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Congestive heart failure

The Heart

The heart is responsible for pumping blood to all organ in the body.

It highly specialized muscle that is expected to work continuously, without rest for a life time.

The heart have left and right side, each side has 2 chamber: the atrium and ventricle. Special valve divided the chamber and prevent blood from following backward, Where the left atrium receive oxygenated blood than pump it to the left ventricle and then pump to the body throw aorta , Where the right atrium receive un oxygenated blood from the body, than pump it to the right ventricle and finally to the lung throw pulmonary vessel, where the whole cycle start again.(1)

What Is Congestive Heart Failure?

Congestive heart failure (CHF) is a chronic condition that affects the chambers of your heart. You have four heart chambers: two atria in the upper half of the heart and two ventricles in the lower half. The ventricles send blood to your organs and tissues and the atria receive blood as it circulates back from the rest of your body. CHF develops when your ventricles cannot pump blood in sufficient volume. Blood and other fluids back up inside your lungs, abdomen, liver, and lower body.(2)

Systolic Failure

Systolic cardiac failure occurs when normal ventricular filling is accompanied by a decrease in forward stroke volume, reflecting an inherent decrease in myocardial contractility. This may ultimately result in signs of decreased cardiac output such as weakness, hypotension, and compromised organ perfusion. Myocardial failure may be identified on echocardiographic examination as a decrease in ejection fraction or percent fractional shortening, caused by an increase in end-systolic diameter with a normal or increased end-diastolic diameter. However, these indices of systolic function are heavily impacted by ventricular preload, and more advanced imaging options (such as strain imaging or tissue Doppler) may be needed to characterize contractile function in the face of concurrent volume overload. Additionally, regional or diffuse wall thinning and decreased wall motion may be observed, which can be further quantified through use of these more advanced imaging options.

Primary myocardial failure, or idiopathic dilated cardiomyopathy (DCM), is a diagnosis of exclusion. This disease may be noted in several dog breeds but is most commonly seen in Doberman Pinschers, and current research is helping to define a genetic basis for this condition. Idiopathic DCM is also seen rarely in the cat. Some clinicians feel that idiopathic DCM may be the longterm result of unidentified viral infections or myocarditis. While DCM traditionally has been thought of in relation to systolic dysfunction, it is now known that diastolic dysfunction also occurs relatively early in the disease process.

Secondary myocardial failure often results from one or more insults leading to cardiomyocyte damage with subsequent cardiac remodeling and fibrosis. Etiologies include prolonged tachycardia (supraventricular or ventricular tachycardias), infiltrative disease (neoplasia), myocardial infarction, nutritional deficiency (taurine, carnitine, selenium [as seen with white muscle disease]), myocarditis (viral, rickettsial, spirochetal, parasitic, fungal), sepsis, drugs (doxorubicin), toxins (lead, cobalt, gossypol), or rarely endocrine disease (severe hypothyroidism). Additionally, chronic pressure or volume overload states can lead to myocardial remodeling and subsequent failure.

Diastolic Failure

Diastolic failure occurs when elevated ventricular filling pressures accompany normal or compensated systolic function. Elevations in

cardiac filling pressures are transmitted to the pulmonary or systemic circulation, ultimately resulting in transudation of fluid and signs of congestion (edema or effusion). In the absence of pericardial or extracardiac disease leading to ventricular compression or restriction, diastolic dysfunction reflects an inherent abnormality in ventricular relaxation, which may be detected relatively early in cardiac disease processes using Doppler echocardiographic techniques. Diastolic dysfunction may occur in diseases resulting in cardiac compression (pericardial effusion, pericarditis, neoplasia), a stiff or noncompliant ventricle (hypertrophic cardiomyopathy, restrictive cardiomyopathy), myocardial infiltration (neoplasia), or remodeling secondary to chronic volume or pressure overload conditions.Functional CHF may also occur if a tumor or other anatomic obstruction impedes venous return to one or both atria. Pericardial disease or effusion leading to decreased ventricular filling may also be thought of as an extracardiac cause of congestive and subsequent low output heart failure. Iatrogenic volume overload (ie, with aggressive diuresis) can lead to CHF in the absence of primary myocardial systolic or diastolic dysfunction; however, this situation can be thought of as “pseudodiastolic dysfunction” because the ventricle is unable to increase its compliance enough to avoid elevated filling pressures.[3]

Clinical signs

- Tachycardia
- edema in the brisket figure(2).
- the liver is enlarged and in catlle may be palpable.
- The respiration is deeper and rate may be slight increase.(4)
- Weakness, unable to rise
- Decreased appetite
- Collapse
- Bulging eyes
- Distension and pulsation of the jugular vein figure (1)
- Lethargy[6]

Figure(1) jugular vein engorgement

What Are the Most Common Types of CHF?

Left-sided CHF damages your left ventricle (the chamber that pumps blood to the body), and is the most common type of CHF. It can cause fluid to build up in your lungs, which makes breathing difficult.

Right-sided CHF may accompany left-sided CHF. Right-sided CHF is when the right ventricle has difficulty pumping blood to the lungs. Blood builds up in your blood vessels, which causes fluid retention in your lower extremities, abdomen, and other vital organs.[2]

1-Right sided heart failure

The right atrium receives systemic and cardiac venous and lymphatic drainage via the cranial and caudal vena cava and the coronary sinus. Right atrial pressure may increase over time with volume overload this lead to increase the capillary pressure and filtration of fluid across the capillary bed this results in the production of edema in dependent subcutaneous tissue and in body cavities. In kidney the increased pressure to the glomerulus causes increased permeability and escape of plasma protein into the urine. Venous congestion is producing from hepatic congestion and is accompanied by impaired digestion and absorption.[4] Dogs are more likely to develop ascites, while pleural and pericardial effusions are more common in cats. Although the effusions resulting from heart failure are most commonly modified transudates, cats may develop chylous pleural effusion.(3)

- **Clinical sign of R CHF**
- Most commonly results in **ascites** (fluid accumulation within the abdomen)
- or **pleural effusion (**fluid accumulation around the lungs)
- Common clinical signs include:
- Abdominal distention
- Increased respiratory rate or difficulty breathing
- Exercise intolerance
- Fainting[5]

2- Left sided congestive heart failure

The pulmonary veins drain into the left atrium. Left atrial pressure may increase over time in response to volume overload; increase in left atrial

pressure are transmitted to the pulmonary veins to the pulmonary capillaries responsible for alveolar perfusion. Pulmonary capillary hydrostatic pressure continues to increase that promote the transduction of fluid and pulmonary edema.(4) mitral valve stenosis, or elevated left ventricular filling pressures. Increases in left atrial pressures are transmitted to the pulmonary veins and, ultimately, to the pulmonary capillaries responsible for alveolar perfusion. As pulmonary capillary hydrostatic pressure continues to increase, Starling forces promote the transudation of fluid and pulmonary edema develops. In dogs, this may manifest as exercise intolerance, nocturnal or general dyspnea, cough, and tachypnea. Syncope may also be noted, especially in small-breed dogs with chronic valvular disease. This may occur in association with coughing (tussive syncope) or a vasovagal-type response to stimulation of left ventricular mechanoreceptors. Coughing may also be triggered in the absence of pulmonary edema secondary to mainstem bronchial compression from left atrial enlargement or from increased airway responsiveness (so-called “cardiac asthma”).

In cats, the left atrium also receives partial venous drainage from the pleural and pericardial space. Thus, additional clinical signs of left heart failure in cats may include pleural or pericardial effusion; these conditions seem to occur with more frequency in biventricular failure, however. Small volumes of pericardial effusion are common in cats with heart failure and are generally of no hemodynamic consequence (pericardio-centesis is not usually required). Cats with heart failure are less likely to show signs of overt coughing than dogs, and syncope is rare unless associated with development of arrhythmias. Exercise intolerance can be difficult to define in cats because they are generally sedentary. The most common clinical signs noted are inappetence, behavior changes, dyspnea, and tachypnea, which many owners do not detect until heart failure is advanced.[3]

Clinical sign of L CHF

Most commonly results in **pulmonary edema** (fluid accumulation within the lungs)

- Common clinical signs include:
- Increased respiratory rate or difficulty breathing
- Coughing
- Exercise intolerance
- Fainting[5]

Figure (2) Brisket edema

Treatment

The treatment of animal with clinical sign of congestive heart failure due to pericarditis or pericardial tamponade focuses on removing the pericardial fluid and preventing or decreased cardiac output, and chronic neurohormonal adaptations. This is accomplished through preload and/or afterload reduction (diuretics and vasodilators), improving cardiac performance (positive inotropes, lusitropes, sympathomimetics, antiarrhythmics), and use of neurohormonal modulators (ACE inhibitors, and potentially β-blockers, aldosterone antagonists, and angiotensin II blocker).[3][4]

Thoracocentesis

Pleural effusion decreases the available area for alveolar ventilation and thus arterial oxygenation. Thoracocentesis is the most effective treatment in animals with significant volumes of effusion and respiratory distress. However, caution should be taken in particularly stressed patients, which may require pretreatment with oxygen, a moderate furosemide dose, and light sedation. Diuretic therapy is relatively ineffective at resolving large volumes of pleural effusion acutely, and hypovolemia with azotemia is likely to develop if such a treatment strategy is used (ie, administering large enough doses) of diuretics sufficient to significantly reduce pleural effusion.[3]

Abdominocentesis

Ascites may produce abdominal discomfort and worsen dyspnea by reducing available lung capacity. In animals with continued ascites in which increasing diuretic therapy is not an option, abdominocentesis may be performed every 2–4 wk to improve patient comfort and quality of life.[3]

Cardiac Glycosides

The digitalis glycosides (digoxin and digitoxin) are relatively weak inotropes, have a narrow nontoxic therapeutic range, and are associated with significantly more adverse effects than pimobendan. Digitoxin is no longer commercially available. Although used increasingly less for its inotropic effects since the introduction of pimobendan, digoxin still plays an important role in cardiac disease, particularly in atrial fibrillation or supraventricular tachycardia with concurrent CHF, because it is the only

available pharmacologic agent that slows AV nodal conduction without concurrent negative inotropic effects. (For a complete discussion).

Rapid (IV) digitalization commonly results in toxicity and is not recommended. Digoxin may be administered at a conservative starting dose of 0.003–0.005 mg/kg, PO, bid. Adequate serum levels are not achieved for 3–4 days, and a digoxin level should be checked 5–7 days after initiation of therapy, 8 hr after the last dose is given. Further dosage adjustments should be conservative and ultimately based on the animal's serum digoxin level and clinical response. If digoxin is used in cats, it may be started at one-fourth of a 0.125-mg tablet every third day for cats <5 kg, and every other day for cats >5 kg. Some larger cats may ultimately tolerate doses as high as one-fourth of a 0.125-mg tablet, sid. An elixir form is available, although cats generally dislike the taste.

β-Adrenergic Blockers as Cardioprotective Agents

It remains to be seen whether β-blockers will improve morbidity and mortality in animals with cardiac disease, but studies in people indicate that there is sound theoretical and experimental basis to support their efficacy. In animals with occult disease or compensated heart failure, they seem to be well tolerated when careful uptitration is followed, stopping at the highest tolerated dose that does not cause weakness, lethargy, or other clinical signs associated with hypotension or reduced cardiac output. Carvedilol and metoprolol are the most commonly used agents in this setting. Carvedilol is generally started at 0.2–0.4 mg/kg, PO, bid in dogs, and gradually uptitrated every 1–2 wk to a maximal dosage of 1.5 mg/kg, bid. Metoprolol is started at 0.5 mg/kg, PO, bid-tid in dogs, and uptitrated to 1 mg/kg, PO, bid-tid. The recommended dosage of metoprolol in cats is 2–15 mg, PO, tid. Atenolol (6.25–12.5 mg/cat, bid) may also be considered for myocardial protection in cats. There are some data associating the use of atenolol in cats with HCM and CHF with a poorer outcome, and consideration should be given to dosage reduction or withdrawal if CHF develops.

Antiarrhythmics

A detailed discussion of antiarrhythmic therapy is covered elsewhere. Many antiarrhythmics have negative inotropic effects, with the potential to worsen active CHF. This is most likely to occur with the use of calcium channel blockers or β-blockers in the treatment of supraventricular tachyarrhythmias. Therapeutic decisions can be challenging when it is suspected that the presence of a tachyarrhythmia is worsening CHF by reducing the time for ventricular filling during diastole. This is further confounded by the fact that animals in heart failure generally have

elevated sympathetic tone, which can worsen tacharrhythmias. Thus, there is some debate as to whether mild to moderate tachyarrythmias (heart rate of up to 180 bpm) in heart failure should be treated, or simply observed while awaiting better therapeutic control of heart failure.

There is little debate as to whether severe sustained tachyarrhythmias (heart rate >180–200 bpm) should be treated. Treatment of significant ventricular arrhythmias (successive ventricular beats demonstrating R-on-T phenomena) or tachycardia (>160–180 bpm) in CHF is generally attempted with class IB antiarrhythmics (lidocaine or mexilitine) or amiodarone. All of these agents possess minimal or mild negative inotropic effects. Sotalol, a class III antiarrhythmic with β-blocking properties, may also be used, although it possesses more negative inotropic effects and may not be tolerated if significant myocardial dysfunction or CHF are present. Chronic bradyarrhythmias as seen with AV block (high grade second or third degree) or sick sinus syndrome may also lead to CHF, and in these animals, pacemaker implantation is the treatment of choice. If pacemaker implantation is not a viable option, anticholinergics or sympathomimetics may be administered. Propantheline is an oral anticholinergic that is dosed at 0.25–0.5 mg/kg, PO, bid-tid. Adverse effects include tachycardia and GI upset. Theophylline is a nonselective PDE inhibitor with modest chronotropic effects, dosed at 9 mg/kg, PO, tid-qid in dogs, and at 4 mg/kg, PO, bid-tid in cats. A sustained-release formula is also available, which is dosed at 10–15 mg/kg, PO, bid in dogs, and at 20 mg/kg, PO, every 24–48 hr in cats. Adverse effects may include restlessness, excitability, tachycardia, or GI upset. Terbutaline is a β-agonist that also possesses modest chronotropic effects and has similar adverse effects to those seen with theophylline. It is dosed at 1.25–5 mg, PO, tid in dogs, and 0.625 mg, bid in cats. Attempts to overcome clinically significant bradyarrhythmias with oral therapy are often unrewarding, although overall clinical signs may improve in some patients.[3]