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FELINE HYPERTROPHIC CARDIOMYOPATHY (FHCM)

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ABSTRACT

The feline hypertrophic cardiomyopathy (HCM) is one of many cardiac diseases known in domestic animals, and it is especially frequent in cats of all ages. HCM is the most common heart disease in cats, affecting almost 15 % of the feline population. The Maine Coon and the Ragdoll breeds of domestic cats are shown to have a special gene that is responsible for the appearance of HCM in these breeds. Hypertrophic cardiomyopathy can be detected by: echocardiography, electrocardiography (ECG), radiography, genetic testing, and a test including N- terminal prohormone of brain natriuretic peptide (NT-proBNP). The most frequently used method used for clinical purposes is echocardiography. Patients with HCM will most of the time not have any clinical signs of the disease, which makes it difficult for the owner to detect that there is something wrong with their cat. Some cats, on the other hand, will get heart arrhythmias which can cause sudden death. There are several prognosis-worsening findings that may occur due to the hypertrophy of the ventricles. These include: Feline Arterial Thromboembolism (FATE), Congestive Heart Failure (CHF), and the Dynamic Obstruction of the Left Ventricular Outflow Tract (DOLVOT). The prognosis for HCM depends on the stage of the disease. In some cats, they are well-tolerated and are associated with normal life expectancy, but in other cats they can result in congestive heart failure, arterial thromboembolism or sudden death.

Key words: cats; echocardiography; heart; heart failure; hypertrophy

INTRODUCTION

Hypertrophic cardiomyopathy, or feline hypertrophic cardiomyopathy, is a heart disease that is one of the most commonly encountered cardiac diseases found within felines [19]. HCM is the most common heart disease in cats, affecting almost 15 % of the feline population [35]. This has been known since the 1970s, but it was investigated

further and studied by scientists and veterinarians in the 1990s [10, 19]. The disease is characterized by unusual hypertrophy of the left ventricular wall and the proximal interventricular septum. The mechanism behind the growth is the growth of fibrous tissue within the cardiac muscle that makes the myocardium stiff [23, 38]. As the severity of the disease progresses, the actual structure of the heart is changed and the heart's function is also affected [15].

The thickening of the walls of the ventricle is associated with a decreased ventricular chamber volume and abnormal ventricular relaxation (diastolic function) in cats with HCM. Since the amount of blood pumped by the heart per minute (cardiac output) is the product of the amount of blood ejected per contraction (stroke volume) and the heart rate in beats per minute, this decreased chamber volume (and subsequent stroke volume) results in an increased heart rate (tachycardia) as a reflex mechanism to maintain cardiac output and blood pressure [10, 22]. Although this reflex increase in heart rate may maintain normal blood pressure in the short term, it is associated with increased consumption of oxygen by the heart muscle, to the extent that oxygen demand may exceed supply. This scenario may result in an energy-starved heart muscle, with subsequent heart cell death and worsening function [38].

Another consequence of an increased heart rate is that the ventricle has less time to fill between contractions, further diminishing the stroke volume and promoting a vicious cycle of reflex tachycardia, decreased time for ventricular filling, and so on. This decreased left ventricular filling also promotes stasis of blood in the left atrium (chamber just before the left ventricle), which ultimately contributes to the development of clinical signs.

AETIOLOGY

Although the definitive cause of feline HCM has not been identified, its prevalence within certain breeds, (i. e., Maine Coon cats, and Ragdolls) has prompted speculation that at least some forms of HCM are genetic in origin. The finding of mutations in an important cardiac protein called myosin binding protein C in affected lines of Maine Coon and Ragdoll cats supports a heritable, genetic component of HCM in these breeds [22, 23, 27]. In most cases, HCM affects mostly middle-aged cats, but cats of all ages are affected. HCM is heritable as an autosomal dominant trait [3]. In cats, this disease is more prevalent in: Ragdolls, Maine Coons, oriental breeds (Himalayan, Burmese, Sphynx, Persians), and Devon Rexes, but it is also commonly diagnosed in Domestic Short Hair cats [22]. Other common causes of HCM that may need to be excluded include: aortic stenosis, dehydration, systemic hypertension, hyperthyroidism, and acromegaly [27].

CLINICAL SYMPTOMS AND PROGNOSIS-WORSENING FINDINGS

It is common for cats with HCM to be asymptomatic, which means that the cat does not show any external and behavioural signs of the disease. The disease may be discovered by the sound of a heart murmur or arrhythmias. Murmurs in patients with HCM have been associated with SAM, which results in LVOTO and mitral regurgitation (MR), as well as with MR caused by hypertrophic remodelling and distortion of the mitral apparatus [20]. Additionally, dynamic right ventricular outflow tract obstruction has been identified as a cause of murmurs in healthy cats and cats with the noncardiac disease. Numerous types of arrhythmias can be identified on a resting ECG, including atrial and ventricular premature complexes (APCs and VPCs), atrial and ventricular tachycardia, and atrial fibrillation [6, 20]. The presence of arrhythmia and an audible gallop have been associated with a worse outcome and a detectable arrhythmia has been associated with a greater risk of sudden death [18].

In other cases, signs of congestive heart failure including laboured or rapid breathing, open-mouth breathing, and lethargy are evident. These signs occur when fluid accumulates in the lung tissue (pulmonary edema) or around the lungs (pleural effusion) secondary to the elevation of left atrial pressure [2, 33].

Prognosis-worsening findings of feline HCM

1. Dynamic obstruction of the left ventricular outflow tract (DOLVOT)

The systolic anterior motion of the mitral valve (SAM) is the most common cause of dynamic obstruction of the left ventricular outflow tract (DOLVOT) and, in turn, the most common cause of a heart murmur with feline HCM (Fig. 1).

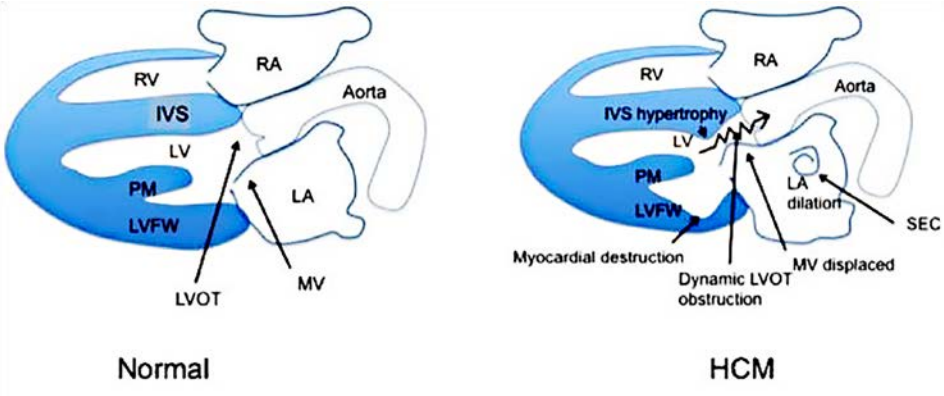


Fig. 1. Illustration of the mechanism of DOLVOT
Art by: Eric de Madron. https://www.vetspecialists.com/vet-blog-landing/animal-health-articles/2020/04/29/hypertrophic-cardiomyopathy-in-cats

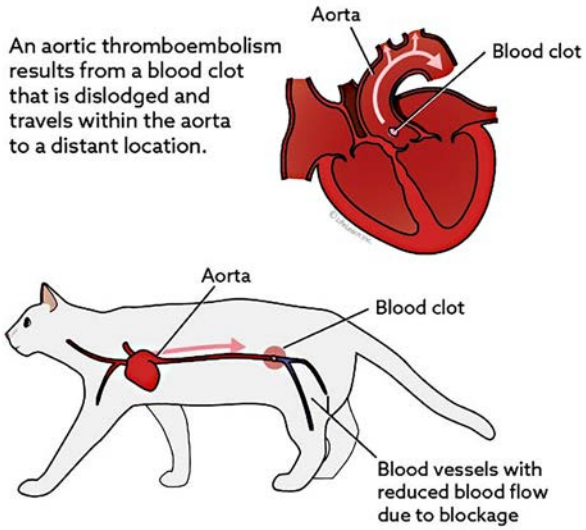


Fig. 2. Illustration of where the thrombus forms and where it clogs the arteries in FATE
Art by Malcolm Weir. https://vcahospitals.com/know-your-pet/aortic-thromboembolism-in-cats

When hypertrophic cardiomyopathy occurs, the proximal part of the interventricular septum becomes enlarged. This, as said earlier, will make for less room for the mitral valve to function, and it will be squeezed against the septum [23]. This leads to a reduction of the mitral valve's function, which again leads to obstruction of the passage.

2. Feline arterial thromboembolisms (FATE)

Diastolic dysfunction, and its consequences of abnormally increased atrial pressure leading to signs of heart failure, and sluggish atrial blood flow leading to Feline arterial thromboembolism (FATE).

A potentially devastating cloud of HCM is thromboembolism. Thromboembolism refers to the development of a clot in the heart (promoted by left atrial enlargement), with ejection of the clot to the systemic circulation [25] (Fig. 2).

When the clot lodges in the peripheral circulation, it may obstruct blood flow to the region of the heart supplied by the blocked vessel. The site of thromboembolism most commonly observed in cats with HCM is the distal aorta (termed a saddle thrombus), and clinical signs of hind limb paralysis and acute pain in the hind limbs may be observed [29]. Thromboembolism is a poor prognostic indicator in cats with HCM [22].

3. Congestive heart failure (CHF)

Congestive heart failure is a common clinical syndrome

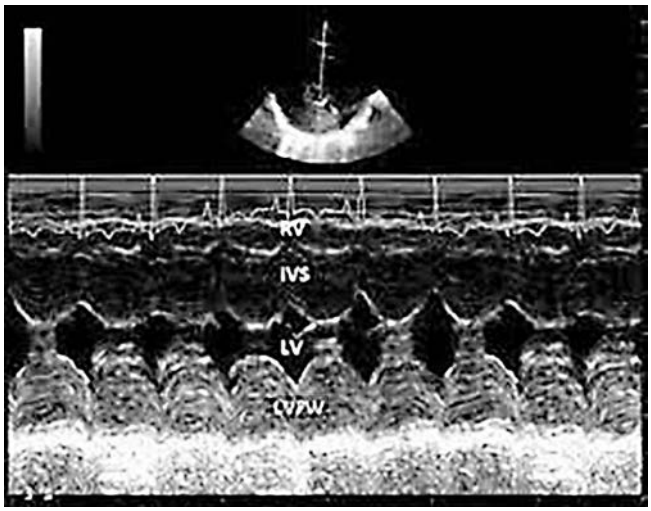


Fig. 3. Two-dimensional and M-Mode echocardiographic examination of a cat with HCM on right parasternal short axis view at the level of the *chordae tendineae*
Note interventricular septum and left ventricular free wall hypertrophy.
LVFW: left ventricular free wall; IVS: interventricular septum and RV: right ventricle.
Art by Silva et al. 2013. https://scielo.conicyt.cl/pdf/amv/v45n1/art02.pdf

that is a complication due to different causes, in this case, hypertrophic cardiomyopathy, and it manifests itself in different ways [28]. The word "heart failure", means that the function of the heart cannot meet the normal physiological demands of the body, which can in the worst-case lead to the death of the animal [23, 25]. One definition that can be used for the syndrome, is that it is a condition where the heart is unable to supply a sufficient amount of blood to the peripheral tissues to meet their metabolic needs [28]. It is a condition where the blood that usually is pumped straight into the aorta from the left ventricle can get stuck in the left atrium, and will end up streaming back to the lungs and into the thoracic cavity [30]. This is a critical condition, because the lungs and thoracic cavity can overflow and the cat will end up having trouble breathing. Due to the respiratory problems involved as a result of CHF, a clear sign of the condition would be that the cat's breathing rate is increasing, as the cat is trying hard to get as much air into its system as possible. Also, the heart rate will increase, and one can in some cases even feel the heartbeat clearly from the chest of the cat [23].

4. Systemic hypertension

Diffuse or segmental LV hypertrophy is common in cats with systemic hypertension and is observed in up to 85 % of cases, although HCM and systemic hypertension may exist concurrently. For many hypertensive cats, LV hypertrophy is only mild to moderate. Blood pressure determination should be considered for all cats with increased LV wall thickness (LOE medium) [13].

Acute stress resulting in severe tachycardia can result in acute deterioration of diastolic function and so precipitate acute left heart failure. Anaesthesia, surgery, intra venose fluid therapy and possibly corticosteroid administration can tip a cat with subclinical disease over into heart failure. However, most cats with HCM that present in heart failure have no apparent exacerbating disease or precipitating event [20, 33].

Exacerbation of clinical symptoms and sudden cat death may occur with increased exertion and stress during the clinical examination and handling. Gentle handling, reduced stress or the use of sedation can be preventive [33].

DIAGNOSTICS

The definitive diagnosis of HCM is almost always made using echocardiography. Other imaging modalities, such as CT and MRI, are used in human medicine [14]. Electrocardiography can reveal changes in some cats with HCM but is not a reliable indicator of disease [31]. Radiography cannot be used to distinguish HCM from the other cardiomyopathies but is valuable for identifying severe left atrium (LA) enlargement.

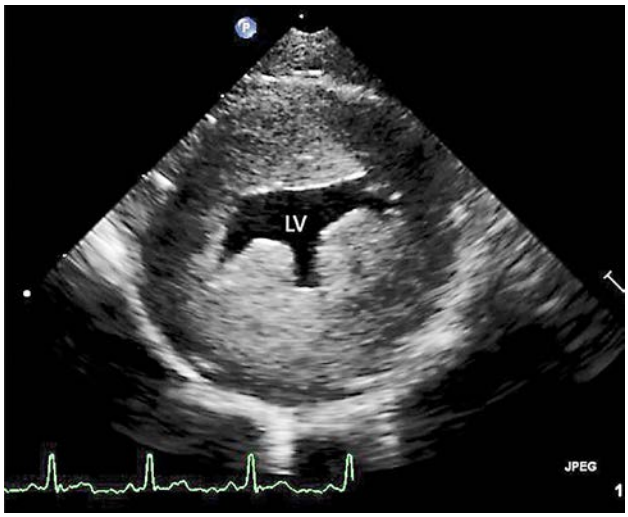


Fig. 4. Two-dimensional short-axis ultrasonographic view of Tux showing concentric left ventricular hypertrophy and papillary muscle hypertrophy, consistent with hypertrophic cardiomyopathy. His left atrium was normal in size on other images. LV, left ventricle

Art by Brent Aona et al., 2021. https://todaysveterinarypractice.com/advances-feline-cardiac-diagnostics/

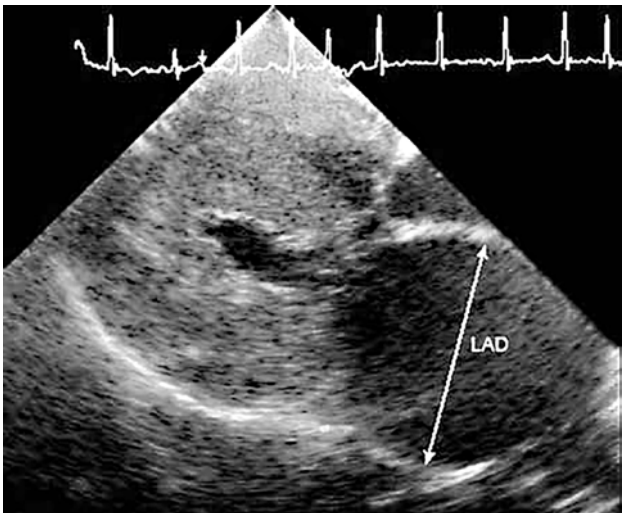


Fig. 5. Left ventricular M-mode of a cat with HCM, demonstrating systolic anterior motion (SAM) of the mitral valve

Art by Fuentes et al., 2003. https://www.vin.com/apputil/content/defaultadv1.aspx?meta=&pId=11149&id=3846601

Echocardiography

Feline HCM has been echocardiographically defined by end-diastolic measurements of the ventricular wall thickness that equals to or exceeds 6 mm (Fig. 3, 4) [10]. Hypertrophy is commonly asymmetric and in Maine coon cats with HCM, it is often the left ventricular posterior wall and papillary muscles that are most affected [19]. Ejection phase indices of the left ventricular systolic performance such as % fractional shortening are usually normal or reflect hyperdynamic ventricular emptying.

Left atrial size in cats with HCM is generally greater than in healthy cats, but left atrial enlargement is not an intrinsic feature of the disease nor required for its diagnosis (Fig. 5) [1]. However, left atrial size is a surrogate measure of hemodynamic burden, and left atrial enlargement has been associated with poor outcomes in people and cats with HCM [26, 33].

Diastolic dysfunction is a characteristic feature of HCM. Diastolic function is typically assessed in cats with HCM using tissue Doppler imaging. Most commonly a pulsed wave tissue Doppler imaging gate is placed at the lateral mitral valve annulus to measure the velocity at which it moves in early diastole (E' wave) [36]. In cats with severe HCM, the E' wave velocity is typically decreased [24].

Cardiac biomarkers

When the left ventriculus (LV) wall is severely thickened, the myocardial blood supply is compromised [34]. This results in ongoing myocyte damage and death, as evidenced by an elevation in cardiac troponin I (cTn I) in cats with HCM [5, 16]. Cardiomyocytes that die are replaced with fibrous tissue (replacement fibrosis), as evidenced by increased concentrations of circulating biomarkers of type I collagen [4]. The measurement of NT-proBNP and cTn I concentrations in serum or plasma has been evaluated extensively as a means of screening for HCM in cats without heart failure (subclinical HCM) in veterinary referral hospitals and also as a means of differentiating heart failure from primary respiratory disease in cats presented for dyspnoea [40]. The measurement of NTproBNP is widely available (through a diagnostic laboratory) which may help to identify patients with preclinical HCM at greater risk when echocardiography is not available [18].

Electrocardiography (ECG)

While the ECG is sensitive for detecting atrial fibrillation, atrial fibrillation is not an easy diagnosis to make in a cat (the ECG may not be specific for atrial fibrillation in cats) [40]. Sinus rhythm with small P waves (which may be obscured by artifact) and frequent APCs and atrial tachycardia often masquerade as atrial fibrillation in cats. The

ECG is insensitive for detecting sporadic APCs and VPCs in any species. A 24-h ambulatory ECG (Holter monitor) is a more sensitive means of identifying these arrhythmias [39].

Other common causes of HCM that may need to be excluded include: aortic stenosis, dehydration, systemic hypertension, hyperthyroidism, and acromegaly. However, there are caveats. Systemic hypertension and hyperthyroidism do not cause severe HCM, so if a cat has severe HCM (arbitrarily defined as diastolic LV wall thickness ≥ 7 mm) and systemic hypertension or hyperthyroidism, it can generally be assumed that these systemic disorders are not the sole cause of the HCM [22, 36].

TREATMENT

There is no documented reason to treat a cat with subclinical HCM that has mild to severe wall thickening and a normal to mildly enlarged LA (stage B1) if the goal is to delay the onset of heart failure. This is because there is no medication (including ACE inhibitors, beta-blockers, and spironolactone) that has been shown to reduce hypertrophy or slow progression of the disease, if it is destined to progress [36]. Therefore, the best that can be done is to:

- Monitor the cat for the development of severe LA enlargement (so that antiplatelet/ anticoagulant therapy can be started);
- Avoid treatments that can trigger heart failure iatrogenically (e. g., injudicious fluid therapy);
- Not breed the cat if it is sexually intact;
- Monitor for the onset of left heart failure, if the LA is moderate to severely enlarged [36].

The treatment of hypertrophic cardiomyopathy in cats is mostly palliative, because there is no way to reduce the enlargement of the cardiac muscle tissue. This makes the goal of the management of HCM to improve the LV ability to fill and pump out blood, together with delaying the onset of congestive heart failure and reducing the risk of further complications such as feline arterial thromboembolism. Beta-receptor blockers, calcium-channel blockers, and ACE-inhibitors are some of the methods for the treatment of HCM in felines. Beta-receptor blockers include drugs such as propranolol and atenolol that reduce the excretion of catecholamines and the influence of the sympathetic nervous system of the heart [32]. Calcium-channel blockers, such as diltiazem and verapamil, have a similar function as beta-blockers. ACE-inhibitors, on the other hand, affect the RAAS by inhibiting the production of angiotensin II and aldosterone. The function of ACE inhibitors is to minimize the fibrosis of the myocardium [7, 17, 24]. The overall function of the treatment of HCM is not to eliminate the disease, since it is chronic, but to minimize the heart rate and to improve the filling of blood in the LV in the diastole [8, 9].

The best way to do the last is to have the owner monitor the cat's sleeping respiratory rate (RR; normal is < 30 breaths.min^{-1}) and to maintain a log. in general, this should only be performed in a cat with evidence of moderate to severe LA enlargement to avoid over vigilance. The owner then needs to be instructed to call a veterinarian when the sleeping RR increases, before the onset of any severe dyspnoea and hopefully avoid the all-too-common weekend or evening visit to an emergency clinic [20].

PROGNOSIS

The prognosis for HCM depends on the stage of the disease. Many cats with mild to moderate HCM never progress to severe HCM and so have an excellent prognosis. However, if followed serially, a significant number of cats will progress to severe HCM [11, 20, 37]. Most cats with severe LV wall thickening and moderate to severe LA enlargement (stage B2) that are not in heart failure will progress to heart failure or experience ATE. For some perspective, one study examined the time from diagnosis of subclinical HCM (stages B1 and B2) to onset of heart failure and found that approximately 7 % of the cats developed heart failure within the first year, 20 % within 5 years, and 25 % within 10 years [12]. Once in heart failure, approximately half were dead within 2 months. Overall, in cats with ATE, 70 % were dead within a week. The last stands in contrast to the average survival of 11.5 months in the 37 % of cats with ATE that survive the acute episode and the 20 % of cats with ATE that lived 4 years or more in other studies [21, 33]. The exceptions to "the most will die within months, not years" rule include cats with TMT (Transient Myocardial Thickening) and those that are in heart failure due to stress, fluid administration or cortico steroid administration. These cats can stabilize after being treated for heart failure and may live for years [33].

CONCLUSIONS

Hypertrophic cardiomyopathy has been known as a common cardiac disease among both felines and humans for decades. The main sign of the disease is heart murmurs, arrhythmias, low blood pressure, and hind limb paralysis. HCM is a chronic disease, and there is no cure for it. This means that it is important to screen the cats that are especially known to carry genes for HCM. These breeds include mainly Maine Coon and Ragdoll, but also other breeds such as British Shorthair, Devon Rex, Sphynx, and other oriental breeds are known to be more commonly affected by the disease than other breeds. For that reason, it is really important with regular screening by echocardiography of cats that are used for breeding purposes to prevent the disease to inherit down later litters.

REFERENCES

1. **Abbott, J. A., MacLean, H. N., 2006:** Two-dimensional echocardiographic assessment of the feline left atrium. *J. Vet. Intern. Med.*, 20, 111—119. DOI: 10.1892/0891-6640(2006)20[111:teaotf]2.0.co;2.
2. **Atkins, C. E., Gallo, A. M., Kurzman, I. D., 1992:** Risk factors, clinical signs, and survival in cats with a clinical diagnosis of idiopathic hypertrophic cardiomyopathy: 74 cases (1985—1989). *J. Am. Vet. Med. Assoc.*, 201, 613—618.
3. **Atkins, C. E., 2004:** Feline hypertrophic cardiomyopathy 2004. In *The 30th Congress of the World Small Animal Veterinary Association Proceedings Online*. Mexico City, Mexico, 2005.
4. **Borgeat, K., Dudhia, J., Luis Fuentes, V., 2015:** Circulating concentrations of a marker of type I collagen metabolism are associated with hypertrophic cardiomyopathy mutation status in Ragdoll cats. *J. Small Anim. Pract.*, 56, 360—365. DOI: 10.1111 /jsap.12332.
5. **Connolly, D. J., Cannata, J., Boswood, A., 2003:** Cardiac troponin I in cats with hypertrophic cardiomyopathy. *J. Feline Med. Surg.*, 5, 209—-216. DOI: 10.1016/S1098-612X(03)00007-X.
6. **Côté, E., Harpster, N. K., Laste, N. J., 2004:** Atrial fibrillation in cats: 50 cases (1979—2002). *J. Am. Vet. Med. Assoc.*, 225, 256—260.
7. **Crowley, S. D., 2006:** Angiotensin II causes hypertension and cardiac hypertrophy through its receptors in the kidney. *Proc. Natl. Acad. Sci. USA*, 103, 47, 17985—17990. DOI: 10.1073/pnas.0605545103.
8. **Fearnley, C. J., Roderick, H. L., Bootman, M. D., 2011:** Calcium signaling in cardiac myocytes. *Cold Spring Harb. Perspect. Biol.*, 3, 11. DOI: 10.1101/cshperspect.a004242.
9. **Fox, P. R., 1988:** Canine and feline cardiology. *Tijd schr. Diergeneeskd.*, 113, 21—25.
10. **Fox, P. R., Liu, S. K., Maron, B. J., 1995:** Echocardiographic assessment of spontaneously occurring feline hypertrophic cardiomyopathy: An animal model of human disease. *Circulation*, 92, 2645—2651. DOI: 10.1161/01.cir.92.9.2645.
11. **Fox, P. R., Oyama, M. A., Reynolds, C., 2009:** Utility of plasma N-terminal pro-brain natriuretic peptide (NT-proBNP) to distinguish between congestive heart failure and non-cardiac causes of acute dyspnea in cats. *J. Vet. Cardiol.*, 11, 51—61. DOI: 10.1016/j.jvc.2008.12.001.
12. **Fox, P. R., Keene, B. W., Lamb, K., 2018:** International collaborative study to assess cardiovascular risk and evaluate long-term health in cats with preclinical hypertrophic cardiomyopathy and apparently healthy cats: The REVEAL Study. *J. Vet. Intern. Med.*, 32, 930—943.
13. **Fuentes, L. V., Abbott, J., Chetbou, V., Côté, E., 2020:** ACVIM consensus statement guidelines for the classification, diagnosis, and management of cardiomyopathies in cats. *J. Vet. Int. Med.*, 34, 1062—1077. DOI: 10.1111/jvim.15745.
14. **Habib, M., Hoss, S., Rakowski, H., 2019:** Evaluation of hypertrophic cardiomyopathy: newer echo and MRI approaches. *Curr. Cardiol. Rep.*, 11, 51—61. DOI: 10.1016/j.jvc.2008.12.001.
15. **Häggström, J., Fuentes, V. L., Wess, G., 2015:** Screening for hypertrophic cardiomyopathy in cats. *J. Vet. Cardiol.*, 17, 134—149. DOI: 10.1016/j.jvc.2015.07.003.
16. **Herndon, W. E., Kittleson, M. D., Sanderson, K., 2002:** Cardiac troponin I in feline hypertrophic cardiomyopathy. *J. Vet. Intern. Med.*, 16, 558—564. DOI: 10.1892/0891-6640(2002)016< 0558:ctiifh>2.3.co;2.
17. **Hori, Y., Iguchi, M., Heishima, Y., 2018:** Diagnostic utility of cardiac troponin I in cats with hypertrophic cardiomyopathy. *Vet. Intern. Med.*, 32, 922—929. DOI: 10.1111/jvim.15131.
18. **Ironside, V. A., Tricklebank, P. R., Boswood, A., 2020:** Risk indictors in cats with preclinical hypertrophic cardiomyopathy: a prospective cohort study. *J. Feline Med. Surg.*, 1, 11. DOI: 10.1177/1098612X20938651.
19. **Kittleson, M. D., Meurs, K. M., Munro, M. J., 2015:** Familial hypertrophic cardiomyopathy in Maine coon cats: an

animal model of human disease. *Circulation*, 99, 3172—80. DOI: 10.1161/01.cir.99.24.3172.

20. **Kittleson, M. D., Coté, E., 2021:** The feline cardiomyopathies: Hypertrophic cardiomyopathy. *J. Feline Med. Surg.*, 23, 1028—1051.
21. **Laste, N. J., Harpster, N. K., 1995:** A retrospective study of 100 cases of feline distal aortic thromboembolism: 1977—1993. *J. Am. Anim. Hosp. Assoc.*, 31, 492—500.
22. **Lernfelt, L., 2010:** Felin hypertrofisk kardiomyopati—patofysiologi och behandling. *Sveriges lantbruksuniversitet Fakulteten för veterinärmedicin och husdjursvetenskap*. Uppsala 2010, 18 pp.
23. **Madron, E., 2004:** *Hypertrophic Cardiomyopathy in Cats*. AVCIM: American College of Veterinary Internal Medicine. https://www.vetspecialists.com/vet-blog-landing/animal-health-articles/2020/04/29/hypertrophic-cardiomyopathy-in-cats. Accessed March 15, 2021.
24. **MacDonald, K. A., Kittleson, M. D., Garcia-Nolen, T., 2006:** Tissue Doppler imaging and gradient-echo cardiac magnetic resonance imaging in normal cats and cats with hypertrophic cardiomyopathy. *J. Vet. Intern. Med.*, 20, 627—634. DOI: 10.1892/0891-6640(2006)20[627:tdiage]2.0.co;2.
25. **McDermott, A., 2018:** Diastole vs. systole: A guide to blood pressure. *Healthline*, 97, 575—580.
26. **Nistri, S., Olivotto, I., Betocchi, S., 2006:** Prognostic significance of left atrial size in patients with hypertrophic cardiomyopathy (from the Italian registry for hypertrophic cardiomyopathy). *Am. J. Cardiol.*, 98, 960—965. DOI: 10.1016/j.amjcard.2006.05.013.
27. **Ortuño, G. C., Sebastián-Marcos, P., Sabater-Molina, M., Nicolas-Rocamora, E., Gimeno-Blanes, J. R., Fernández del Palacio, M. J., 2019:** Genetics of feline hypertrophic cardiomyopathy. *Clin. Gen.*, 98, 3, 203—214. DOI: 10.1111/cge.13743.
28. **Parmley, W. W., 1985:** Pathophysiology of congestive heart failure. *Am. J. Cardiol.*, 55, 9—14. DOI: 10.1016/0002-9149(85)90790-8.
29. **Payne, J. R., Brodbelt, C., Fuentes, V., 2015:** Cardiomyopathy prevalence in 780 apparently healthy cats in rehoming centers (the CatScan study). *J. Vet. Cardiol.*, 17, 244—257. DOI: 10.1016/j.jvc.2015.03.008.
30. **Reina-doreste, Y., Stern, J. A., Keene, B. W., 2014:** Case-control study of the effects of pimobendan on survival time in cats with hypertrophic cardiomyopathy and congestive heart failure. *J. Am. Vet. Med. Assoc.*, 245, 534—539. DOI: 10.2460/javma.245.5.534.
31. **Romito, G., Guglielmini, C., Mazzarella, M. O., 2018:** Diagnostic and prognostic utility of surface electrocardiography in cats with left ventricular hypertrophy. *J. Vet. Cardiol.*, 20, 364—375. DOI: 10.1016/j.jvc.2018.07.002.
32. **Rush, J. E., 1998:** Therapy of feline hypertrophic cardiomyopathy. *ScienceDirect.*, 20, 627-—634.
33. **Rush, J. E., Freeman, L. M., Fenollosa, N. K., 2002:** Population and survival characteristics of cats with hypertrophic cardiomyopathy: 260 cases (1990—1999). *J. Am. Vet. Med. Assoc.*, 220, 202—207. DOI: 10.2460/javma.2002.220.202.
34. **Sciagrà, R., 2016:** Positron-emission tomography myocardial blood flow quantification in hypertrophic cardiomyopathy. *Q. J. Nucl. Med. Mol. Imaging*, 60, 354—361.
35. **Schipper, T., Van Poucke, M., Sonck, L., Smets, P., Ducatelle, R., Broeckx, B. J. G., Peelman, L. J., 2019:** A feline orthologue of the human MYH7 c.5647G>A (p.(Glu1883Lys)) variant causes hypertrophic cardiomyopathy in a Domestic Shorthair cat. *Eur. J. Human Gen.*, 27, 1724—1730.
36. **Schober, K. E., Zientek, J., Li, X., 2013:** Effect of treatment with atenolol on 5-year survival in cats with preclinical (asymptomatic) hypertrophic cardiomyopathy. *J. Vet. Cardiol.*, 15, 93—104. DOI: 10.1016/j.jvc.2013.03.003.
37. **Schober, K. E., Chetboul, V., 2015:** Echocardiographic evaluation of left ventricular diastolic function in cats: hemodynamic determinants and pattern recognition. *J. Vet. Cardiol.*, 17, 102—133. DOI: 10.1016/j.jvc.2015.02.002.
38. **Vezzosi, T., Schober, K. E., 2019:** Doppler-derived echocardiographic evidence of pulmonary hypertension in cats with left-sided congestive heart failure. *J. Vet. Cardiol.*, 23, 58—68. DOI: 10.1016/j.jvc.2019.01.007.
39. **Ware, W. A., 1999:** Twenty-four-hour ambulatory electrocardiography in normal cats. *J. Vet. Intern. Med.*, 13, 175—180.
40. **Wurtinger, G., Henrich, E., Hildebrandt, N., 2017:** Assessment of a bedside test for N-terminal pro-B-type natriuretic peptide (NT-proBNP) to differentiate cardiac from non-cardiac causes of pleural effusion in cats. *BMC Vet. Res.*, 13, 394. DOI: 10.1186/s12917-017-1319-6.

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