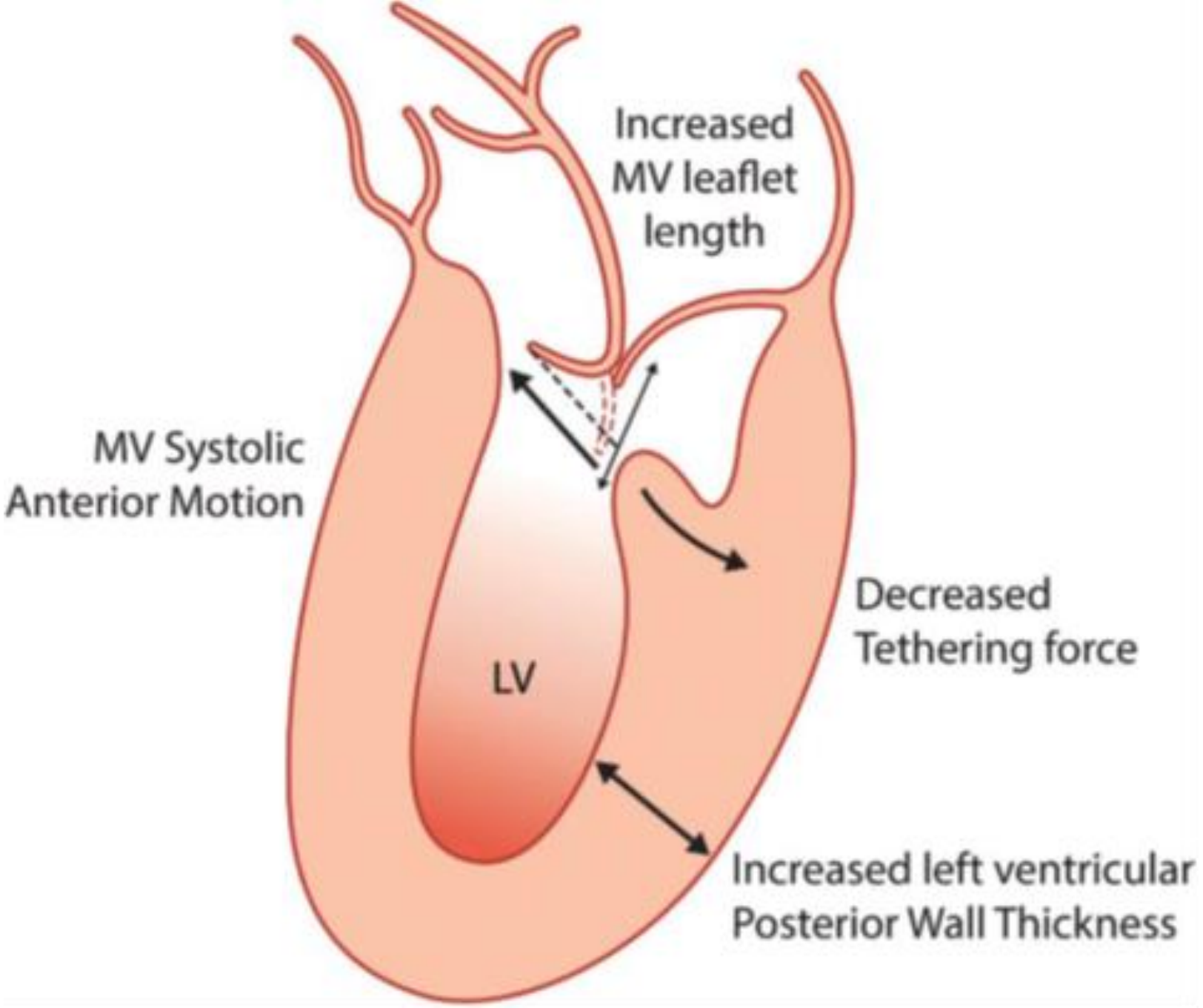


A heart affected by HCM showing significantly thickened left ventricular walls and papillary muscles, with an enlarged left atrium. A thrombus is present in the body of the left atrium (black asterisk). S = interventricular septum; F = free wall of the left ventricle; P = base of the papillary muscles; A = body of the left atrium.



- It is now believed that the changes occurring in the architecture of the hypertrophied left ventricle (mainly the papillary muscles) can displace the chordae tendineae and the anterior leaflet of the mitral valve into the narrowed LVOT and contribute to the obstruction of systolic flow.
- The obstruction of the LVOT increases the afterload, causing further hypertrophy that reduces the size of the ventricular chamber and is responsible for the elevation of left ventricular and atrial filling pressures and the reduction of coronary perfusion (major imbalance between oxygen supply and demand), thereby inducing ischemia and myocardial fibrosis.

Diagram of SAM. When the mitral valve leaflet is sucked back into the left ventricular outflow tract (LVOT), this can cause a heart murmur. However, a localized area of hypertrophy in the same place would also cause an obstruction to blood flow and cause a heart murmur.



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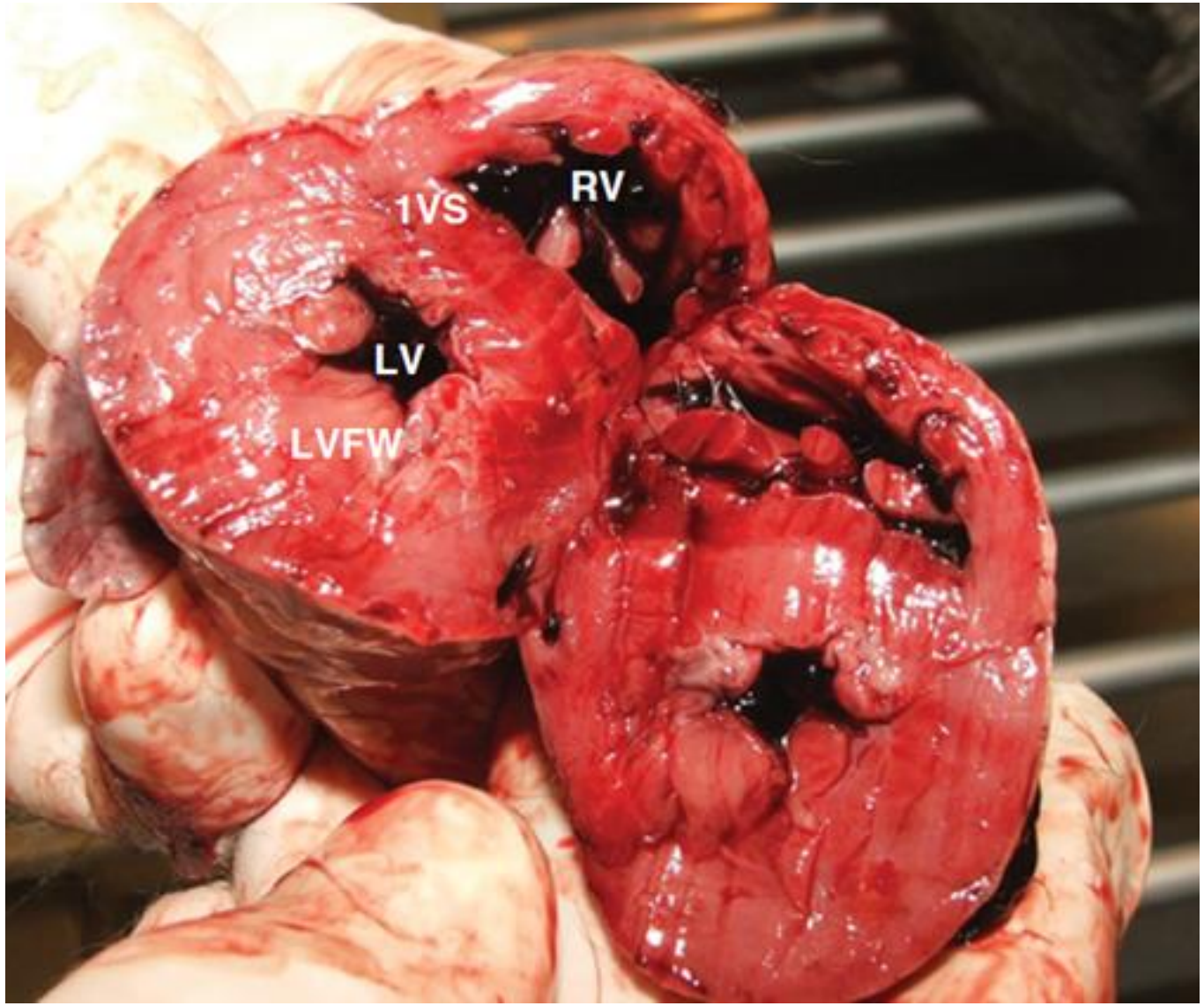


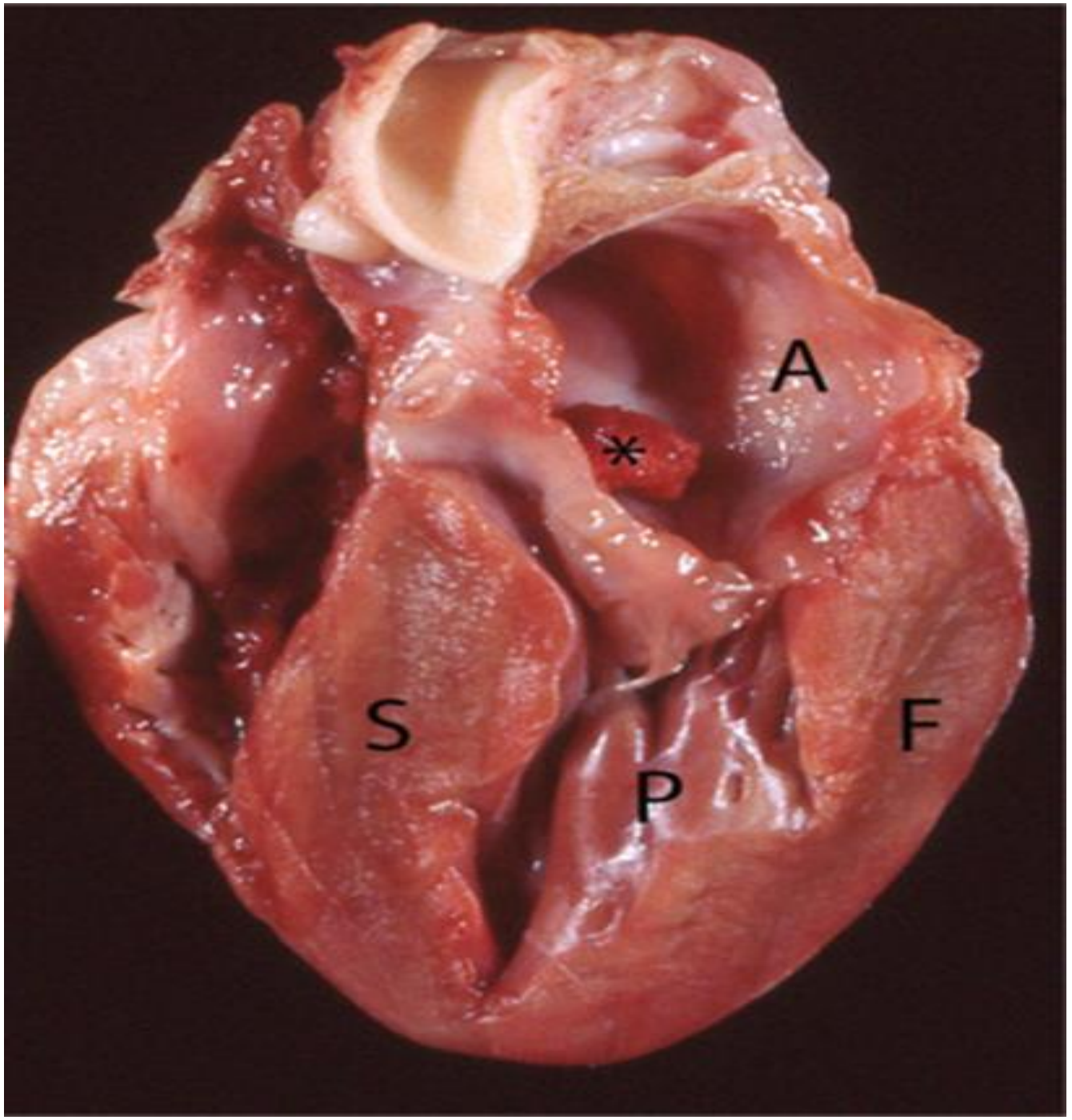
❑***Complications = ICC + pulmonary edema + pleural effusion:***

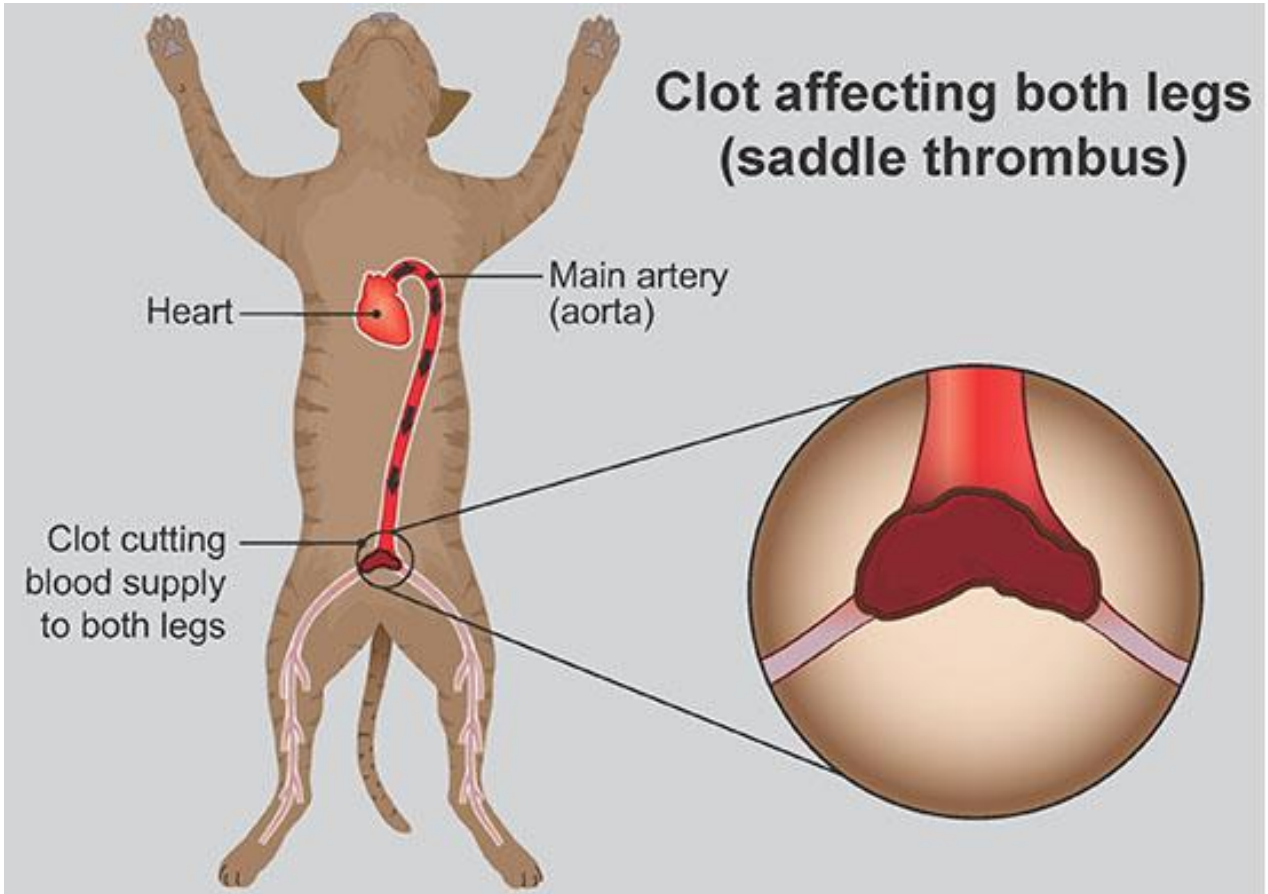
- Congestive heart failure (ICC), diastolic dysfunction, LVOT obstruction, and increased atrial pressures are responsible for pulmonary edema and pleural effusion.

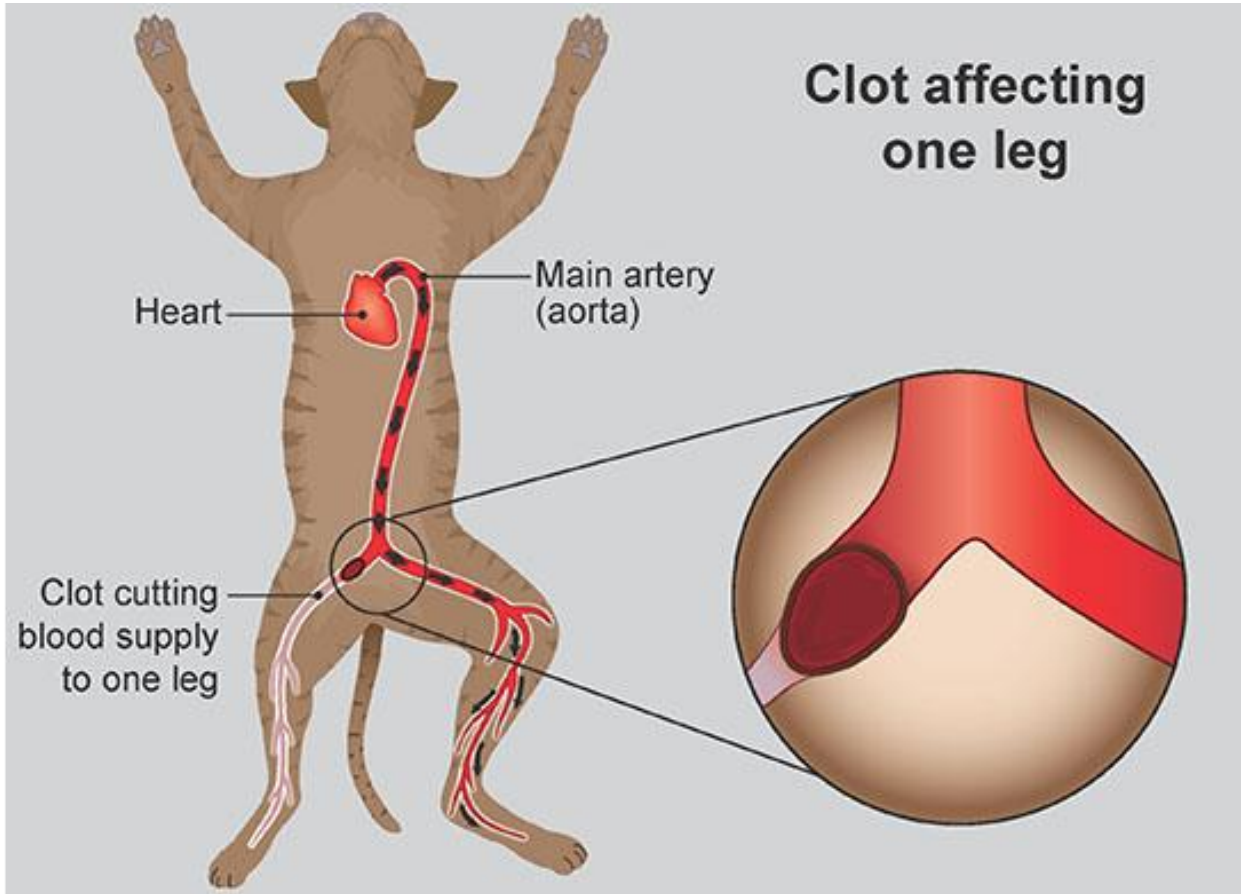
❑***Complication = Arterial or aortic thromboembolism (TEA; Aortic thromboembolism [ATE]:***

- TEA can occur in some cats affected by HCM (12 to 17%) associated with left atrial dilation.
- Although the distal aorta (saddle embolism) is the most common site (71%) of cardiogenic embolism, other arteries such as the right brachial, renal, mesenteric, coronary, and cerebral arteries may also be embolized.

A heart affected by HCM showing significantly thickened left ventricular walls and papillary muscles, and an enlarged left atrium. A thrombus is present in the body of the left atrium (black asterisk). S = interventricular septum; F = free wall of the left ventricle; P = base of the papillary muscles; A = body of the left atrium

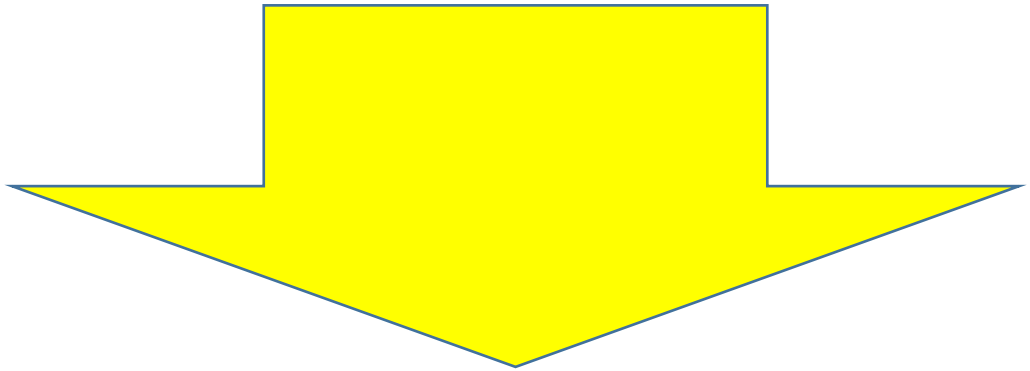



Clot affecting both legs
(saddle thrombus)
Heart
Main artery
(aorta)
Clot cutting
blood supply
to both legs


Clot affecting
one leg
Heart
Main artery
(aorta)
Clot cutting
blood supply
to one leg

Cat with an ATE affecting one limb.

- The staging classification of feline HCM described in the table below has been adopted by the American College of Veterinary Internal Medicine.



Stade	Description
A	Chats qui risquent de développer une cardiomyopathie ou qui y sont prédisposés, mais qui ne présentent actuellement aucun signe de maladie. Cela peut être lié à la race ou aux antécédents familiaux.
B	Les chats de ce groupe sont diagnostiqués avec une CMH (épaississement accrue de la paroi ventriculaire gauche) mais ne présentent aucun signe clinique. Ce groupe est divisé en deux catégories distinctes : **B1 –** Chats présentant un faible risque de développer une insuffisance cardiaque congestive (ICC) ou une thrombo-embolie artérielle (ATE). C'est parce qu'ils n'ont pas de dilatation auriculaire gauche (Cependant, ils peuvent manifester une faible dilatation de l'oreillette gauche). **B2 –** Chats présentant un risque plus élevé de développer une ICC ou une ATE de manière imminente. Cela s'explique par la présence de la dilatation auriculaire gauche modérée à sévère.
C	Chats qui présentent actuellement ou ont présenté des signes d'ICC ou d'ATE.
D	Chats devenus réfractaires au traitement conventionnel de l'ICC.

Clinical presentation

- Most cats with cardiomyopathy are diagnosed at an average age of 5 to 7 years (between 3 months and 18 years).
- The average age of cats with HCM is around 6 years, with an age range from 8 months to 16 years.
- Most cats with HCM are asymptomatic and are recognized when a heart murmur or a gallop sound is discovered during a routine examination.
- Some asymptomatic cats show no abnormalities upon auscultation.

- Various studies have shown that nearly 50% of apparently healthy cats (showing no signs of heart disease) present with heart murmurs; however, the presence of a heart murmur warrants further evaluation.
- In one study, 70% of cats with HCM showed no auscultatory abnormalities, so the absence of these signs does not exclude the presence of heart disease.
- Cats with occult cardiomyopathy (RCM, DCM, CMA, unclassified cardiomyopathies) are often not identified until clinical signs are present, including dyspnea, tachypnea, lethargy, lack of appetite, hind limb paresis, collapse, or sudden death.

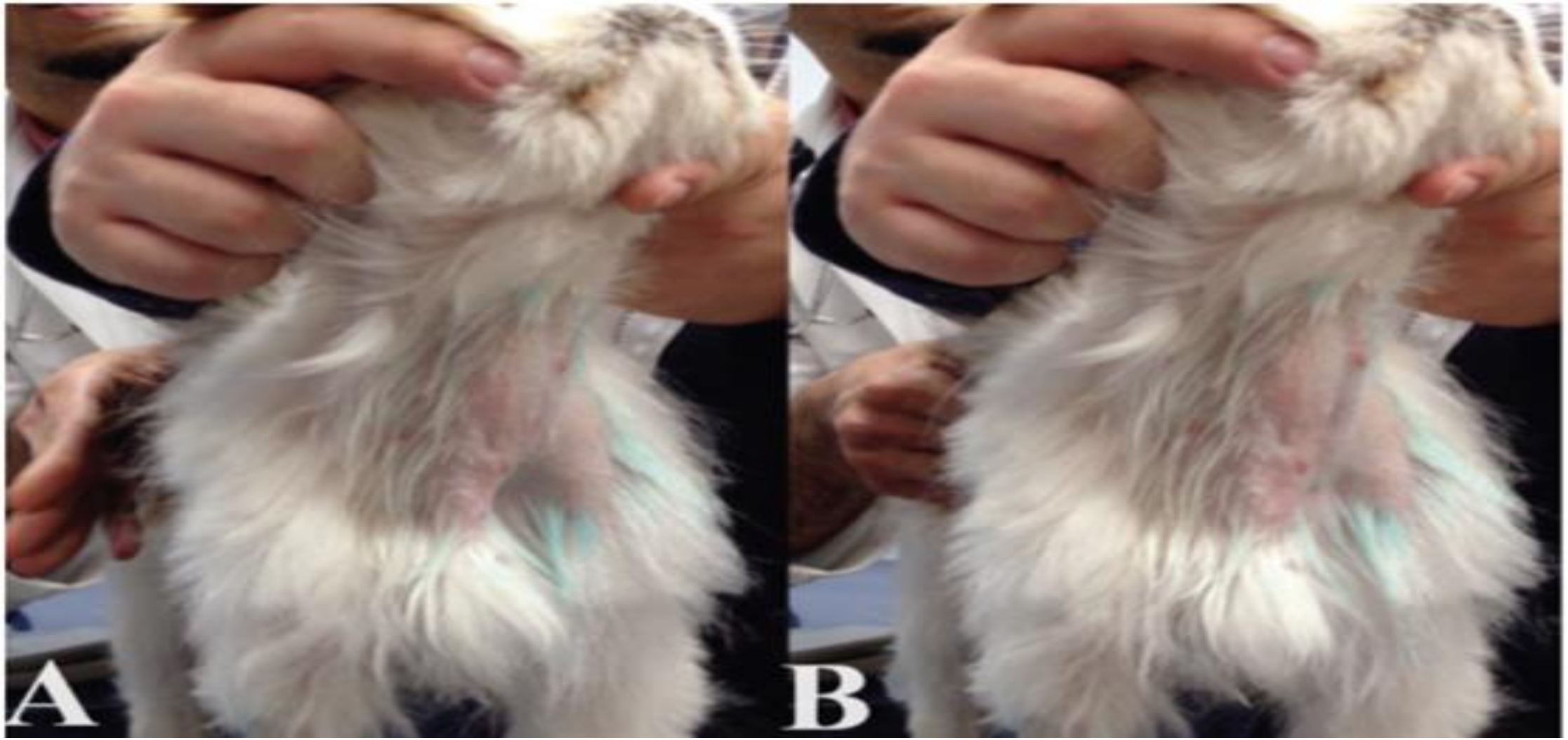
- There are no characteristic combinations of clinical signs of HCM, and cats may present with any combination of signs listed in the table below:

Signes cliniques	Caractéristiques possibles
Anomalies de l'auscultation cardiaque	• Souffle cardiaque : Variable, jusqu'au grade IV avec un PMI (point of maximal intensity) sur MV/LVOT (mitral valve/ left ventricular outflow tract). • Bruit de galop : Généralement B4 (S4). • Trouble du rythme : arythmie, pauses, bradycardie ou tachycardie • Bruits cardiaques étouffés ou faibles
Anomalies du pouls	• Pouls artériel hypokinétique, hyperkinétique, variable ou alternant. • Pouls veineux jugulaire proéminent. • Distension veineuse jugulaire.

Signes cliniques	Caractéristiques possibles
Thromboembolie artérielle	• Apparition soudaine d'une parésie ou d'une boiterie (membres postérieurs ou antérieurs). • Signes d'ischémie des membres (membre sans pouls, froid, pâle). • Pouls diminué ou absent (ou des signaux de flux Doppler). • Fermeté ou contraction des muscles squelettiques avec douleur musculaire. • Neuropathie des motoneurones inférieurs
Signes respiratoires	• Tachypnée. • Détresse respiratoire. • Toux (moins fréquent). • Bruits pulmonaires adventifs (Pulmonary adventious sounds). • Bruits bronchiques altérés (Altered bronchial sounds). • Crépitements pulmonaires (pulmonary crackles) • Signes d'épanchement pleural.

Signes cliniques	Caractéristiques possibles
Mort subite (cardiaque)	• Avec ou sans signes prémonitoires.
Signes du système nerveux central	• Dépression. • Syncope. • Signes d'accident vasculaire cérébral hémorragique ou thrombotique (altération de la conscience, convulsions, altération de la posture associée à une lésion cérébrale).

Signes cliniques	Caractéristiques possibles
Signes oculaires	• Dégénérescence rétinienne centrale (carence en taurine) • Hémorragie rétinienne, décollement, tortuosité vasculaire, cécité (liée à l'hypertension).
Autres signes potentiels	• Antécédents d'inactivité. • Pâleur ou cyanose des muqueuses. • Hypertrophie de la thyroïde. • Taille et/ou contour rénal anormal. • Perte de poids. • Hypothermie. • Apparence faciale anormale (acromégalie).



Hepato-jugular reflux sign. A/Jugular veins before applying abdominal pressure; B/A marked distension of the jugular veins is noted when pressure is applied to the cranial abdomen.

Cat with acute hind limb paresis due to aortic thromboembolism: The thrombus having a 'saddle' appearance (Saddle thrombus) resulting from a left atrial thrombus caused by hypertrophic cardiomyopathy.

- The differential diagnosis includes cardiomyopathies secondary to systemic disease (hypertension, hyperthyroidism, hypersomatotropism, or acromegaly), degenerative valvulopathy, certain congenital heart diseases (e.g., aortic stenosis), and myocarditis.
- Differential diagnoses for respiratory signs include feline lower respiratory tract disease (feline asthma), pneumonias, non-cardiogenic pleural effusion, non-cardiogenic pulmonary edema, and pulmonary hemorrhage.

Diagnosis

Blood pressure

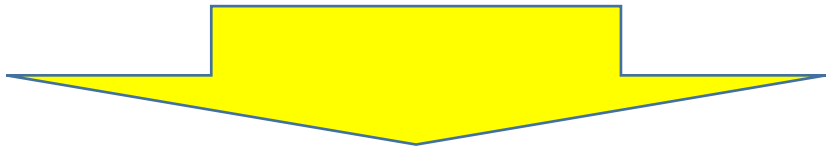
- Systemic blood pressure is generally normal in cats with cardiomyopathy, unless the cause of the myocardial disease is systemic hypertension.
- The cat with CHF or arterial thromboembolism may be hypotensive and may present with hypothermia and reduced peripheral perfusion.
- Cardiogenic shock is not specific to a particular form of feline cardiomyopathy, but often suggests that an acute event such as a regional wall infarction or a thromboembolic event has occurred.

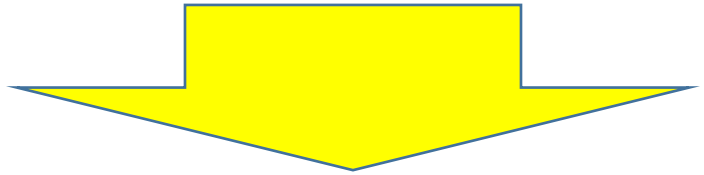
Laboratory tests

A serum biochemical profile may reveal abnormalities related to heart failure, thromboembolism, recent treatment, or an underlying systemic disease.

Biomarkers, particularly NT-proBNP and troponin I (cTnI), may be released by affected ventricles:

- The sensitivity of a high NT-proBNP value in diagnosing subclinical HCM can be as high as 94% or more, although its value as a screening test is still uncertain.
- It has been shown that NT-proBNP can differentiate cats with respiratory signs due to congestive heart failure from cats with primary respiratory disease.



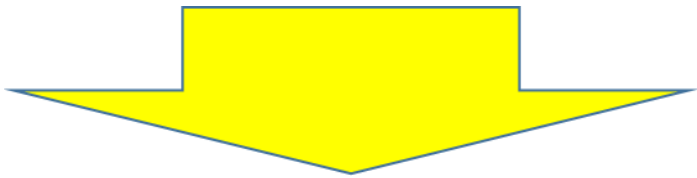


- An elevated NT-proBNP warrants further diagnostic evaluation.
- A normal NT-proBNP value should not be used to rule out heart disease if other results are favorable.
- Troponin I can also be elevated in cats with moderate to severe hypertrophic cardiomyopathy with or without congestive heart failure.
- Serum thyroxine should be measured in older cats (> 7 years) showing cardiac signs, to rule out hyperthyroidism as a potential cause of hypertrophic cardiomyopathy.

Echocardiography

Echocardiography is an excellent non-invasive examination that allows for the assessment of morphological and functional abnormalities of cardiac structures:

- The diagnosis of hypertrophic cardiomyopathy relies on identifying diastolic wall thickness hypertrophy (of the left ventricle) > 6 mm on two-dimensional echocardiography or M-mode.
- Hypertrophy can be diffuse or segmental and may be symmetrical or asymmetrical.



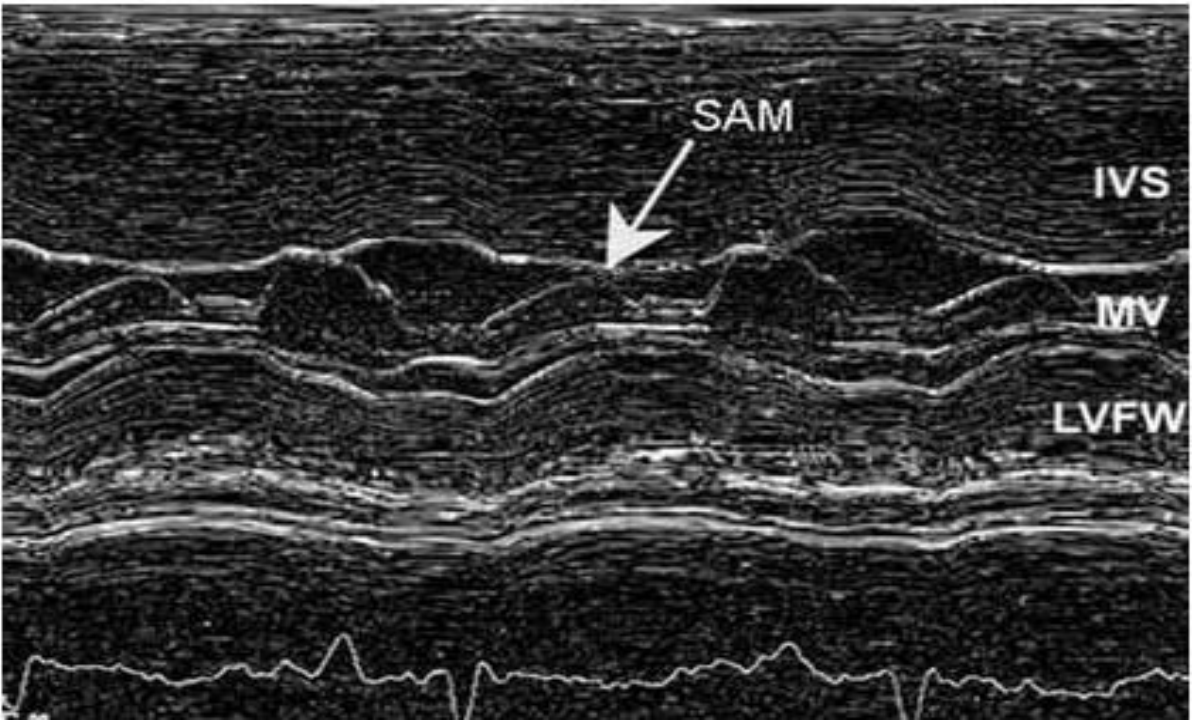
- Asymmetric hypertrophy of the interventricular septum (interventricular septum [IVS]) causing left ventricular outflow tract (LVOT) obstruction.
- Hypertrophied papillary muscle.

- Decreased internal dimensions of the left ventricle (obliteration of the telesystolic cavity).
- Fraction of shortening = FR (Fractional shortening [FS]) is normal to increased.
- The FR may fall below the reference range in advanced diseases.

- Left atrial dilation (identified with a left atrial dimension > 16 mm).
- The dilated left atrium may contain thrombus.

- Mild pericardial effusion.
- Mild hypertrophy of the right ventricle. It should be noted that HCM rarely affects the right ventricle.
- Mitral insufficiency (regurgitation) > 90% of cases.

- SAM of the mitral valve (anterior systolic movement of the mitral valve): In M-mode, the mitral valve moves upward towards the interventricular septum during systole.



SAM with obstruction of the LVOT

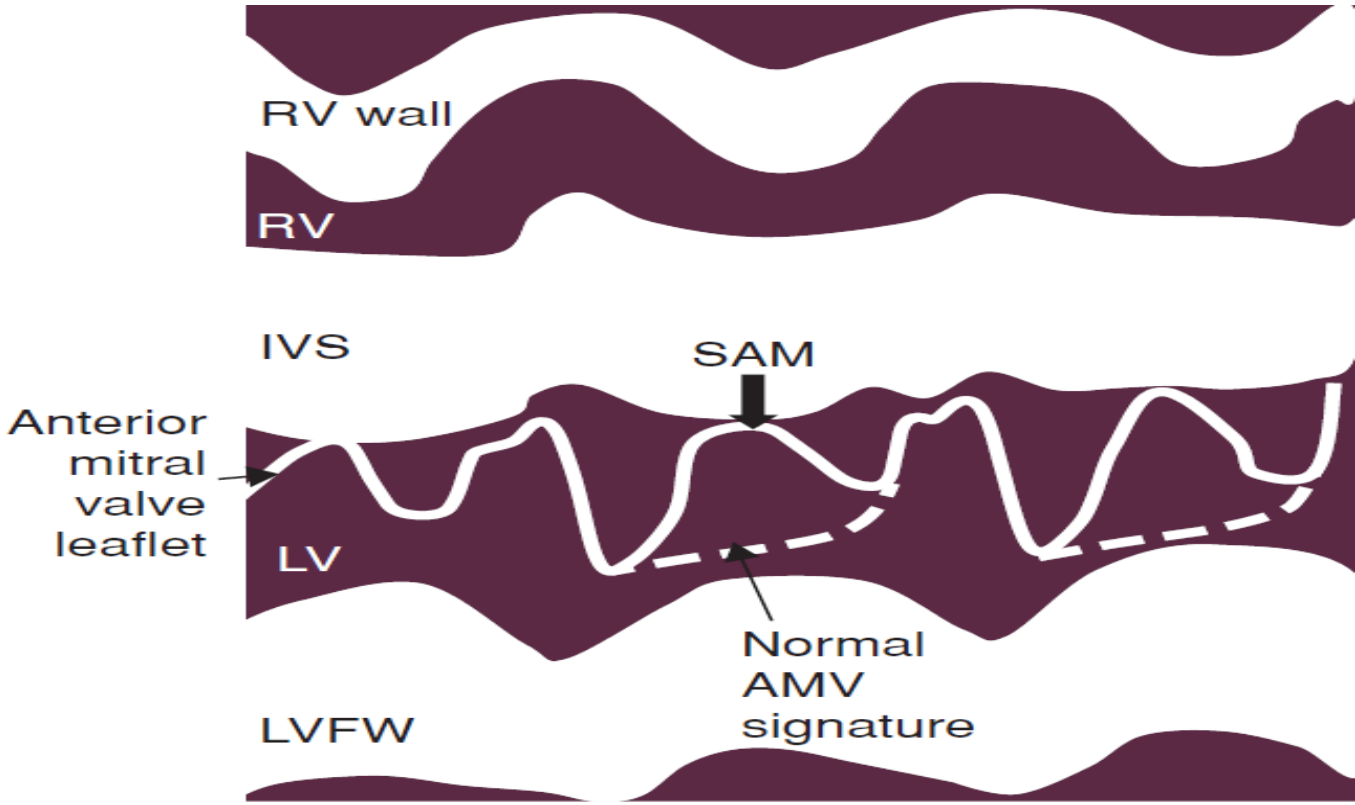
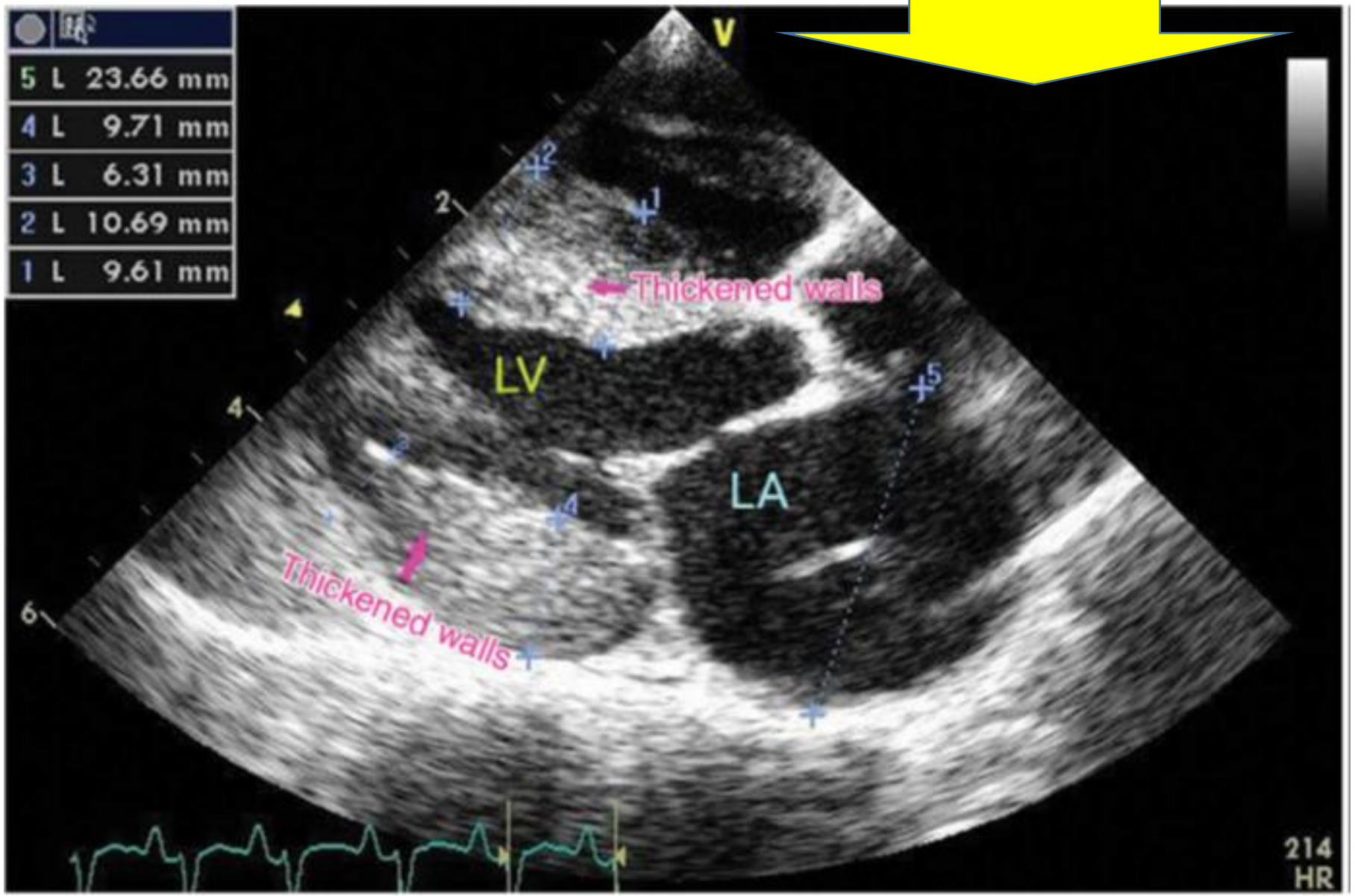
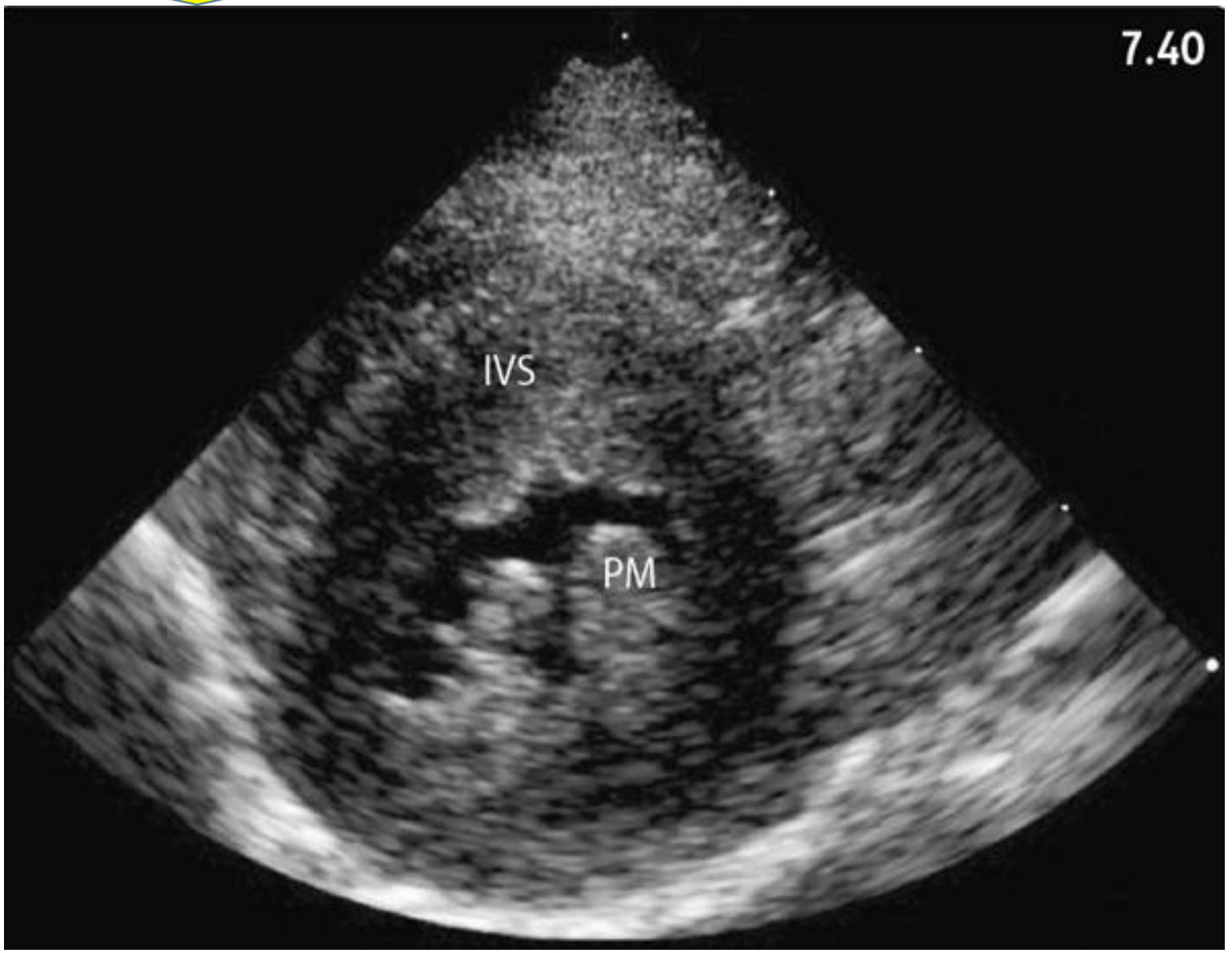


Diagram illustrating SAM on M-mode echocardiography

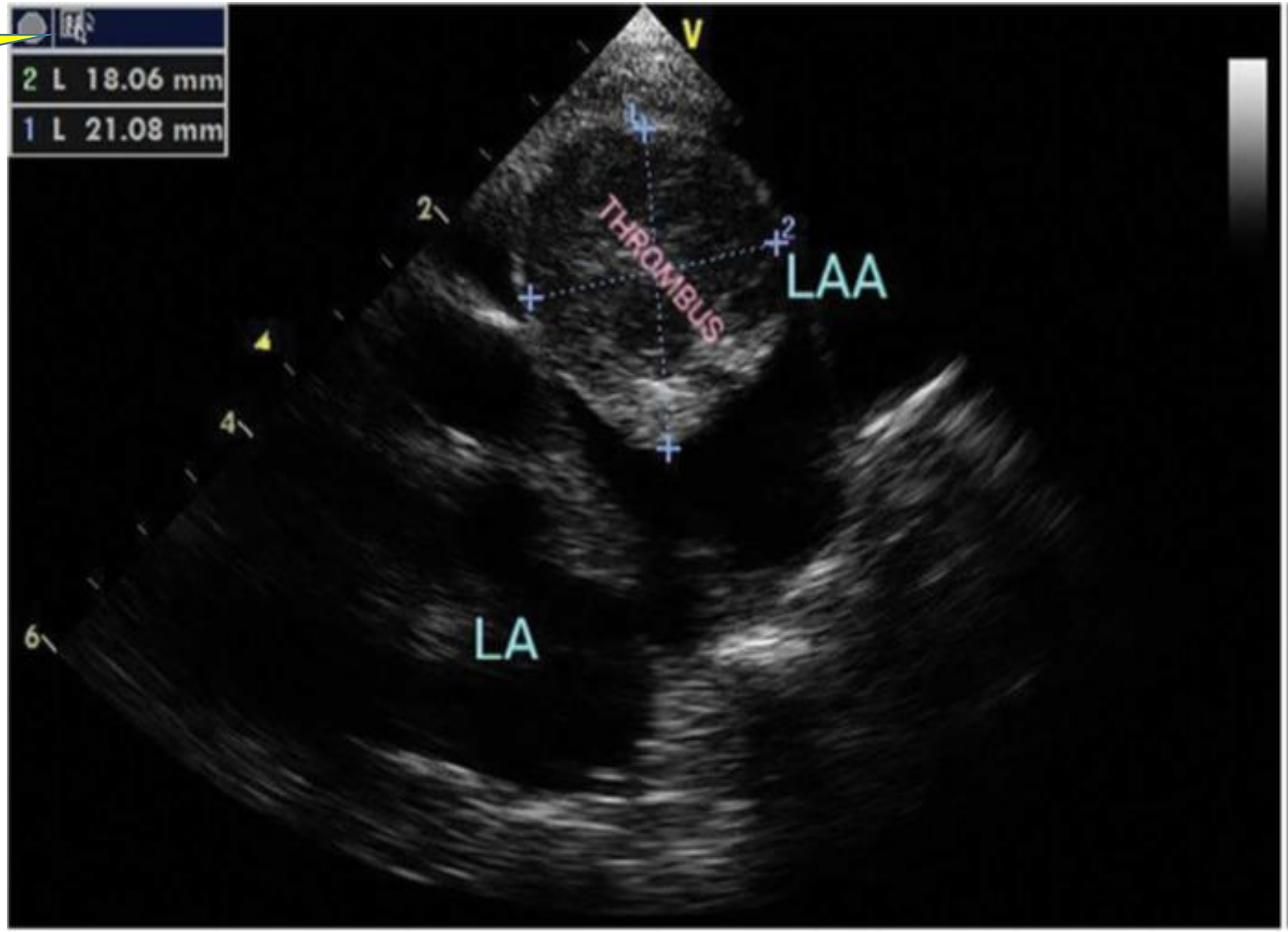


HCM on echocardiography, in right parasternal axis view. The left atrium (LA) is large and the ventricular walls are very thick.

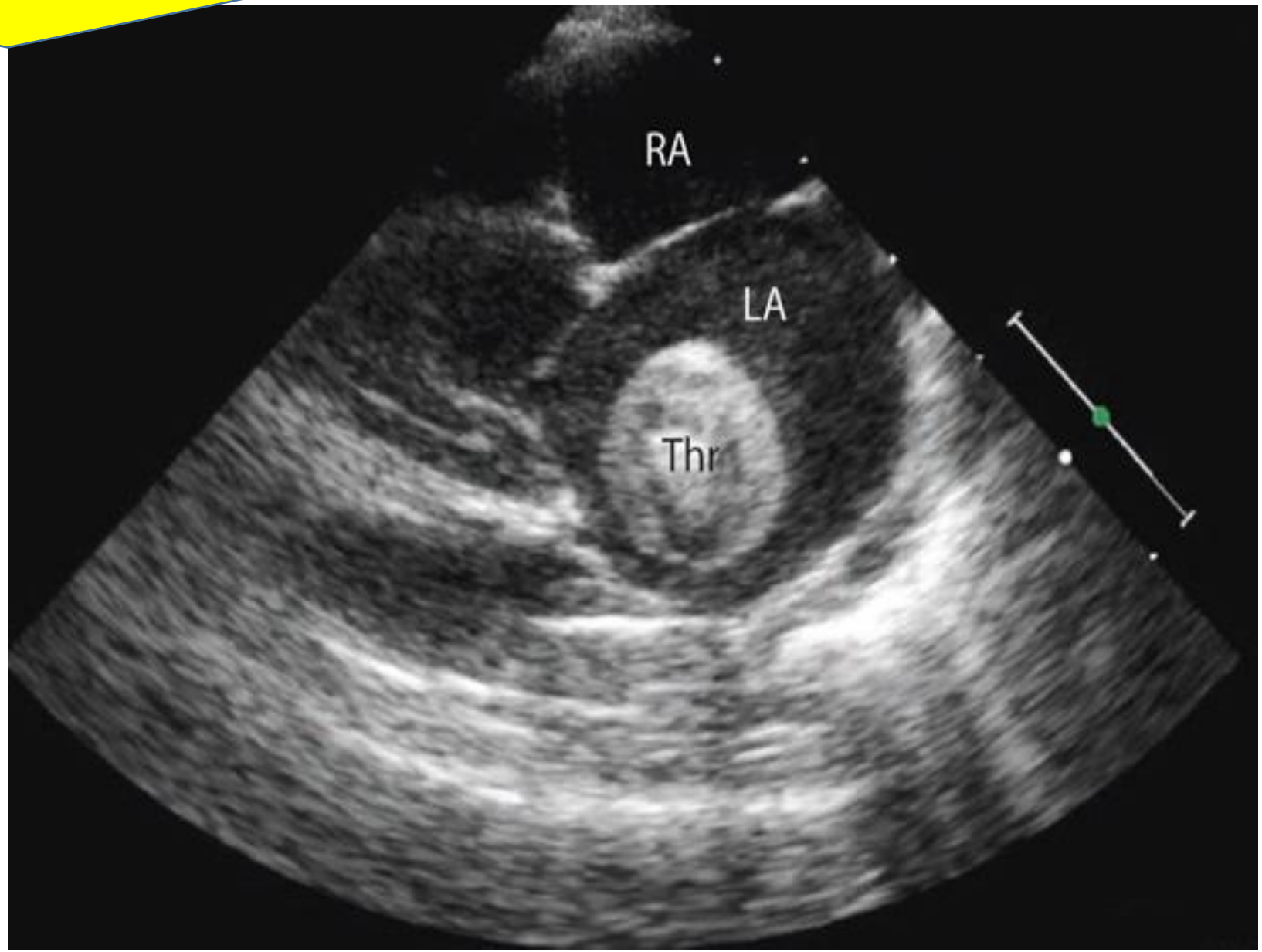
Right parasternal echocardiographic view in short axis of a cat with HCM. Concentric hypertrophy of the interventricular septum and the free wall of the left ventricle is observed. The papillary muscles are prominent. IVS, interventricular septum; PM, papillary muscle.



Echocardiographic image of a thrombus in the left atrium (LA). This is due to significant left atrial dilation and lack of blood flow. LAA = left atrial appendage. LA = left atrium.



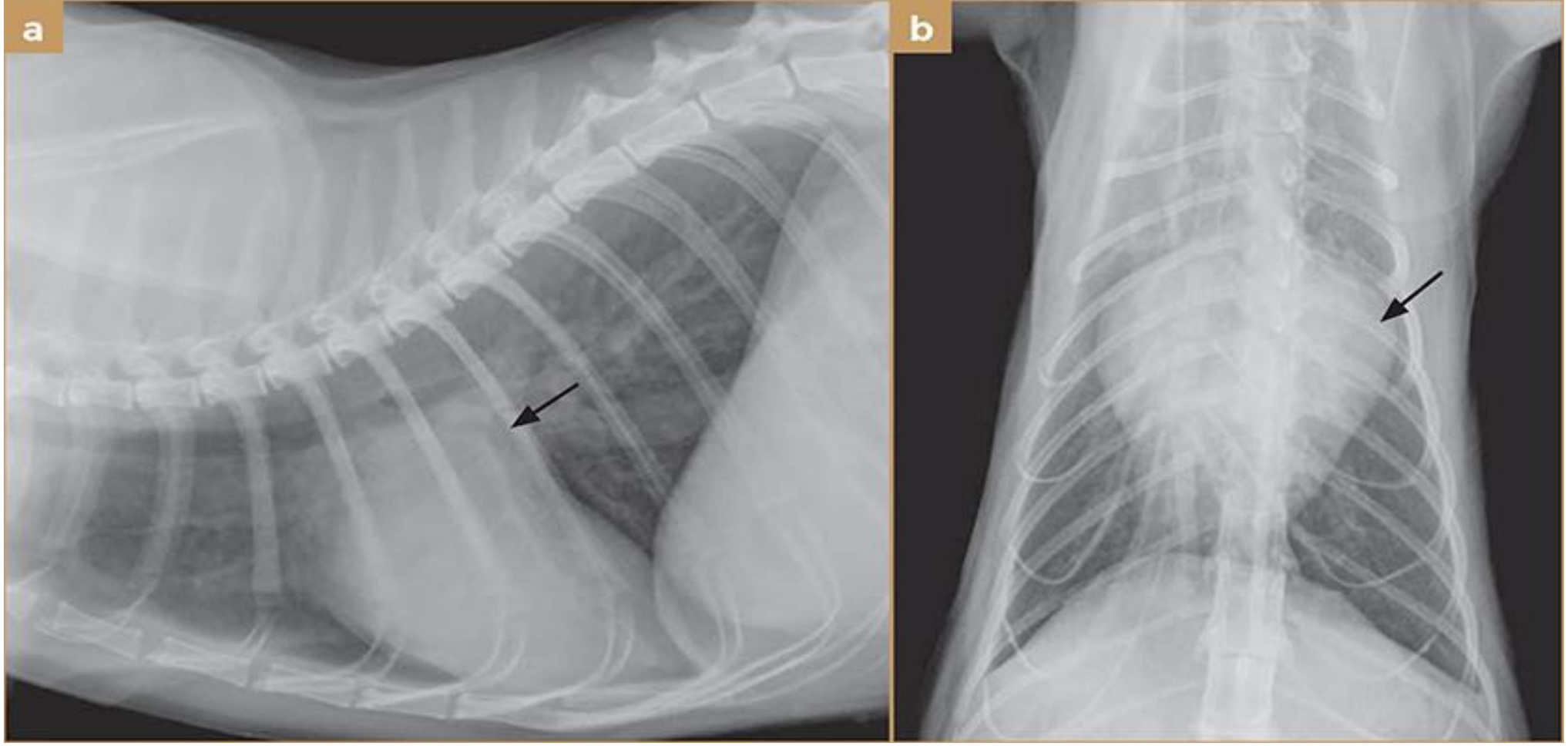
Right parasternal echocardiographic view of a cat showing a large thrombus in the left atrium. LA, left atrium; RA, right atrium; Thr, thrombus.



Thoracic X-ray

Chest X-rays may help identify the following abnormalities:

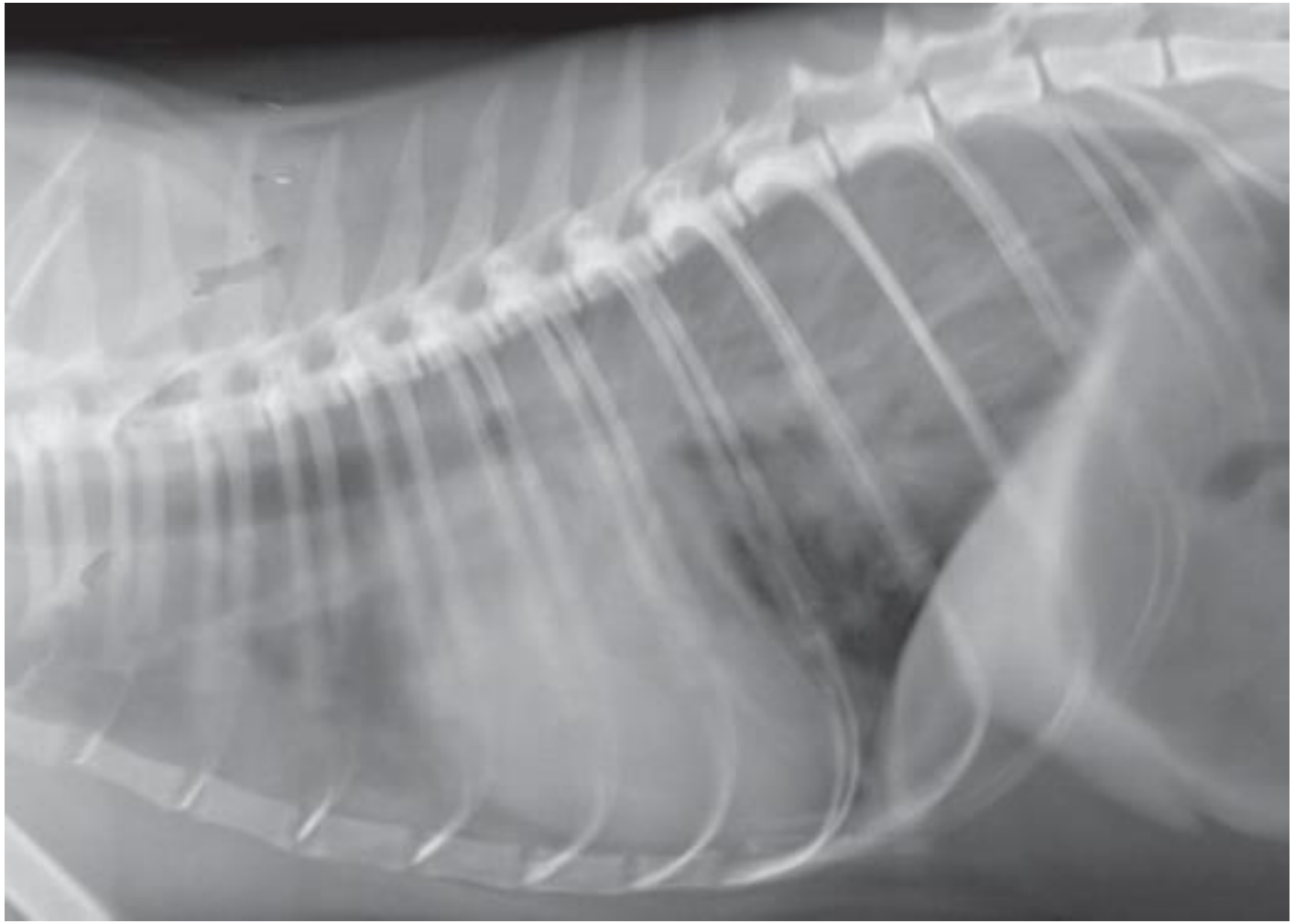
- Cardiomegaly, although it may be subtle, especially in the absence of atrial hypertrophy or if the main anomaly is concentric ventricular hypertrophy.
- Cardiomegaly, often characterized by a long cardiac silhouette on the lateral view.
- A generalized (rounded) enlargement of the cardiac silhouette is generally associated with pericardial effusion.
- The classic 'Valentine Heart' shape on the dorsoventral view results from severe left atrial dilation or (severe biatrial dilation).



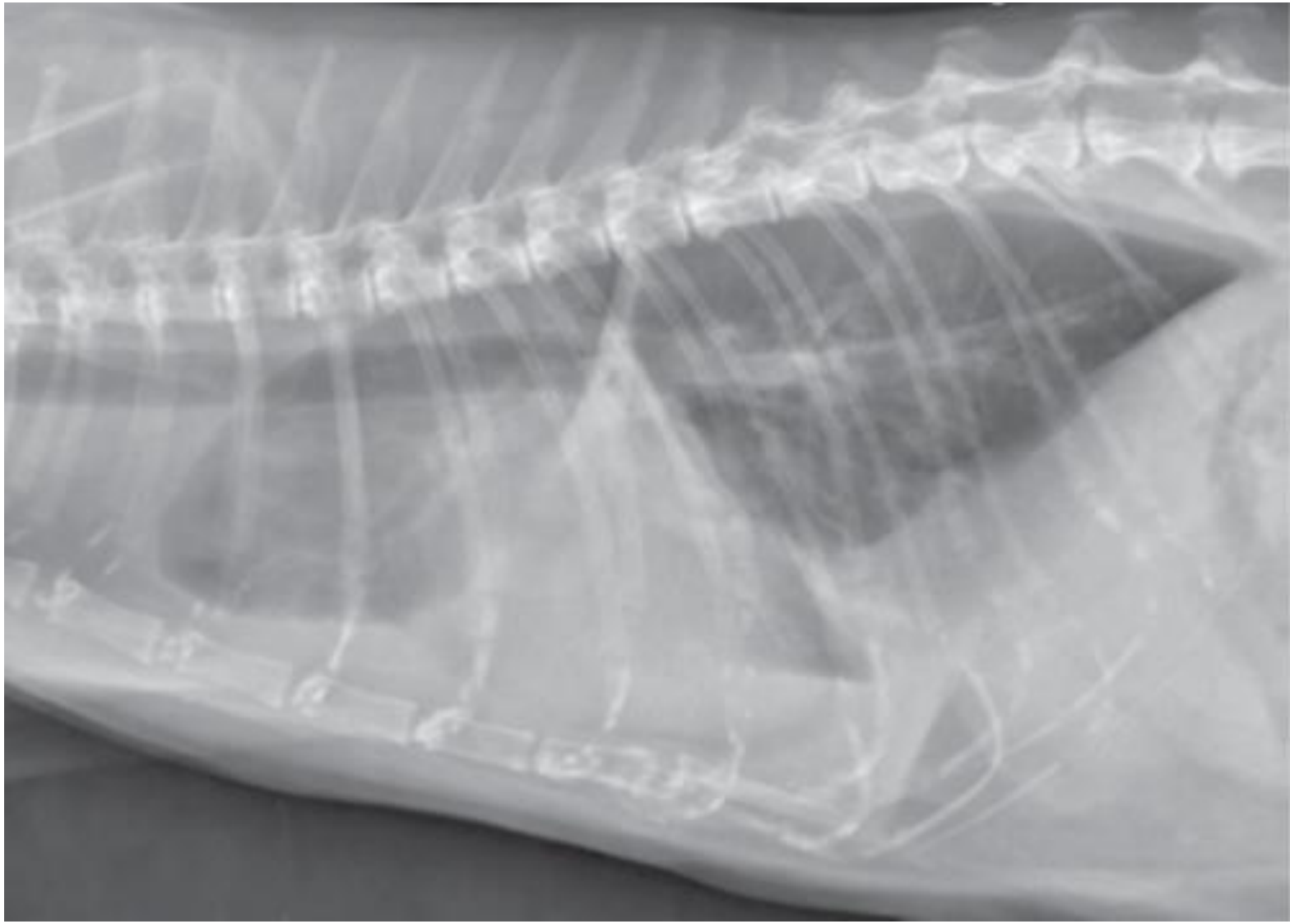
a) and b) Thoracic radiographs of a cat with severe hypertrophic cardiomyopathy and moderate congestive heart failure. Pulmonary venous distension, diffuse interstitial edema, left atrial enlargement (arrows). b) The classic 'Valentine' heart shape due to left atrial dilation can be seen on the dorsoventral view.

- Pulmonary venous congestion, an irregular or diffuse interstitial-alveolar pattern, and/or pleural effusion are present in cats with heart failure.

Radiograph of a cat with HCM = pulmonary edema + cardiomegaly.
Aerophagia is also present.



Radiograph of a cat with HCM. The thoracic radiograph suggests an enlarged cardiac silhouette, difficult to discern due to the effusion. Pleural effusion is present, indicated by the retraction of the pulmonary lobes from the body wall and blunting of the costophrenic angles. A diffuse interstitial to alveolar pattern is present.



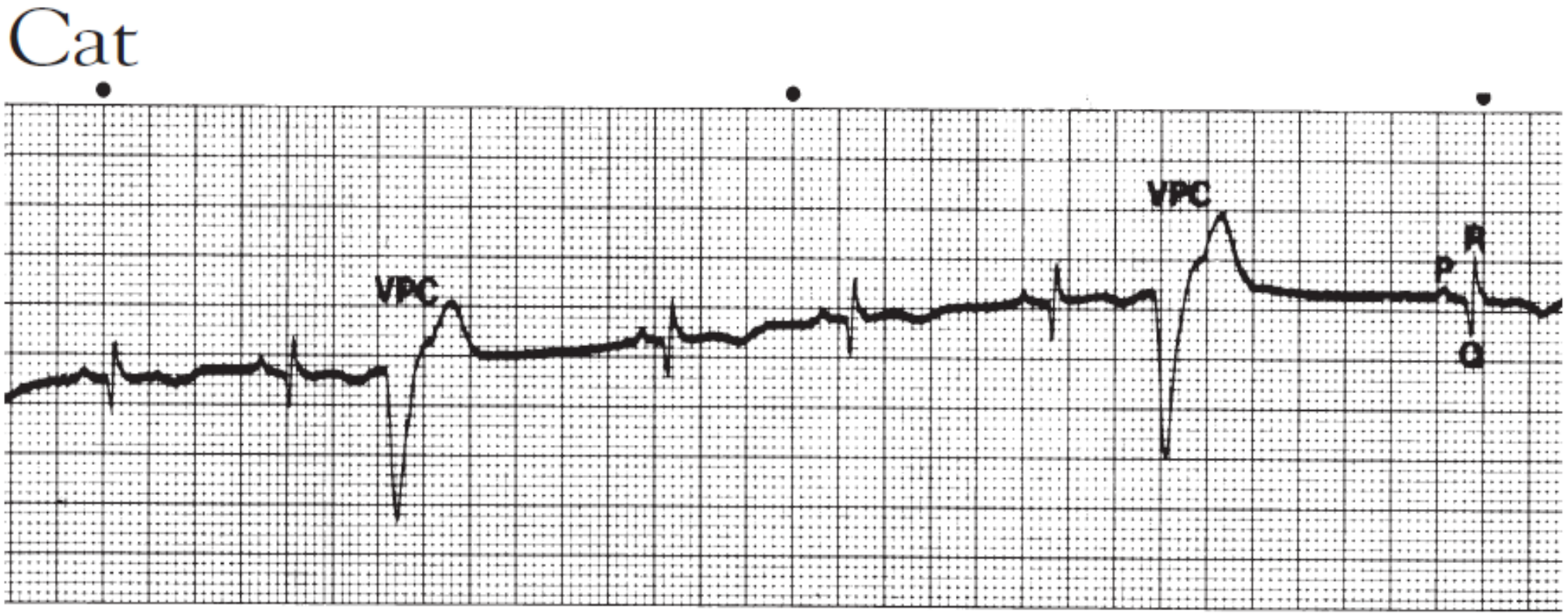
Electrocardiography (ECG)

ECG is highly variable in cats with HCM.

However, this is the most specific examination to evaluate arrhythmias in patients with HCM:

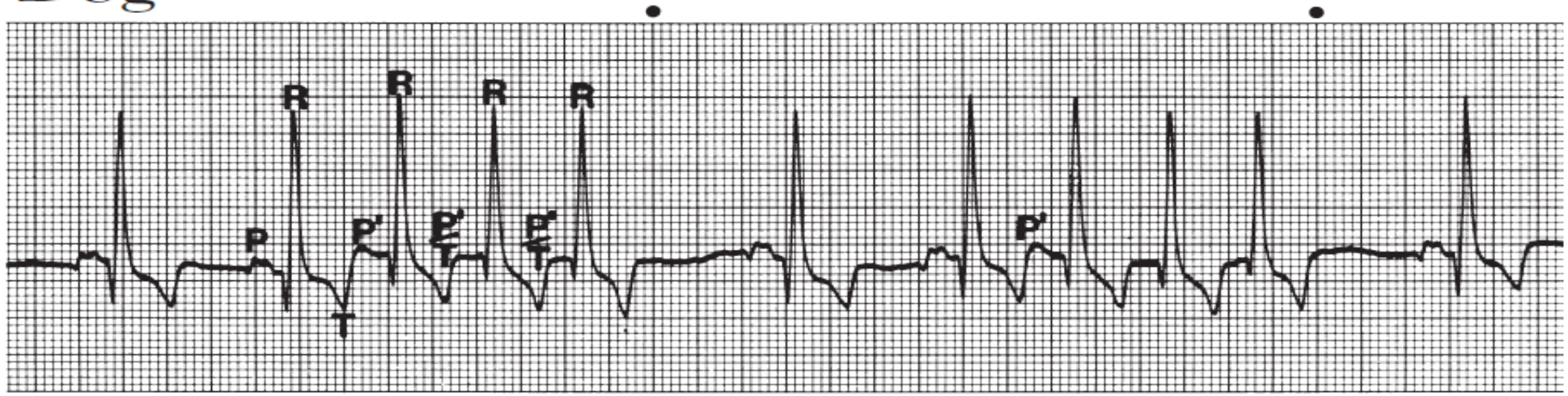
- Ventricular arrhythmias (ventricular extrasystoles [VE], ventricular tachycardia) have been frequently reported in cats with decompensated and compensated HCM.
- Supraventricular arrhythmias: atrial extrasystoles (AE) and atrial tachycardia (supraventricular) may be present in cats with HCM, but are less common than ventricular arrhythmias. Atrial fibrillation (rare) appears in cats with terminal phase HCM and severe left atrial dilation.

Ventricular extrasystoles (VE)/ Ventricular Premature Complexes

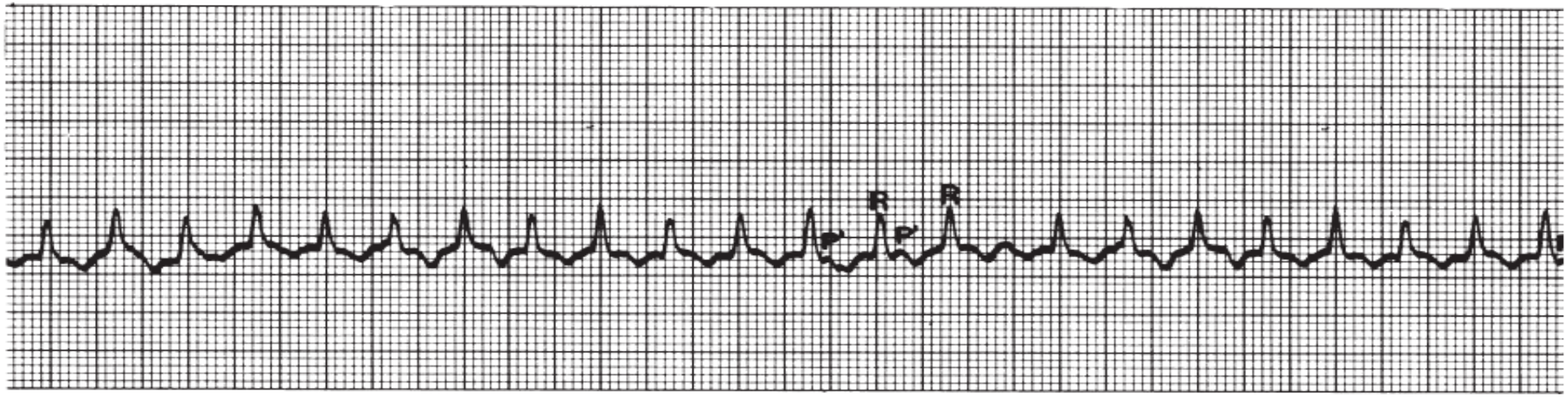


Atrial Tachycardia

Dog

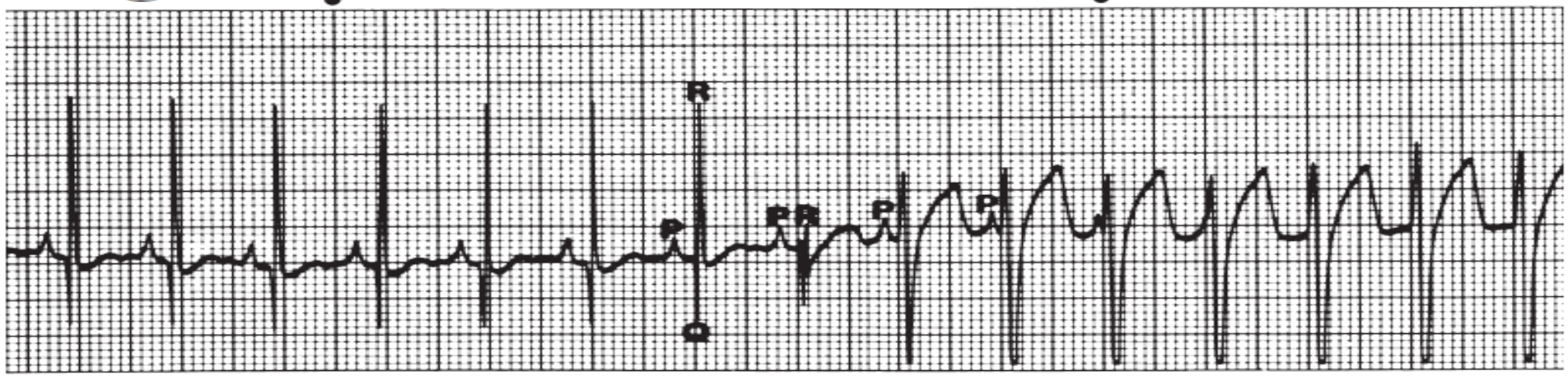


Cat

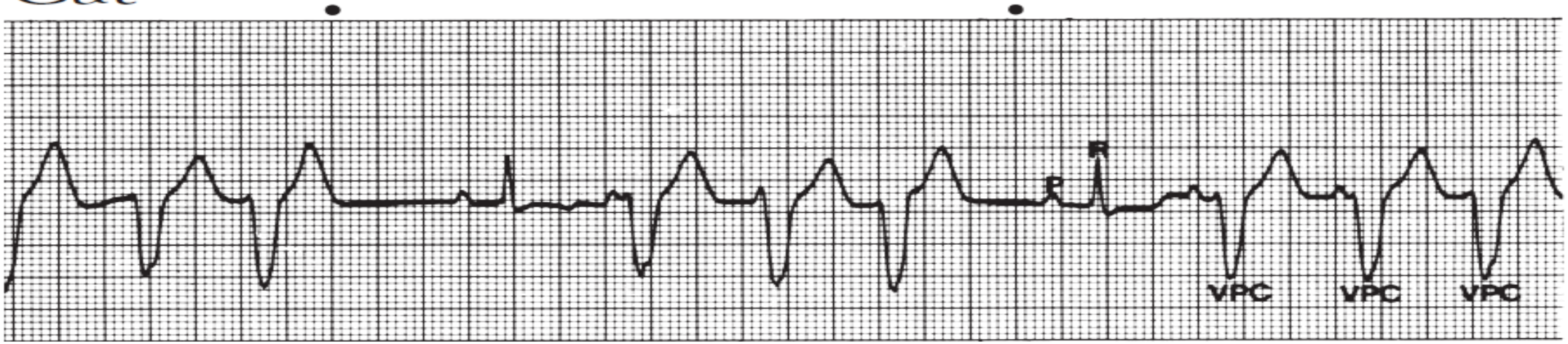


Ventricular Tachycardia

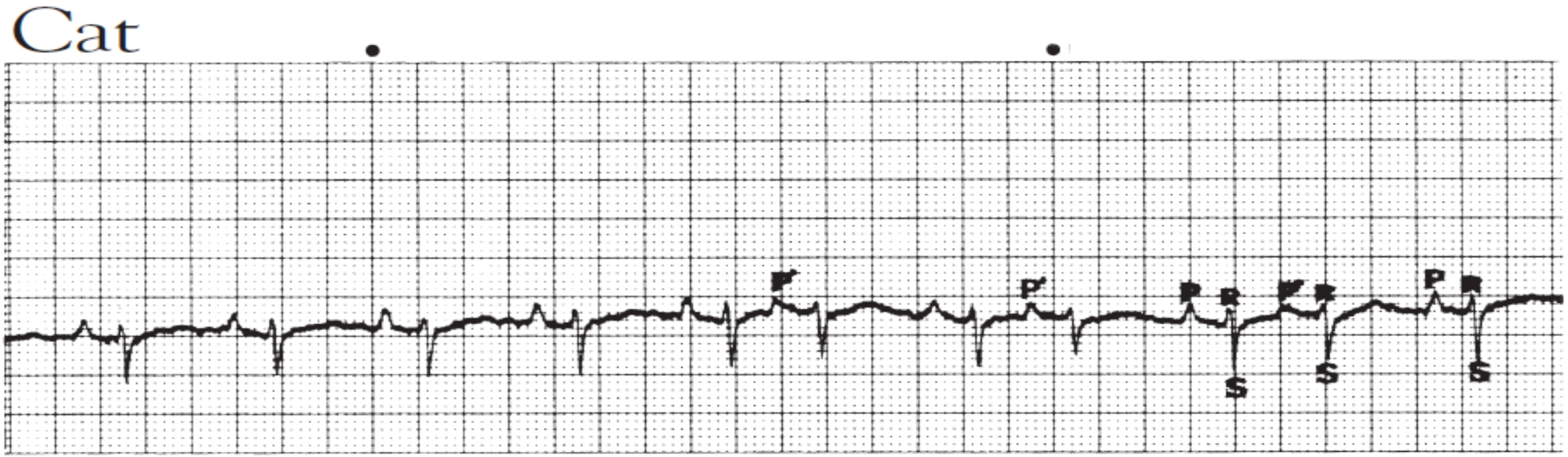
Dog



Cat

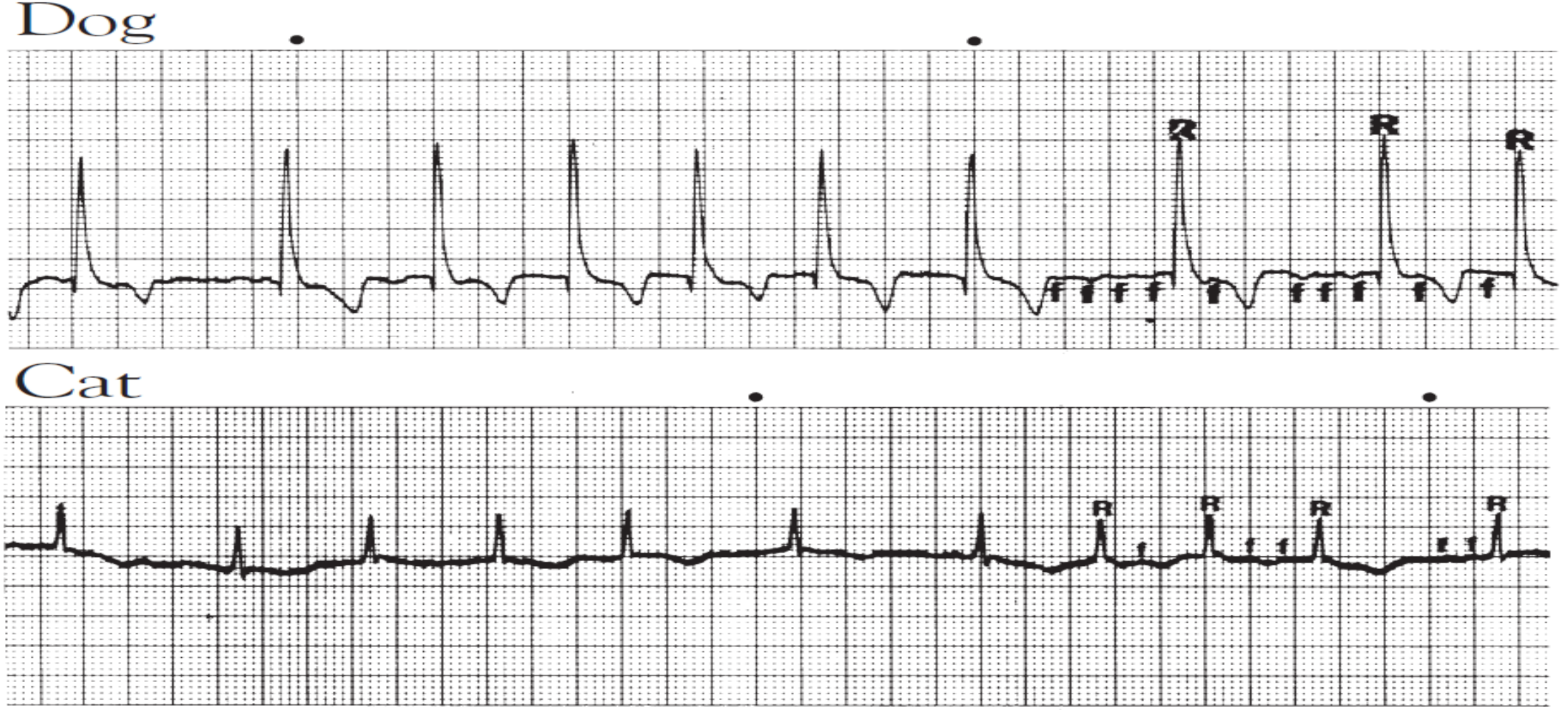


Atrial extrasystoles (AE)/ Atrial Premature Complexes

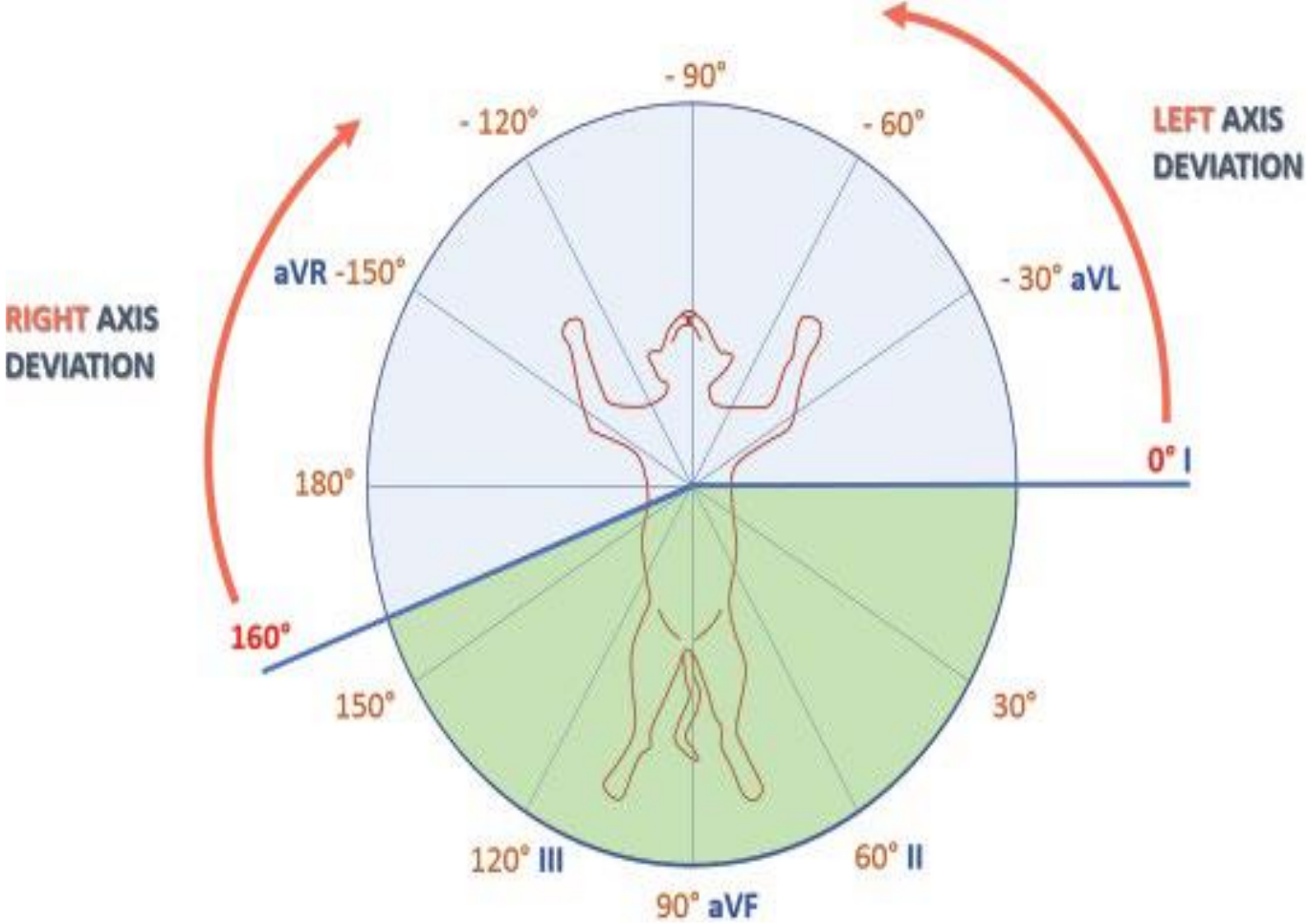


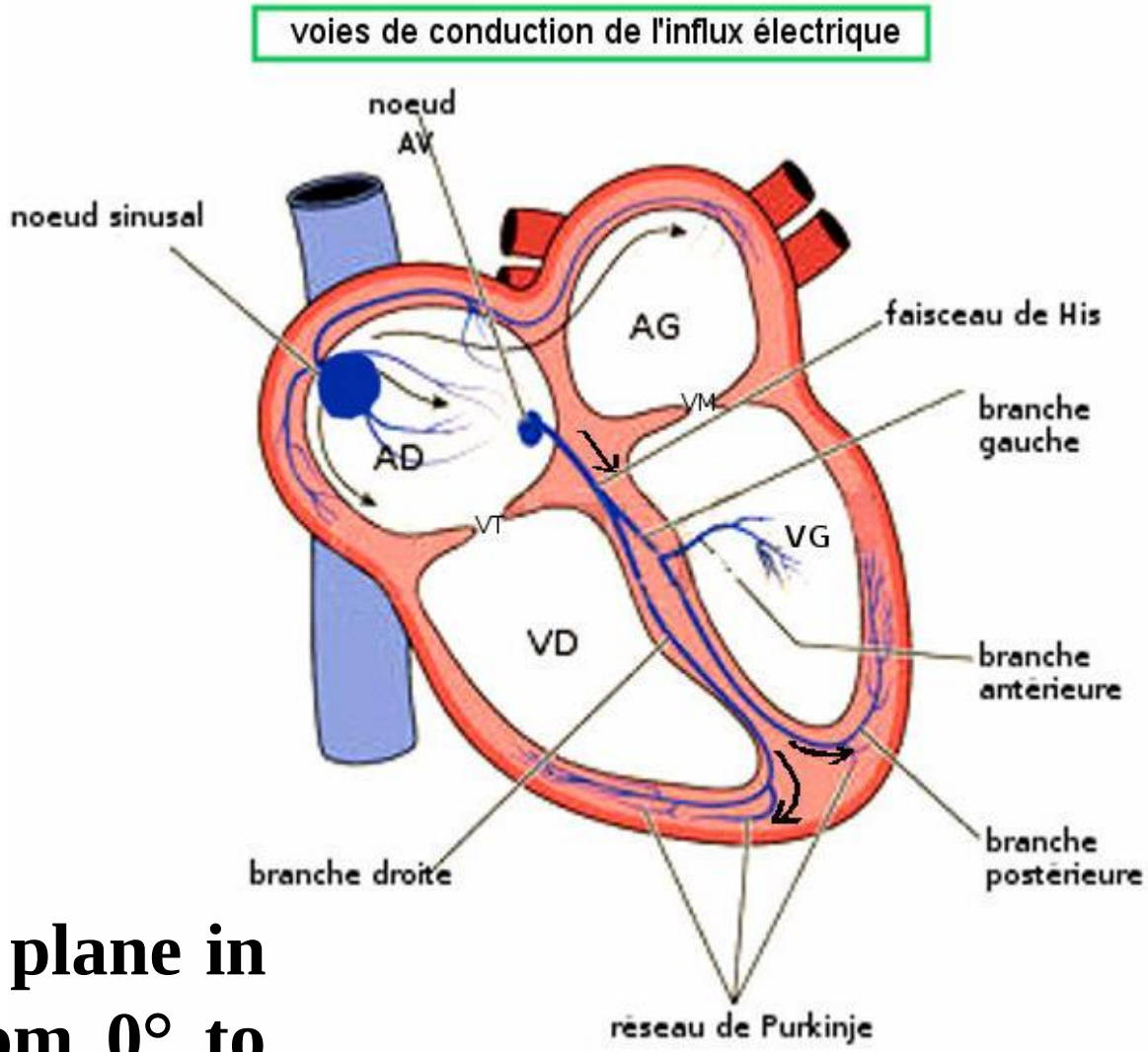
Atrial Fibrillation

Atrial fibrillation



- Several studies have suggested that most cats with ventricular tachyarrhythmias have an underlying structural heart disease.
- Conduction abnormalities may be present: A left deviation of the cardiac axis (from 0° to -90° = hyper-left cardiac axis) suggesting a left anterior fascicular block is common in cats with HCM, but it is also present in cats with other pathologies such as hyperthyroidism.

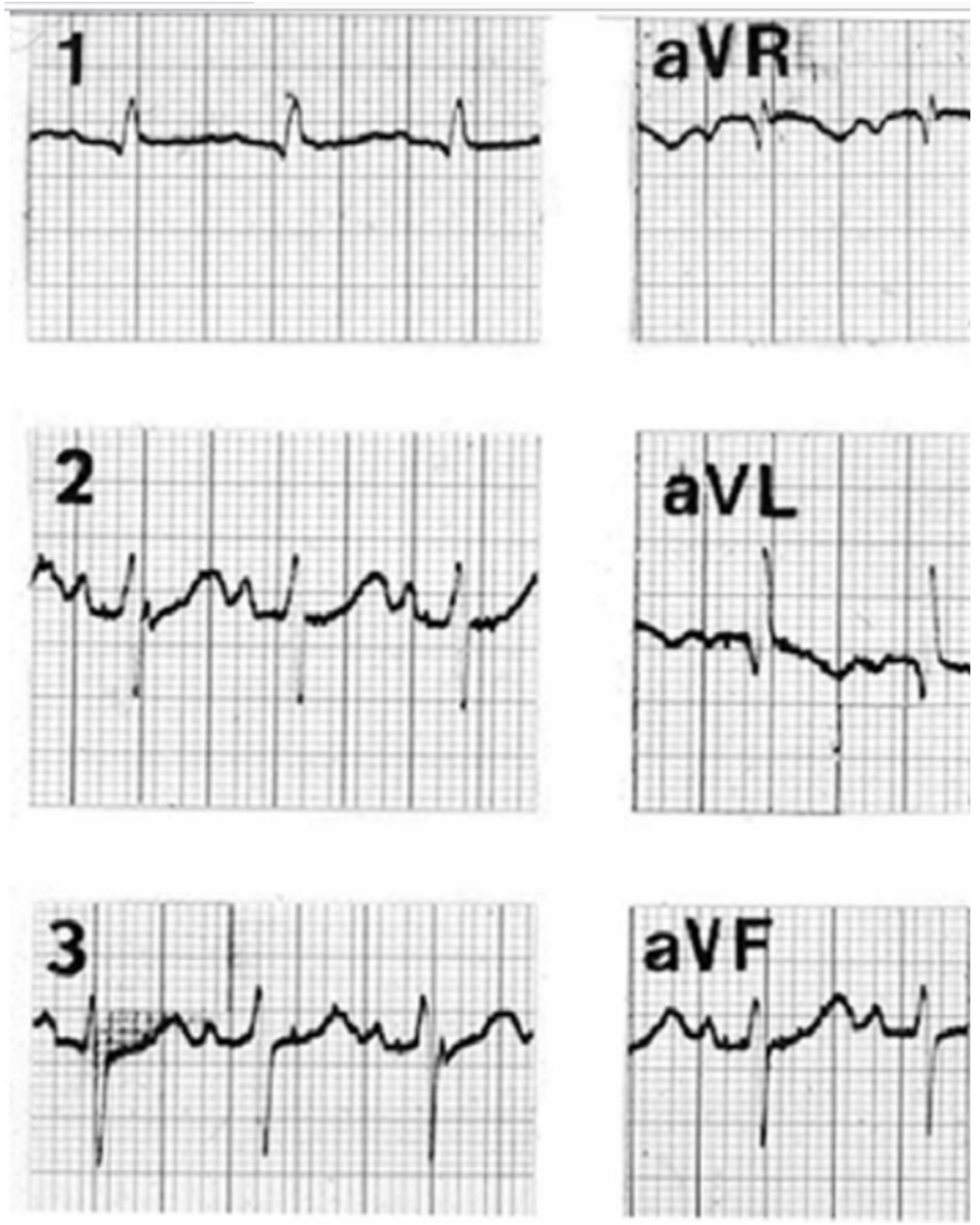




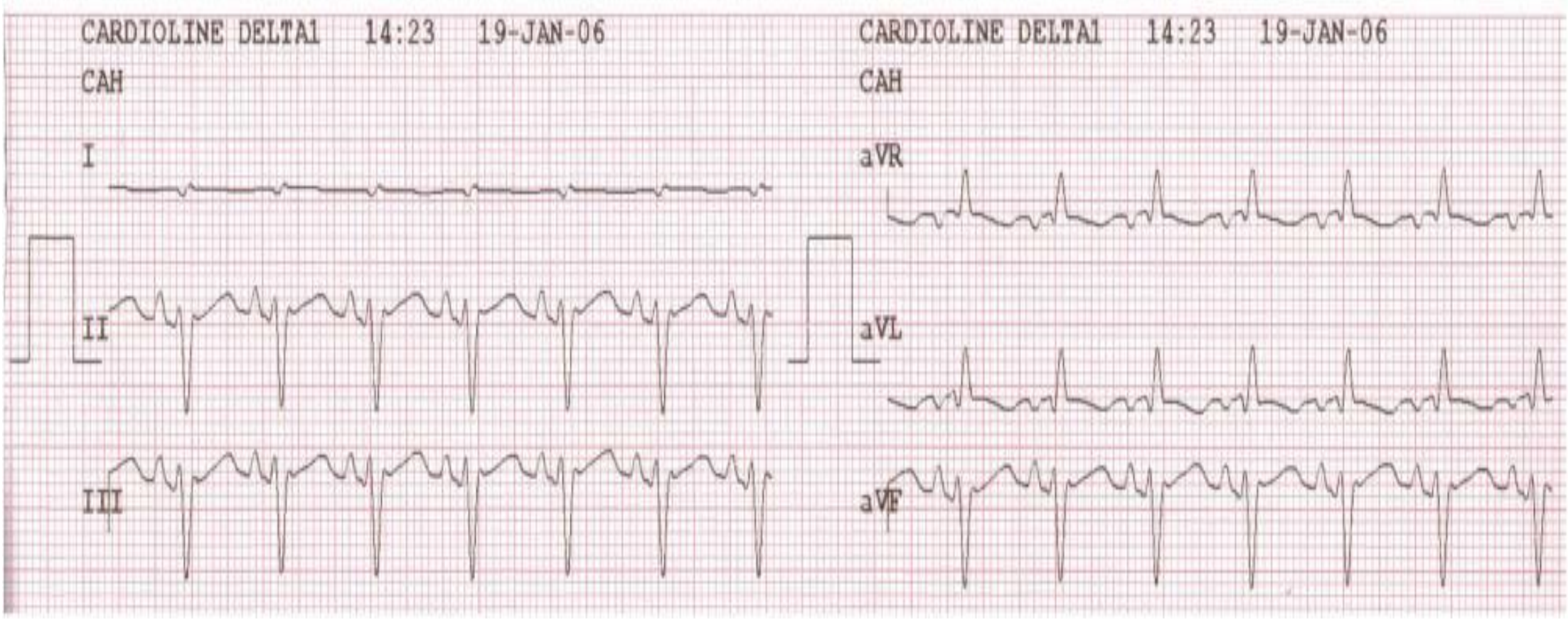
Mean electrical axis (MEA) in the frontal plane in cats. The normal MEA in cats varies from 0° to +160°. A MEA less than 0° is said to be deviated to the left, whereas a right axis deviation is present if the MEA is greater than +160°

The left anterior fascicular block is characterized by:

- A normal to slightly prolonged QRS.
- Presence of qR complexes (small Q waves) in leads I, aVL.
- Presence of rS complexes (deep S waves) in leads II, III, aVF/ Prolonged peak time of the R wave in aVL).
- Hyper leftward eclectic axis (up to -90°).

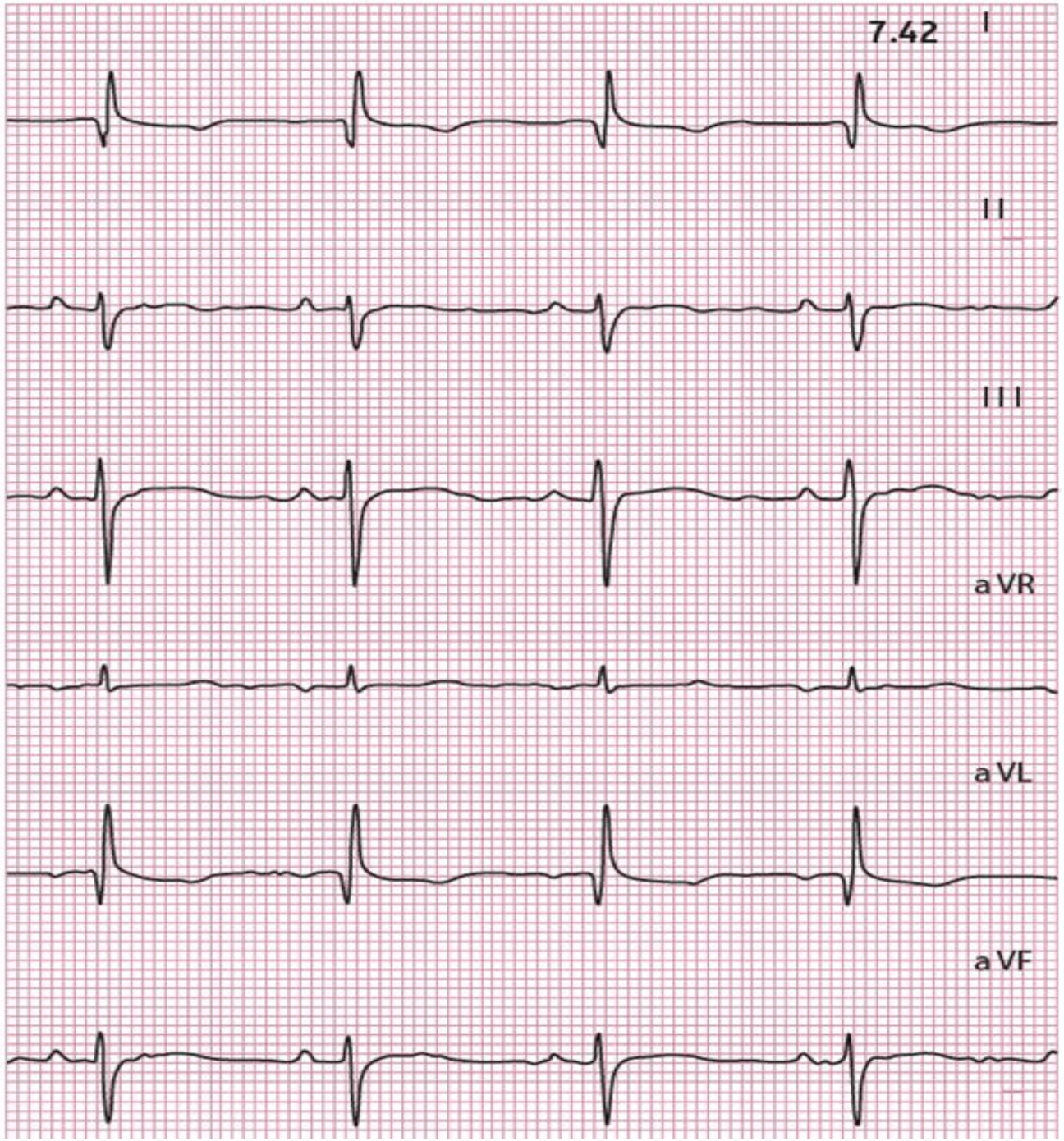


Left anterior fascicular block. Leads II, III, and aVF show deep S waves. A leftward deviation of the left axis (MEA of about −90 degrees).



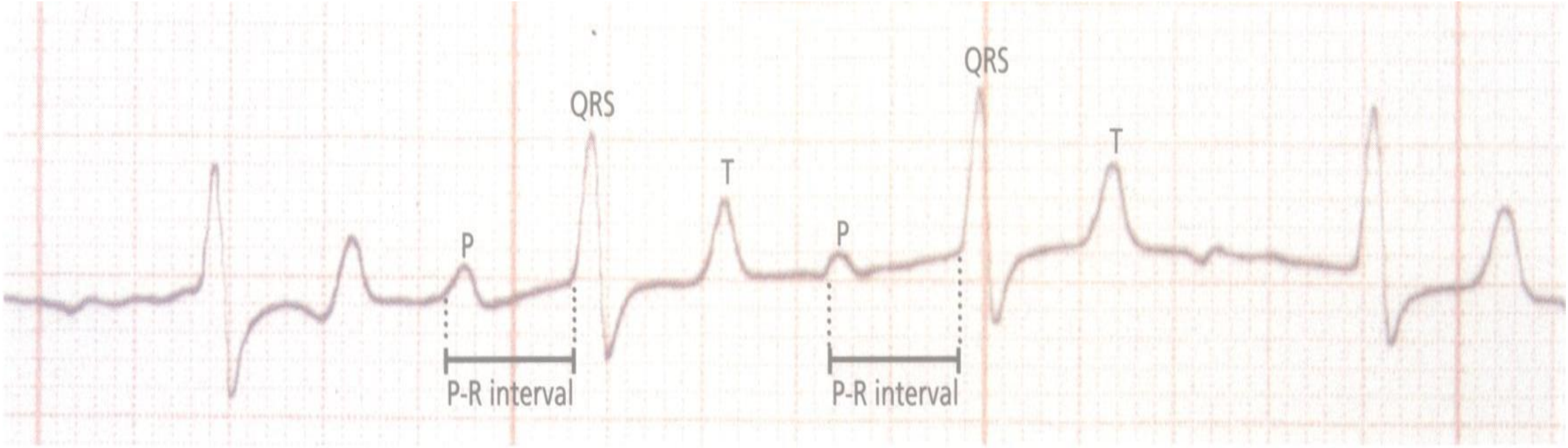
ECG of a cat with HCM showing a sinus rhythm with a left axis deviation between –30° and –60° (Hyper left axis; Left Axis Deviation), also referred to as left anterior fascicular block.

Presence of qR complexes in leads I, aVL/ Presence of rS complexes in leads II, III, aVF.



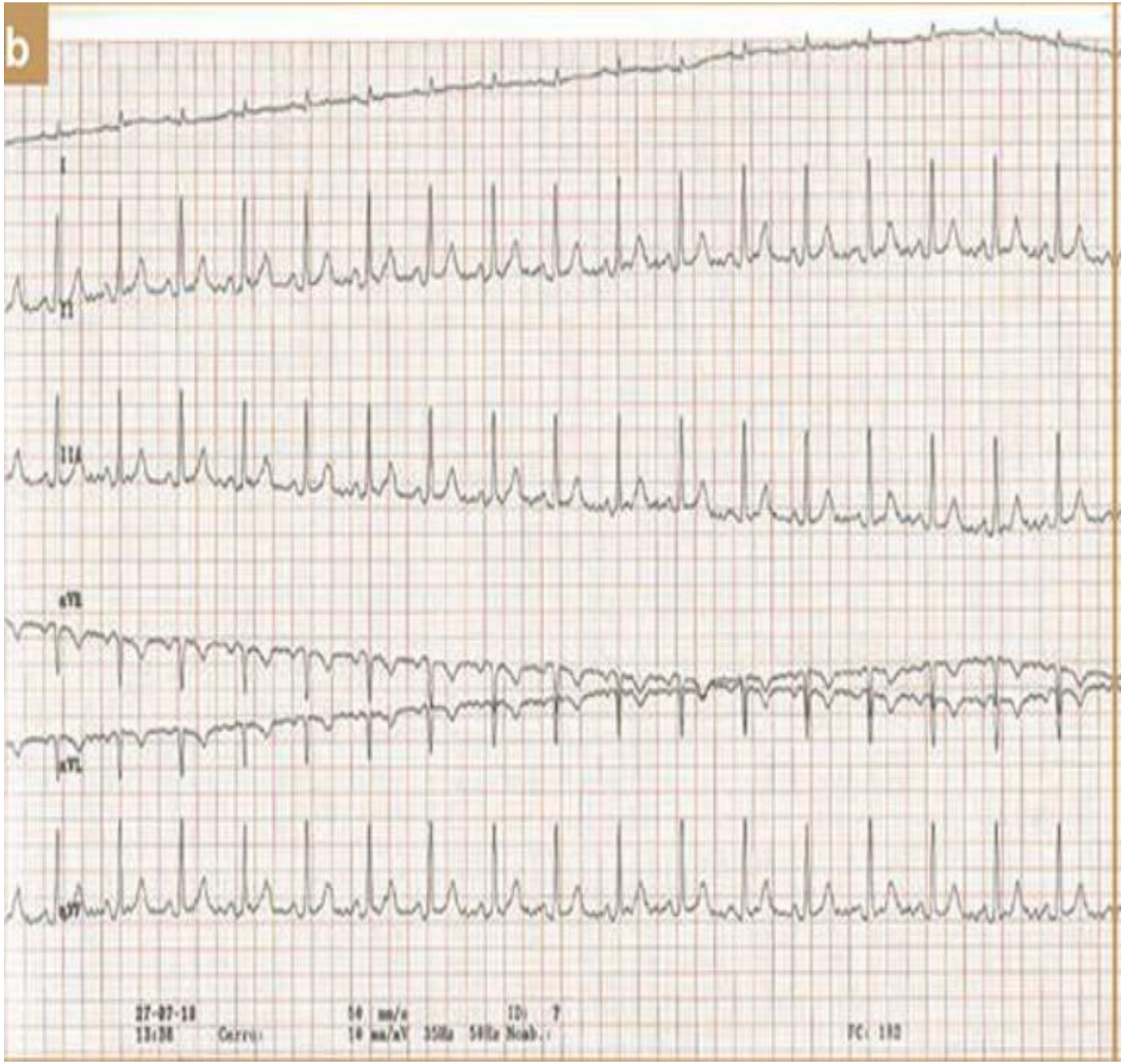
- A normal ECG, atrioventricular blocks (AVB), and sinus bradycardia are also observed in cats with HCM.
- An increase in the amplitude of the R wave and/or the duration of the P wave favor hypertrophy of the left ventricle and left atrial enlargement, although these findings are not sensitive.
- A 24-hour Holter monitoring may be necessary to assess the heart rate in cats presenting episodes of weakness or syncope.

First degree AV block: prolonged PR: - PR >0.13 s in dogs. - PR >0.05 s in cats.



ECG of a dog showing a very prolonged P-R interval = first degree AV block (AVB1).

Electrocardiograms of a cat with HCM. Normal sinus rhythm and increased amplitude of the R wave in DII (1.4 mV, DII; normal < 1.0 mV), suggesting left ventricular hypertrophy; 50 mm/s, 10 mm = 1 mV.



Prognosis

The prognosis for cats with HCM is variable:

- Asymptomatic patients may live several years after diagnosis, with a mean survival time > 3 years.
- A modest number of cats with mild HCM do not experience disease progression and live a normal lifespan.
- Other cats have a more aggressive form of the disease and die suddenly at a young age, develop CHF, or suffer from thromboembolic complications.
- The average survival time for cats with CHF is 6 to 12 months.

The indicators of poor prognosis are given below:

- Age: older cats have a darker prognosis.
- Marked left ventricular hypertrophy (> 9 mm).
- Significant enlargement of the left atrium.
- CHF: In cases of acute CHF, the survival rate with hospitalization is about 80%. Some patients have a shorter survival duration (a few months) while others may survive well beyond a year (some up to 2 to 3 years).

- Thromboembolisms: Cats with thromboembolic complications generally have a poor long-term prognosis, with an average survival of 6 months; up to 30% mortality during the hospitalization period; however, some cats live for several years.
- Patients with systolic dysfunction have a poor prognosis; however, survival may improve with the addition of pimobendan.
- Heart rate > 200 bpm.
- Diminished response to diuretics.

Management

The management of cats with HCM focuses on the following points:

- The standard treatment for feline patients with CHF includes the administration of furosemide and an angiotensin-converting enzyme (ACE) inhibitor.
- Patients with respiratory signs should receive supplemental oxygen and mild sedatives (to reduce stress associated with dyspnea).
- Pimobendan may be included if there is echocardiographic evidence of systolic dysfunction.
- Proper management of arrhythmias is important.

- The prevention of thromboembolic complications with antithrombotics (clopidogrel) is indicated for cats at risk of this complication or those showing signs of arterial thromboembolism.
- It is important to monitor renal values and electrolyte disturbances during the treatment of CHF.
- Hypotension, hypothermia, and bradycardia are not uncommon in patients with CHF (cardiogenic shock); therefore, cats need to be monitored.
- Various medications (beta-blocker, calcium channel blocker, ACE inhibitor, or spironolactone) administered to patients with CHF are not indicated in asymptomatic subjects with HCM.

Drugs used in the management of cardiomyopathy in cats.

Drug	Dosage	Comment
Atenolol	0.5–2 mg/kg PO q12–24h	In cats with dynamic left ventricular outflow tract obstruction. Consider long-term treatment if improvement of hypertrophy. Start with low dose and increase slowly over a few weeks
Enalapril, benazepril	0.25–0.5 mg/kg PO q12–24h	Start q24h
Furosemide	1–2 mg/kg IM or IV q1–8h 1–3 mg/kg PO q8–24h	Monitor blood potassium concentration
Pimobendan	0.25 mg/kg PO q12h	Only when low-output failure confirmed by echocardiography
Clopidogrel	18.75 mg PO q24h	In cats after episode of thromboembolism, or if severe left atrial enlargement +/- visible thrombus