VetTalk

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Management of Cardiovascular Disease in the Household Dog

Introduction

As awareness of cardiovascular disease increases in human medicine, there is now a trend of increasing awareness within veterinary medicine as well. This ACVP newsletter will focus on the most common forms of cardiovascular disease and their treatments in dogs with a following newsletter on heart disease in cats in order to make this reading more succinct. The availability of information on this topic can be overwhelming so an overview of cardiovascular disease and the medications used to manage it will be assessed in this article, hopefully creating a better understanding for veterinarians about which agents are most appropriate for their patients depending on which type of heart disease they suffer from.

Prevalence

Cardiovascular disease (CVD) is not just a condition that affects humans, but our four legged patients as well! Heart disease may be due to impaired cardiac filling as seen with mitral and tricuspid valvular stenosis or it may be from dramatically increased afterload such as chronic pulmonary hypertension. Even though diastolic and systolic functions of the heart are related, both tend to be comprised in animals with myocardial disease. The more veterinarians are learning about CVD, the more documentation is being recorded about the prevalence of certain breeds correlating with specific forms of CVD. One example of heart failure is Dilated Cardiomyopathy (DCM) which primarily affects large breed dogs such as Dobermans, Irish Wolfhounds, Great Danes, and Newfoundlands, but also male Dalmatians and American and English Cocker Spaniels. Of these dogs, 50% will develop the arrhythmia Atrial Fibrillation (AFib). Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC) appears to be exclusively represented by the Boxer breed which is why it is also known as Boxer Cardiomyopathy. On the other hand, Myxomatous Mitral Valve Disease (MMVD), which makes up 75% of canine congestive heart failure, is prevalent in smaller breeds such as Cavalier King Charles Spaniels, Papillons, Chihuahuas, Dachshunds, and Toy Poodles. This disease normally affects older dogs with the exception of Cavaliers and Dachshunds who may experience onset of this disease as early as 2 to 4 years of age. As with humans, there are also many congenital heart diseases, but these are often surgically corrected, such as Patent Ductus Arteriosus (PDA) or Tetralogy of Fallot, and are therefore not included in this newsletter as the focus is primarily on pharmacologic management.

Pathophysiology/Etiology

The heart is essential for operation of the circulatory system via ejecting blood into the aorta and pulmonary arteries meeting perfusion requirements of metabolic tissues. It also receives blood from the pulmonary and systemic veins in order to drain capillary beds and maintain appropriate distribution of the circulating blood volume. Cardiac performance is essentially determined by stroke volume and cardiac output including preload, after load, heart rate, myocardial contractility, and ventricular synchrony. Alteration of *any* of these operations can lead to cardiac dysfunction. Cardiac failure (i.e. heart failure) is defined as the physiologic state in which the heart cannot eject

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or receive blood properly resulting in manifestation of erratic blood volume homeostasis and clinical signs and symptoms of heart failure. If cardiac dysfunction causes an accumulation of sodium and water, edema and congestion result in clinically recognized congestive heart failure (CHF). The four main functional classifications of veterinary heart failure per the Textbook of Veterinary Internal Medicine are:

1. Heart failure resulting from impeded cardiac filling
   a. pericardial disease with restricted filling such as constrictive pericarditis or pericardial effusion with tamponade
   b. valvular inflow obstruction such as AV valve stenosis or neoplastic anatomic obstruction
   c. intrinsic myocardial disease with impaired diastolic function such as hypertrophic cardiomyopathy or restrictive cardiomyopathy
2. Heart failure resulting from increased resistance to ejections
   a. increased resistance to the ejection of blood (after load) such as thromboembolism of great vessels, pulmonary hypertension, or discrete outflow tract obstruction
3. Heart failure resulting from impaired ejection or volume overload
   a. primary and secondary myocardial disease with impaired systolic function such as dilated cardiomyopathy or ischemic, infectious, or toxic myocardial disorders
   b. misdirected blood flow resulting in volume overload such as valvular insufficiency or arteriovenous fistulas
   c. chronic high-output states such as thyrotoxicosis or chronic anemia
4. Heart failure resulting from arrhythmias and conduction disorders
   a. sustained tachyarrhythmias such as supraventricular tachycardia and atrial fibrillation
   b. chronic bradyarrhythmias such as complete heart block

Since there are so many nuances involved with CVD, and this newsletter isn't meant to be a textbook on cardiology, these functional classifications will be further examined from a treatment perspective.

Clinical Signs and Symptoms

There are many signs and symptoms of CVD depending on which type of heart disease the dog is afflicted with. Commonly dogs will present with exercise intolerance, dyspnea, coughing, pitting edema, lethargy, cyanosis of the membranes, and may also experience consistent or intermittent syncope. Mitral valve regurgitation will often present in a young male Cavalier with coughing, labored breathing, and exercise intolerance. A depressed, coughing, exercise intolerant Doberman with a rapid, irregular heart beat is likely to have DCM. Sick sinus syndrome often presents in a middle age female Miniature Schnauzer that has intermittent fainting yet a Boxer who also faints is likely to have arrhythmic cardiomyopathy. These problems, if not addressed right away by a veterinarian, can become life threatening just as they would in a human. Cardiovascular disease can lead to pulmonary edema, renal failure, decreased blood flow to critical organs and if not medically managed, will eventually lead to death.

Summary of Clinical Signs

1. New onset lethargy
2. Decreased interest in food
3. Exercise intolerance
4. Coughing or wheezing
5. Increased panting
6. Edema
7. Cyanotic membranes
8. Episodes of unexplained syncope
9. Orthopnea

Diagnosis

The primary means of diagnosing a dog with cardiovascular disease is through a complete history and thorough physical exam, followed by radiographs, electrocardiogram, and/or an echocardiogram. A CBC and Chemistry Panel may be useful for identifying and/or ruling out other disease processes such as diabetes, pancreatitis, renal disease, or an underlying infection. Chest radiographs, an electrocardiograph, and/or an echocardiograph present the practitioner with an even better understanding to what is, or is not, auscultated on physical exam. It is very important to discuss with the owner about when they noticed changes in their pets behavior and keeping good records of when any of these changes become more noticeable with regards to time of day or in relation to certain activities.

Diagnostic Algorithm

1. Assess history, physical exam, and clinical signs
   a. Auscultation of the heart and lungs and assessment of pulses
      i. Detect abnormal sounds such as a gallop, murmur, or thrill
      ii. Heart beat that is muffled as an indication of pericardial effusion in absence of obesity
      iii. Detect tachycardia or

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bradycardia
   iv. Observe rapid, irregular, or faint pulses
b. Mucous membranes cyanotic in absence of primary pulmonary disease
c. Determine from owners about when the change in behavior occurred

2. Rule out iatrogenic disease
   a. Current or past medications
   b. Exposure to possible household toxins (plants, owner medications, household cleansers)
3. Preliminary Lab Results
   a. CBC
      i. Identify a possible underlying infection
   b. Chemistry Profile
      i. Serum chemistry profiles may identify a comorbid disease state such as diabetes or renal disease
4. Radiographs
   a. Identify mitral regurgitation or dilated cardiomyopathy
   b. Detect the degree of enlargement of pulmonary veins
5. Electrocardiography (ECG)
   a. Measures electrical impulses via electrical leads to observe patterns among heartbeats and rhythms
   b. Diagnose rhythm disturbances
      i. Atrial Fibrillation
      ii. Sick Sinus Syndrome
6. Echocardiography (Echo/TTE)
   a. Ultrasound waves or Doppler techniques to produce images of heart
   b. Can confirm tentative diagnosis
   c. Detect enlargement of chamber of the heart and large vessels
      i. Usually more severe the chamber enlargement, the more the disease severity
   d. Detecting cardiac tumors
   e. Detecting pericardial disease

Diagnostic Tests

In order to increase quality of life and extend the patient's overall survival, medical treatment must be implicated as soon as possible. Diagnostic testing should be performed to better understand which form of CVD the animal is experiencing in order to treat and alleviate symptoms and decrease chances of morbidity and mortality.

1. Thorough history and physical exam with auscultation of heart and lungs
2. CBC
3. Chemistry Panel
4. Radiographs
5. ECG
6. Echo or TTE

Treatment

There are several drug classes available for veterinarians to manage CVD in their patients and each class is specific to where in the heart the problem is occurring. Because of the intricacies of cardiovascular disease, the choice of therapy, or therapies, must be individualized to the afflicted animal specifically. Within each drug class are often specific medications that may be chosen over another due to safety, comorbid disease states, side effects, and cost.

ACE Inhibitors

Angiotensin-converting enzyme inhibitor (ACEi) is a group of medications that are typically used to treat hypertension and congestive heart failure. Their mechanism of action is to inhibit the conversion of angiotensin I to angiotensin II which results in vasodilation since angiotensin II stimulates aldosterone that normally causes sodium and water retention that cause congestion and edema. ACEi's also contribute to vasodilation by increasing concentrations of vasodilating kinins and prostaglandins. Commonly used ACEi's in dogs are benazepril, enalapril, and lisinopril though captopril may also be used but it is older and its use has decreased. Ramipril is a newer ACEi and has not been studied as much in animals but appears to have similar pharmacodynamic effects. Benazepril may decrease the likelihood of developing cardiomyopathy in some dogs but studies on this are controversial. Enalapril is also useful to delay the onset of CHF in dogs with mitral regurgitation and is often used with digoxin, furosemide, pimopendan, and spironolactone. The dose of benazepril is 0.25-0.5 mg/kg by mouth every 12 to 24 hours. The dose of captopril in dogs is 1-2 mg/kg by mouth every 8 hours. The dose of enalapril in dogs is 0.5 mg/kg every 12 to 24 hours by mouth. The dose of lisinopril in dogs is 0.5 mg/kg by mouth once daily. The dose of ramipril is 0.125-0.25 mg/kg by mouth once daily. Benazepril, enalapril, lisinopril, and ramipril are generally well tolerated though they can cause azotemia in at-risk patients, especially those on high doses of diuretics. All ACEi's are contraindicated for pregnant bitches.

Antiarrhythmic Agents

There are actually 4 groups of drugs within this class. The first medications discussed are disopyramide and quinidine which are class 1a antiarrhythmics that block inward fast sodium channels and depress myocardial Phase 0 of depolarization to prolong action potential duration in order to control ventricular arrhythmias. Efficacy studies of disopyramide in animals have not been reported and it has a very short half-life in dogs. It is

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being included for sake of completeness. The dose is 7-30 mg/kg by mouth every 4 hours and side effects have not been specifically reported in dogs though in humans adverse reactions are secondary to this medication's anticholinergic effects such as constipation and dry mucous membranes. At higher doses it can induce arrhythmias in human patients. Quinidine has the same mechanism of action as a Class Ia but can also be used to convert atrial fibrillation to sinus rhythm (NSR). The dose of quinidine gluconate is 6-20 mg/kg every 6 hours IM or by mouth while the conversion from AFib to NSR is 6 -11 mg/kg IM every 6 hours and most dogs will convert within 24 hours. Side effects include nausea, vomiting, diarrhea, hypotension, tachycardia, and AV block. Class Ib consists of lidocaine, phenytoin, mexelitine, and tocainde, and they block fast sodium channels and depress Phase 0 of depolarization to treat ventricular arrhythmias. Lidocaine, though the most likely to be used for acute ventricular arrhythmias, would not be sent home with a patient and is therefore will not be discussed in this article. Phenytoin has poor efficacy and questionable absorption but if used the dose is 30 mg/kg by mouth every 8 hours. Side effects are sedation, gingival hyperplasia, skin reactions and CNS toxicity. Mexiletine's use in veterinary medicine is also not common but its dose is 5-8 mg/kg by mouth every 8 to 12 hours. Side effects include excitation, tremors, and vomiting. Tocainide also has limited use in animals but clinical studies do demonstrate efficacy. The dose is 15-20 mg/kg by mouth every 8 hours and side effects include anorexia, vomiting, and ataxia. Class II antiarrhythmics are the Beta Blockers but due to their limited use for heart failure specifically, they are discussed later on with the exception of sotalol. Sotalol is unique in that it is a Class II antiarrhythmic beta blocker (β1, β2) but also a Class III antiarrhythmic as well. Its action is similar to propranolol but also has some potassium channel blocking activity. Similar to atenolol, it is more water soluble and relies less on the liver for clearance. This medication is indicated for the management of refractory ventricular arrhythmias and refractory atrial fibrillation. The dose is 1-2 mg/kg by mouth every 8 to 12 hours though with medium to large breed dogs, typically clinicians begin with 40 mg per dog every 12 hours and increase to 80mg if no response is noted. Side effects include dyspnea, bronchospasm, nausea, vomiting, and proarrhythmic activity. Amiodarone is a Class III antiarrhythmic and its mechanism of action is to block the outward potassium channels in cardiac tissues. It also prolongs the action potential and delays both myocardial repolarization and refractory period in the cardiac tissues. It is used to treat refractory ventricular arrhythmias and is reserved for life-threatening arrhythmias refractory to the treatments. Usually a loading dose is given followed by a maintenance dose and proper monitoring such as ECG and CBC should be performed while the dog is on this treatment due to the array of side effects. An example of an amiodarone dosing regimen for ventricular arrhythmias is 10-15 mg/kg by mouth every 12 hours for 1 week then 5-7.5 mg/kg by mouth every 12 hours for 2 weeks, then 7.5mg/kg by mouth every 24 hours. An example of a dosing regimen for atrial fibrillation is a 15 mg/kg by mouth loading dose for 5 days followed by 10 mg/kg by mouth once daily thereafter. If you must use this medication in a Boxer or Doberman, the dose is 200mg by mouth every 12 hours for 1 week then 200 mg by mouth once daily thereafter. Side effects include decreased appetite, prolongation of the QT interval, bradycardia, CHF, hypotension, AV block, pulmonary fibrosis, neutropenia, thyroid dysfunction, hepatopathy, and anemia. Dobermans appear especially affected by amiodarone experiencing high levels of anorexia, lethargy, hepatic toxicity, and vomiting. Class IV antiarrhythmics are the non-dihydropyridine calcium channel blockers and due to their unique pharmacologic mechanism, will be discussed in another section.

Beta Blockers

These medications work to block beta-adrenergic receptors in order to slow the heart rate and are used to treat supraventricular tachyarrhythmias and premature ventricular contractions. Choices for use in dogs include atenolol, propranolol, and metoprolol. While carvedilol, selective for β1, β2, and α receptors, has been very successful in humans for heart failure, its absorption in dogs is very unpredictable and therefore its use is not recommended. Atenolol is selective for β1 though at higher doses β2 blockade can occur. Metoprolol tartrate is also relatively selective for β1 though at higher doses it can exhibit some β2 blockade. Propranolol is non-selective and targets β1 and β2 in the myocardium, bronchi, and vascular smooth muscle and has membrane stabilizing activity. Both metoprolol and propranolol are lipophilic and therefore cross the blood brain barrier. The dose of atenolol in dogs is 6.25-12.5 mg per dog by mouth every 12 to 24 hours or 0.5-1 mg/kg by mouth every 12 to 24 hours. The dose of metoprolol tartrate in dogs is 0.25-1 mg/kg by mouth every 12 to 24 hours. The dose of propranolol in dogs is 0.1-0.2 mg/kg by mouth every 8 hours. These medications can cause extreme bradycardia and heart block which is why often times the phrase "start low and titrate slow" is heard from pharmacists and when discontinuing therapy, institute a gradual tapering down regimen. The non-selective beta blockers, such as propranolol, may produce bronchospasms in certain patients since β receptors occur in

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lung tissue as well cardiovascular tissue. These medications can also cause lethargy and mask clinical signs associated with hypoglycemia so use these judiciously in patients with diabetes. In any patient with overt heart failure, greater than 1st degree heart block, or sinus bradycardia, these medications are contraindicated. The exceptions to this CHF rule are metoprolol succinate, bisoprolol, and carvedilol but these are rarely used in veterinary medicine and are only mentioned for sake of completeness.

Calcium Channel Blockers (dihydropyridine)

Dihydropyridine calcium channel blockers, amlodipine and nifedipine, are a group of medications that block calcium channels and also act as vasodilators. They decrease calcium influx in cardiac as well as smooth muscle and are normally used to treat systemic hypertension and are often used in combination with a beta blocker. The initial dosing for amlodipine in dogs is 0.1 mg/kg by mouth every 12 to 24 hours. The dose for nifedipine in animals has not been established. Side effects include hypotension and bradycardia though a few cases of gingival hyperplasia have been observed in dogs.

Calcium Channel Blockers (non-dihydropyridine)

These calcium channel blockers, diltiazem and verapamil, are non-dihydropyridines and will block calcium entry into cells by blocking voltage-dependent slow calcium channels. This results in vasodilation, negative chronotropic effects, and negative inotropic effects with a predominant effect on the SA and AV node. These are used for many reasons such as control of supraventricular arrhythmias, systemic hypertension, hypertrophic cardiomyopathy, atrial flutter, AV nodal re-entry arrhythmias, and other forms of tachycardia. The chronic dose of diltiazem for supraventricular tachyarrhythmias is 0.5-1.5 mg/kg by mouth every 8 hours. The dose for diltiazem for atrial fibrillation when used with digoxin (0.0005 mg/kg BID) is 3 mg/kg by mouth every 12 hours. Verapamil is generally given IV as oral formulations are not absorbed sufficiently and for this reason diltiazem is generally preferred in veterinary patients. Nevertheless, the initial dose is 0.05 mg/kg IV every 10-30 minutes for a max cumulative dose of 0.15 mg/kg and the oral dose, per literature, is 1-5 mg/kg by mouth every 8 hours. Side effects generally include bradycardia, lethargy, GI distress, AV block, hypotension, rashes, or elevations in liver function tests. Diltiazem is contraindicated in patients with severe hypotension (<90 mmHg systolic), sick sinus syndrome, or 2nd/3rd degree AV block and should be used with caution in dogs with heart failure.

Cardiac Inotropic Agents

The first group of medications in this drug class are digitoxin and digoxin, though only digoxin is available in the USA. Digoxin, which originates from the Foxglove plant, acts as a cardiac inotropic agent. This means that it increases cardiac contractility while also decreasing heart rate via suppression of the AV node to inhibit re-entrant cardiac arrhythmias. This medication's complicated mechanism of action revolves around its ability to inactivate cardiac muscle sodium-potassium ATPase and increase intracellular calcium. It also has neuroendocrine effects that include sensitization of baroreceptors to decrease heart rate via increasing vagal tone. Its use in dogs for the treatment of heart failure is due to its positive inotropic effects and also for its ability to decrease heart rate. It can also be used to decrease ventricular response for supraventricular arrhythmias as it suppresses the AV node. It is often used in combination with an ACEi and a diuretic such as furosemide. The dose is varied depending on which veterinary cardiologist you speak with or which literary source you refer to. For heart failure alone the starting dose is 0025-.005 mg/kg by mouth every 12 hours. For CHF in the presence of AFib, the dose is 0.005-0.0075 mg/kg by mouth every 12 hours. It is a narrow therapeutic index drug and has the ability to cause a variety of unwanted arrhythmias if not dosed correctly. For these reasons it is appropriate to monitor serum digoxin concentrations in your patient to determine optimum therapy. In dogs, the therapeutic range is 1-2 ng/ml approximately 8 to 10 hours after a dose for treating atrial fibrillation while a more tightly controlled level of 0.8-1.2 ng/ml is recommended for treating heart failure in the presence of AFib or DCM. A maximum level is 2.5 ng/ml is recommended as adverse events are noted above this dose. While determining the ideal dose, a patient can be monitored with an ECG to detect digoxin induced adverse arrhythmias. It can also cause such side effects as vomiting, anorexia, and diarrhea. Dobermans appear to be most sensitive to these adverse effects. It is also important to note that high levels of potassium will decrease digoxin's clinical effects and low potassium will enhance its effects leading to toxicity. The second group in this class consists of one drug that functions as a positive inotrope and vasodilator (inodilator), pimobendan, for use of the management of congestive heart failure and either valvular insufficiency or cardiomyopathy. This medication inhibits phosphodiesterase III and increases intracellular concentrations of cAMP and may inhibit some phosphodiesterase V in the pulmonary circulation. Its inotropic effects are due to its action as a calcium sensitizer to increase troponin C with contractile proteins. The dose is 0.25-0.3 mg/kg by mouth every 12 hours. Side effects include anorexia, lethargy, diarrhea, dyspnea, azotemia, weakness, pleural effusion, syncope,

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sudden death, and ascites. This medication also has the potential risk to be arrhythmogenic, and if furosemide is added, then some activation of the renin angiotensin aldosterone system may occur.

Diuretics

Furosemide is a loop diuretic that exhibits its mechanism of action by inhibiting the sodium-potassium-chloride co-transporter in the ascending loop of Henle to decrease the sodium, chloride, and potassium reabsorption from the tubule. It is the most effective of the diuretics and creates very dilute urine. Furosemide also increases intrarenal prostaglandin production via PGI2 to increase renal blood flow which causes vasodilation in other tissues. One of its uses in dogs is to treat edema cause by heart disease. The dose is 2-6 mg/kg every 8 to 12 hours either IV, IM, SQ or PO. A common starting dose though for heart failure dogs is 2 mg/kg by mouth every 12 hours then lower to 1-2 mg/kg by mouth every 12 hours. Adverse effects are related to its diuretic effects such as loss of fluid creating dehydration and electrolyte losses. Another group of diuretics are chlorothiazide and hydrochlorothiazide, thiazide diuretics which are really just mentioned for sake of completeness as these are not used very often in veterinary medicine as they are sulfonamide analogs and their pharmacokinetics are not well described in animals. Their mechanism of action is to inhibit the sodium-chloride cotransporter in the luminal side of the distal tubule therefore leading to sodium and water diuresis but these medications have far less of a diuretic effect than furosemide. The dose for chlorothiazide is 20-40 mg/kg by mouth every 12 hours. Hydrochlorothiazide is dosed at 2-4 mg/kg by mouth every 12 hours. Side effects are limited to electrolyte imbalance and perhaps reactions in animals allergic to sulfonamides. The third group of diuretics mentioned in this newsletter are the potassium-sparing diuretics, specifically spironolactone and triamterene, which interfere with sodium reabsorption in the distal renal tubule by competitively inhibiting the action of aldosterone and are used to treat congestion caused by heart failure and are often used with an ACEi. The difference between them is that triamterene does not have the competitive inhibiting effect on aldosterone and for treating congestive heart disease in animals, spironolactone is more frequently used. However, the dose of triamterene is 1-2 mg/kg by mouth every 12 hours. The dose for spironolactone is also 1-2 mg/kg by mouth every 12 hours. Side effects of both include electrolyte abnormalities (especially hyperkalemia), dehydration, anorexia, vomiting, lethargy, and ataxia.

Vasodilators

Hydralazine is a vasodilator and antihypertensive agent that relaxes vascular smooth muscle in arteriolar vascular beds and helps reduce vascular resistance and improve cardiac output. The mechanism by which it dilates arterioles and decreases cardiac afterload is not exactly understood but it is used primarily for treatment of CHF, valvular heart disease, and other CVD that has high peripheral vascular resistance. The dose is 0.5 mg/kg by mouth once daily titrated up to 0.5-2 mg/kg by mouth once to twice daily. Side effects include hypotension and dangerously reduced cardiac output. Nitroglycerin is another vasodilator, specifically a nitrovasodilator used for heart failure of pulmonary edema. Its mechanism of action is to relax vascular smooth muscle via generation of nitric oxide. The dose is 4-12 mg topically every 12 hours or the owner may apply ½ inch of 2% ointment for every 5 pounds of body weight to skin without hair (i.e. pinnae of ear or axilla) every 8 hours. The primary side effect is hypotension, rash at site of application, and tolerance that can develop with repeated use. It's very important to advise owners to apply this medication with gloves! Irbesartan and losartan are another group of vasodilators that function as angiotensin receptor blockers (ARB). The metabolism of these medications in dogs is uncertain and it appears that losartan, another ARB, does not show effect in dogs since it does not produce an active metabolite. On the other hand, irbesartan does appear to function though due to cost, it is rarely used and ACEi's are used instead. The dose of irbesartan is 30-60 mg/kg by mouth every 12 hours and side effects are minimal and limited to hypotension.

Prognosis

Cardiovascular disease, once present, will remain a lifetime complication for your patient. If your patient has an arrhythmia, the prognosis is slightly more grim. When an arrhythmia is detected on routine examine, a Holter monitor may be worn so that that veterinarian may interpret the results and evaluate the frequency and complexity of the arrhythmia over the course of 24 hours. Pimobendan has demonstrated a 4 month survival versus placebo in Dobermans with DCM while Fish Oil in Boxers has demonstrated some improvement in ventricular premature contractions (VPC's) and a decrease in syncopal episodes but not a decrease in mortality. For heart failure in the absence of arrhythmias, the outlook is slightly more encouraging. Management focuses on enhancing quality of life by improving clinical signs and increasing survival. Prognosis depends on how the patient presents and what stage they are per the New York Heart Association and International Small Animal Cardiac Health Council guidelines. If they have asymptomatic myocardial disease and are treated right away, then likely they have many years left with their families. However, those dogs that have

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severe and fulminant heart disease, often requiring oxygen therapy, survival may only be a few months and euthanasia may even be the most humane option.

Role of Veterinary Pharmacist

Treating pets with cardiovascular disease offers a unique opportunity for veterinary pharmacists as many of the aforementioned products must be compounded because they are not palatable or in a dose properly concentrated for our patients. While pimobendan is approved for veterinary use and comes in chewable tablets, others such as atenolol and furosemide do not. Often these heart failure patients experience polypharmacy and pharmacists have the ability to address drug compatibility issues. Veterinary pharmacists can even combine medications into one acceptable dosage form improving compliance making life much easier for owners and their pets. In addition to making compounded medications, veterinary pharmacists also need to provide proper counseling on how to administer these medications, such as gloves with nitroglycerin, and what side effects to look for like gingival hyperplasia with amlodipine administration. When dispensing digoxin if the owner is reporting side effects of anorexia and diarrhea in their dog, the veterinary pharmacist may contact the referring DVM to see if the most recent digoxin levels are therapeutic or perhaps the patient does need to come in to have a new blood level drawn. Cost is another area veterinary pharmacists must consider. Perhaps atenolol and enalapril are financially within the budget while pimobendan is just too costly. Pharmacists can work with veterinarians in order to evaluate the best treatment protocol which will properly medicate the patient and also financially benefit their owners. It is also important to consider dosing intervals; owners that work may only be able to give once or twice daily dosing. Being supportive and counseling on the various classes of cardiovascular medications to the best of our ability is critical in earning and keeping our owners trust!

For Further Reading

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