findings. The echocardiography should include not only M-mode and color Doppler, but also spectral Doppler measurements. These measurements are ideally performed by a board certified veterinary cardiologist. Systemic arterial hypertension should be excluded in every cat above the age of 6 years and in every younger cat that show possible signs of chronic renal insufficiency (which may cause hypertension). Besides hypertension there are several other conditions that may lead to the echocardiographic changes that one would expect to see with a HCM. For example hyperthyroidism results not only in an eccentric, but also a mild concentric hypertrophy of the left ventricle. Even more confusing is a congenital mitral valve dysplasia, which can result in identical findings on a physical examination and on an echocardiogram as the obstructive form of HCM. The only way to differentiate these two conditions is to treat the cat with atenolol, as the echocardiographic changes in mitral valve dysplasia are reversible, but those of HOCM are not.

How can dilated cardiomyopathy (DCM) be diagnosed?

Physical examination cannot be used to diagnose DCM. The hallmark of echocardiographic findings is a severe systolic dysfunction of the left ventricle. This can be established based on an enlarged systolic diameter of the left ventricle, which usually also cause a severely reduced fractional shortening. Often there is also a compensatory eccentric hypertrophy of the left ventricle. If the echocardiographic findings fit with DCM, one should always ask himself whether there is a curable underlying condition present. For example DCM caused by taurine deficiency is completely reversible after stopping the deficient diet and apply supplementation. Another example that can resemble of DCM on an echocardiogram is a cat with an end-stage hyperthyroidism. With proper medications these cats can also be cured.

How can hypertrophic cardiomyopathy (HCM) be diagnosed?

Physical examination cannot be used to diagnose HCM. Only the obstructive form of HCM causes a cardiac murmur, so cats with the non-obstructive form of the disease do not usually have any abnormalities on physical exam (i.e. no murmur or gallop sound). The diagnosis of HCM is based on the echocardiographic finding of concentric hypertrophy of the left ventricle, in the absence of a pressure overload. What does this mean in the practice? Either the left ventricular free wall or the interventricular septum must be thickened, i.e. 6 mm or thicker in diastole. How can we exclude pressure overload? A congenital (fixed) aortic stenosis and a dynamic left ventricular outflow tract obstruction (DLVOTO) can be excluded based on physical exam (type of murmur) and based on echocardiographic
create an intraventricular gradient (by causing stenosis) and may also impair the function of the mitral valve. The Burmese breed is clearly predisposed for this disease. The myocardial form of RCM is much more difficult to diagnose, because both ventricles look on ultrasound normal, but both atria are severely dilated. Some cardiologists use the mitral inflow pattern (established with PW-Doppler) and tissue Doppler characteristics of the left ventricle to differentiate this condition from an unclassified cardiomyopathy (UCM), but this question is often academic, as the therapy and prognosis of both conditions are similar.

How can we diagnose arrhythmogenic right ventricular cardiomyopathy (ARVC)?

This is a quite rare condition, which might lead to symptoms of ventricular tachycardia or in a later stage of the disease to a right-sided congestive heart failure. A normal echocardiogram does not rule out the presence of ARVC in a cat with ventricular premature complexes. The end stage of ARVS is characterized by a severe eccentric hypertrophy of the right ventricle. Echocardiographically this condition cannot be differentiated from a tricuspid valve dysplasia. Because feline cardiac disorders are very challenging conditions, the best piece of advice for the practitioners is to refer these patients to a board certified veterinary cardiologist.

**How do we treat hypertrophic cardiomyopathy?**

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The question we should ask ourselves first is not HOW, but WHY should we treat hypertrophic cardiomyopathy (HCM)? Can we cure HCM? Unfortunately not. Currently HCM is still an incurable disease. Why should then we treat all those cats with HCM if we cannot cure them? Well, there are several reasons why a therapy in case of an incurable disease still can be justified. Some of the most important reasons are the following:

- Quality of life could be improved.
- Life expectancy may be longer.
- The asymptomatic stage of the disease may be prolonged.

Before we go into the details of therapy, we must make a number of subgroups of the disease. Depending on which subgroup a cat falls into, the therapy advice will be different.

The first question is: Is the cat symptomatic or asymptomatic?

**ASYMPTOMATIC CATS**

If the cat is asymptomatic, then the HCM is a coincidental finding, and the only reason why we should treat these cats is either to prolong its longevity or the asymptomatic stage of its condition. What should we give? If we try to apply the basic rules of “evidence based medicine” we will get disappointed, because there are no double-blinded, placebo-controlled, prospective clinical trials available that investigate the effect of various medications. How can we suggest to an owner of an asymptomatic cat to give twice daily oral medication to her cat for the coming maybe 10 years? I do not think that we can talk about improvement of the quality of life if the owner has to hunt for the cat twice daily and using all kinds of tricks try to push the pills down the throat of her pet, whereas nobody knows whether the medication guarantees a prolongation of the life expectancy and/or the asymptomatic stage of the disease.

The next question we need to ask ourselves before prescribing any medication is: Does the cat have a left ventricular outflow tract obstruction (LVOTO)? So yes, how severe is it? This parameter can be established during echocardiography. If a severe LVOTO is present (with a pressure gradient exceeding 80 mmHg), atenolol is the first choice. This medication could reduce the severity of LVOTO and can even cure the disease, if the LVOTO is the result of a congenital mitral valve dysplasia, which echographically indistinguishable from an obstructive form of HCM. Because according to research data atenolol works approximately 12 hours in the cat, this medication should be administered...
to the clinical and echocardiographic findings an upward titration may be necessary.

No scientific evidence supports the chronic use of other medications, such as an ACE-inhibitor or an aldosterone receptor antagonist in the asymptomatic stage of HCM.

**SYMPTOMATIC CATS**

What is the recommended treatment for cats with symptoms? It depends which symptoms the cat has.

Because cats with peracute paralyse posterior are most of the time euthanized at the time of the diagnosis, no long-term therapy is generally applicable.

What do we do with cats that are dyspnoic because of congestive left sided heart failure? It depends whether the cause of dyspnea is pulmonary edema or liquothorax. If the cat has pulmonary edema, repeatedly given parenteral furosemide is the only medication that can help, together with rest in an oxygen cage. In case of liquothorax the only way to help cats is to remove as much pleural effusion as possible with a butterfly needle. Cats in congestive heart failure should not be treated with atenolol. Long term maintenance therapy in cats with congestive heart failure is a combination of furosemide and an ACE-inhibitor. The dosage of both medications should be as low as necessary to avoid renal failure and to maintain eupnea. Almost per definition, every cat in heart failure has a big left atrium and thus has an increased chance for having a thrombus formed in the left auricle. Currently no effective medication is known that could prevent thromboembolic complications in the cat. Acetylsalicylic acid has been used for a long time, but its efficacy has never been established. The main concern to use this NSAID together with an ACE-inhibitor and furosemide is the potential to create renal failure. The effect of clopidogrel, a drug with a different working mechanism, in this patient population has not been investigated yet.

Because cats that experience syncope or seizure are diagnostic challenges they should be referred to board certified cardiologists.

**HOW TO RECOGNIZE PULMONARY HYPERTENSION?**

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What is pulmonary hypertension? Pulmonary hypertension is defined as an increased pressure in the pulmonary artery. Pulmonary hypertension is not a single disease, but a condition that can be caused by various underlying disorders.

Which conditions can lead to pulmonary hypertension?
1. Chronic hypoxemia by living in a high altitude or chronic respiratory disorders, like tracheal collapse or lung fibrosis or lung tumor. Hypoxemia causes vasoconstriction of the pulmonary artery branches to reduce ventilation-perfusion mismatch. This is a physiologic response of the body, which eventually may lead to increased pressure in the pulmonary artery.
2. Congestive left-sided heart failure.
3. Congenital large-volume left-to-rights shunts, such as a large patent ductus arteriosus or a large ventricular septum defect.
4. Abnormalities of the pulmonary artery itself, such as a thrombus or embolus, vasculitis, or worms like adults of Dirofilaria immitis and Angiostrongylus vasorum.

Which symptoms can be caused by pulmonary hypertension? Mild pulmonary hypertension may not cause any symptoms. Severe pulmonary hypertension however may lead to the following symptoms: syncope, shock and congestive right sided heart failure (cor pulmonale). Most of these dogs also suffer from dyspnea because the primary disease process that leads to pulmonary hypertension also causes dyspnea. If physical examination and thoracic radiographs fail to reveal the cause of dyspnea, the patient should be immediately referred to a veterinary cardiologist to look for signs of a possible pulmonary hypertension.
How can pulmonary hypertension be diagnosed? The only non-invasive technique that can diagnose pulmonary hypertension is Doppler echocardiography (1). From the velocity of the tricuspid and pulmonary valve insufficiencies the systolic and diastolic pressures of the pulmonary artery can be calculated, respectively. This examination requires the knowledge and experience of a board certified veterinary cardiologist. Supporting evidence is a uniform dilation of the pulmonary artery and an eccentric and concentric hypertrophy of the right ventricle.

What is the prognosis? It depends on the underlying cause of the pulmonary hypertension. If the underlying disease can be cured (e.g. French heartworm infection), the pulmonary hypertension is reversible. In other cases, like a right to left shunting patent ductus arteriosus no definitive cure exists.

What is the therapy? To reduce the pressure in the pulmonary artery a vasodilator should be given that works only on the pulmonic artery branches. The best pulmonary arterial vasodilator is oxygen. If causative therapy is not possible to remove the cause of pulmonary hypertension, various oral medications (phosphodiesterase-V-enzyme inhibitors such as pimobendan and sildenafil) may be added to the oxygen therapy to reduce the pressure in the pulmonary artery. These medications can only be given based on a Doppler ultrasonographic diagnosis of severe pulmonary hypertension to avoid severe adverse effects. Therefore these medications in this setting should only be prescribed by board certified cardiologists.

Reference

In 2009 a group of international leading veterinary cardiologists published guidelines about the diagnostics and therapy of the most common cardiac disease of the dog: the myxomatous valve degeneration of the mitral valve (1).

Myxomatous valve degeneration is a progressive disease. Because every stage of the disease requires a different type of therapy, 4 stages have been differentiated: from A through D.

Stage A: dogs of predisposed breeds without any detectable abnormalities belong to this category. This is for example a young, healthy Cavalier King Charles spaniel without a cardiac murmur. These dogs do not need any therapy, despite the fact that each of these dogs will develop the disease before the age of 10 years.

Stage B: these dogs have a characteristic cardiac murmur (systolic with a point of maximal intensity in the region of the mitral valve), but no clinical symptoms at all. All cardiologists agree that if the heart is not enlarged (on echocardiogram or chest films) these dogs do not need any treatment. However, the board members did not reach a consensus about the dogs in stage B, if the heart is enlarged. Because there is no prove that any medication would be able to prolong the symptom-free period of dogs in this stage, many cardiologists do not recommend any medication. Others do recommend to start with a life long use of an ACE-inhibitor. According to the strongest currently available evidence ACE-inhibitors are unable to delay the onset of congestive heart failure (2). No other medications have been investigated in this stage of the disease yet.
Stage C: these dogs are either having, or have ever had congestive heart failure but thanks to adequate medication lung edema is not any more present. All board members agreed that congestive left sided heart failure (= cardiac lung edema) can only be diagnosed with a combination of physical examination AND thoracic radiographs. The most important abnormalities are: tachypnea with or without dyspnea, tachycardia (with the lack of respiratory sinus arrhythmia) as well as enlarged left atrium in combination with interstitial and/or alveolar pattern on the thoracic radiographs, typically in the hilar region. Before starting any kind of therapy (except for oxygen in cases of a severe pulmonary edema) thoracic radiographs must be taken to evaluate the lungs for the presence or absence of pulmonary edema. The reason for making thoracic radiographs is that coughing and dyspnea can be caused by a number of other disorders than lung edema, even in an elderly small breed dog. Keep in mind that a cardiac murmur may be a coincidental finding by such a patient and the treatment should address the cause of dyspnea (in this case lung edema) and not the murmur! Giving “heart failure medications” to dogs that are suffering from a primary respiratory disorder (e.g. tracheal collapse or pulmonary fibrosis or neoplasia) may have detrimental effects on the patient. Once cardiac lung edema has been confirmed by thoracic radiographs, treatment with parenteral furosemide and oral pimobendan may be initiated. This is a lifelong daily therapy. If the patient is stable and eats well, an ACE-inhibitor may be added as a third medication. The fine tuning of the dosages of the oral maintenance furosemide and ACE-inhibitor should be done based on careful monitoring of the respiratory rate and effort as well as the renal function. As you can see, until this stage the patient can be managed in every first line practice, as the only instrumentations that are necessary to make therapeutic decisions are a stethoscope and thoracic X-ray.

Stage D: these are dogs that do not respond to the highest recommended dosages of the above mentioned “heart failure medications” (i.e. furosemide, pimobendan and an ACE-inhibitor). These dogs need to be referred to a board certified veterinary cardiologist for further diagnostics.

References