STABILIZATION OF THE CONGESTIVE HEART FAILURE PATIENT

J.S. Wohl, DVM, ACVIM, ACVECC D.K. Macintire, DVM, MS, ACVIM, ACVECC
Auburn University Critical Care Program
Auburn University, AL

Pathophysiologic Review

Congestive heart failure refers to a state of vascular congestion and edema associated with increased filling pressures on one or both ventricles. Left heart failure involves congestion of the pulmonary venous circuit and pulmonary edema. The pulmonary venous circuit is a low pressure system and is therefore unable to accommodate severe hemodynamic imbalances. As a result, pulmonary edema can have a rapid onset. Right heart failure involves the systemic venous circuit, which is a high capacitance system for accommodating changes in blood volume and pressure. Thus, congestion has a more insidious onset and a more chronic presentation. Edema due to right heart failure occurs as ascites, pleural effusion, and peripheral edema. Generalized heart failure involves congestion of both the pulmonary and systemic venous circuits.

**Cardiac output (CO)** is the product of heart rate (HR) and stroke volume (CO = HR x SV) and stroke volume is directly related to contractility and inversely related to afterload.

\[
CO = HR \times \{ \text{Contractility x Preload} \} \div \text{Afterload}
\]

In the normal heart, heart rate is the major determinant of cardiac output. However, pathologic changes in contractility, ventricular filling (preload), and afterload affect stroke volume and may limit the ability of heart rate changes to affect cardiac output.

**Preload** is related to the filling pressure on the ventricle during diastole. The force of myocardial contraction is a function of sarcomere length. The length to which fibers are stretched is determined by filling pressure (preload). Within physiologic limits, increases in filling pressure will result in more forceful contractions and increases in stroke volume and cardiac output (Starling’s Law). Venous constriction (causing increased venous return) or volume expansion will therefore increase preload. Decreased venous return, increased intrathoracic pressure (e.g., pneumothorax, positive pressure ventilation), and increased ventricular wall pressure (e.g., pericardial tamponade) will limit preload and cardiac output. Ischemia, fibrosis, incomplete ventricular relaxation (e.g., during tachycardia), and increased ventricular wall thickness will decrease ventricular compliance (distensibility) and limit the effect of preload on cardiac output. In heart disease characterized by chamber dilation, excessive pressures are required to increase ventricular filling. Edema formation then results from increased hydrostatic pressure in the venous circuit.

**Afterload** is the cumulative effect of forces opposing ventricular ejection. Systolic blood pressure is the product of force generated by the myocardium and impedance to blood ejected from the ventricle. Thus increases in afterload need to be matched by increases in preload or stroke output will suffer. However, intrinsic characteristics of chamber pressure, radius, and wall thickness affect afterload as well. Thus, increased preload will enlarge ventricles and increase
afterload. Increasing afterload, forces the heart to expend a greater degree of contractile energy to maintain wall tension leaving less available energy for myofibril shortening and ejection. This can be tolerated in the normal heart but will limit stroke volume during heart disease.

**Contractility** is an intrinsic feature of cardiac muscle that affects the force and velocity of contraction independent of preload and afterload. Chemical and hormonal features regulate cardiac contractility. Calcium released for the sarcoplasm reticulum interacts with troponon and tropomyosin in the cytosol. This interaction induces a reaction between actin and myosin. Calcium reuptake into the sarcoplasm reticulum follows contraction. During heart failure, the conversion of chemical energy to work occurs at a reduced rate. While there is no reduction of intracellular calcium, reuptake of calcium by the sarcoplasm reticulum is diminished. Decreased storage, synthesis, and release of myocardial norepinephrine and decreased density of beta-adrenergic receptors also occur in progressive heart failure and limits contractility.

**Compensatory mechanisms** in heart failure involve the sympathetic nervous system, the renal-adrenal-pituitary axis, and hypertrophic remodeling of cardiac muscle. Decreasing cardiac norepinephrine stores makes the heart more responsive to circulating catecholamines secreted by the adrenal gland. Increased circulating catecholamines mediate alpha-adrenergic vasoconstriction which increases preload (via venoconstriction) but also increases systemic vascular resistance and afterload. As cardiac output diminishes so does renal blood flow. As a result, the renin – angiotensin system is activated and vasoconstriction and increased sodium resorption occurs (via adrenal secretion of aldosterone). This mechanism expands blood volume as a means of augmenting cardiac output. If cardiac function cannot respond to these changes by increasing cardiac output, a positive feedback response ensues where continued decreases in renal blood flow leads to further retention of sodium and fluid. The resulting increase in venous hydrostatic pressure mediates edema formation. Atrial natriuretic protein is an endogenous hormone that dampens rennin and aldosterone secretion, and angiotensin mediated vasoconstriction and may prevent worsening edema formation. Cardiac hypertrophy results from chronic increased wall tension. Myocardial fiber tension is affected by pressure, volume and wall thickness. In response, new myofilaments are generated to distribute forces over a larger cross sectional area. Hypertrophy will initially increase cardiac pump function at normal sarcomere length and wall stress. Concentric hypertrophy occurs in response to pressure overload and myofibrils are generated in parallel formation causing wall thickness to increase relative to chamber size. In response to chronic volume overload, end diastolic volumes are increased and wall thickness is decreased relative to chamber size (eccentric hypertrophy). Eccentric hypertrophy is achieved by slippage of myofibrils within cells and cell elongation.

**Causes of Heart Failure**

Canine cardiomyopathy is the most common example of example of heart failure mediated by myocardial dysfunction. Cardiomyopathy is characterized by decreased ejection fraction, decreased stroke volume, tachycardia, and decreased cardiac output. Initially, the ventricle
operates at a larger end diastolic volume and filling pressure to maintain cardiac output. With
cronicity, ventricular compliance decreases and increasingly higher filling pressures are required
to maintain end diastolic volume and edema formation results. Systolic mechanical overload can be
due to either pressure or volume abnormalities. In pressure overload (eg. aortic stenosis,
pulmonary hypertension) concentric hypertrophy results to compensate for increased afterload.
Ventricular compliance diminishes with concentric hypertrophy and higher filling pressures are
required to maintain end diastolic volume. As concentric hypertrophy progresses, contractility
will eventually diminish and cardiac ouptut becomes more dependent on preload. Thus,
increased diastolic volume in cardiac disease associated with pressure overload indicates advanced
heart disease. In cardiac disease characterized by volume overload ( cardiovascular shunting,
valvular insufficiency) eccentric hypertrophy maintains wall stress and the ventricle initially
becomes more compliant. With progression, contractility is depressed and eventually, cardiac
output cannot be maintained by further increases in preload or hypertrophy. Diastolic
mechanical inhibition occurs in cardiac diseases affecting the ventricular wall or pericardium.
Hypertrophic cardiomyopathy (HCM) in cats and dogs with pericardial effusion experience
decrease venous return and cardiac filling. In cats with HCM, filling pressures on the left side are
exceeding ly high due to a stiff hypertrophied left ventricle. Resulting tachycardia also impedes
cardiac filling and propagates heart failure. Pericardial effusion increases extramural pressure on
the ventricular wall, aming the ventricle less compliant. The right ventricle is more susceptible to
because of lower normal filling pressures. As pericardial pressure approaches ventricular filling
pressure, the drastic decreases in diastolic volumes occur (tamponade) and cardiac output cannot
be maintained.

Clinical Manifestations of heart disease

In animals with heart failure, dyspnea is the most common presenting complaint in
patients presenting to emergency hospitals. Auscultation provides the fist clues in classify
the cause of dyspnea as due to either heart disease or respiratory disease. Dogs with congestive
heart failure are often tachycardic at rest or develop an increased heart rate with mild exercise.
Murmurs, gallops, or arrhythmias are commonly present in heart disease but may also occur in
patients with primary respiratory disease. Lack of a murmur or tachycardia small breed dog
generally rules out congestive heart failure as the cause of dyspnea. Always palpate pulses
simultaneously with auscultation to check for pulse deficits that may indicate arrhythmias. Pulse
deficits (lack of a detectable pulse with each auscultable heart beat) can indicate atrial fibrillation,
premature beats, or ventricular tachycardia. Most animals in heart failure have weak pulses
secondary to decreased stroke volume and increased peripheral vascular resistance. If pulses
seem to get weaker during inspiration, pericardial effusion with pulses paradoxus should be
suspected. Absent pulses and posterior paralysis can be seen in cats with cardiomyopathy and
distal aortic thromboembolism. Murmurs in cats are often heard best low along the sternal
border. Dogs with cardiac disease commonly have a history of coughing (moist, nocturnal),
exercise intolerance, labored breathing, cachexia, and sometimes collapse. Cats rarely have a
history of coughing with cardiac disease. Coughing is more commonly seen with bronchial
asthma and other pulmonary diseases of cats. Pulmonary crackles rather than wheezes are heard with pulmonary edema. Percussion can be used to detect dull areas where there is pleural effusion. Breathing is often labored, exhibiting both inspiratory and expiratory dyspnea, and the respiratory rate is increased.

Mucous membranes may be injected, pale, or cyanotic. Animals with cardiac disease may have an increased hematocrit from chronic hypoxia, resulting in injected membranes. Pallor can be seen with poor perfusion secondary to cardiogenic shock. Cyanosis indicates hypoxemia, secondary to pulmonary edema. Cats with congestive heart failure are often hypothermic and they sometimes have a slow heart rate (100-120) with weak pulses.

Signs of right sided heart failure include jugular venous distension, ascites, pleural effusion, and hepatosplenomegaly. These signs are particularly prominent in dogs with pericardial disease, but can also be seen in dogs with biventricular failure secondary to dilated cardiomyopathy or pure right sided failure associated with congenital heart defects or heartworm disease.

**Management of Congestive Heart Failure**

Emergent presentations of congestive heart disease will generally require a triad of therapy consisting of (1) a diuretic (to decrease preload), (2) oxygen supplementation, and (3) venodilatory therapy. Morphine (0.1-0.2 mg/kg SQ) can be given as initial therapy to dogs that are very anxious and excited. It is anxiolytic, causes mild pulmonary venodilation, and reduces tachycardia. Other features of the management of congestive heart failure may vary with the underlying cardiac disease.

**Mitral regurgitation** is the most common cause of congestive heart failure (CHF) in dogs. Thirty percent of small breed dogs older than 10 years old are affected. Predisposed breeds include Cavalier King Charles Spaniels, Poodles, Miniature Schnauzers, Chihuahuas, Fox Terriers, Cocker Spaniels, Boston Terriers, and Dachshunds. Mitral regurgitation is usually secondary to endocardiosis, a degenerative disease in which the valve leaflets become knobby and thickened. Acute decompensation can occur if chordae tendinae rupture resulting in increased regurgitant fraction, elevated left atrial pressure, and fulminant pulmonary edema. Rarely, acute decompensation occurs with an atrial tear and bleeding into the pericardial sac causing cardiac tamponade. Affected dogs usually have a holosystolic murmur located low at the left 5th intercostals space. Radiographic findings include left atrial enlargement evident as bulging of dorsocaudal border of the heart on the lateral view. A cardiac bulge at 2-3 o’clock on the VD view signifies left auricular enlargement. The appearance of pulmonary edema varies with severity. Early signs of CHF is pulmonary venous congestion seen best in the cranial lobar veins on the lateral view. In dogs, pulmonary edema is perihilar then dorsocaudal, then bilaterally symmetrical with progressive fluid build up. Sever pulomanry edema may appear as fluffly opacities or air bronchograms. Typical electrographic findings include wide P waves (>0.04 s) indicative of left atrial enlargement, tall R waves (> 3 mV in lead II) indicative of left ventricular enlargement and varying degrees of ventricular or supraventricular arrhythmias.

Echocardiography reveals hyperechoic and thickened mitral valve leaflets, levft atria: aorta ratio
greater than 1, normal to increased shortening fraction, and normal appearing ventricular wall motion.

Aggressive therapy is indicated when patients present with severe pulmonary edema, tachypnea, cayanosis, and/or labored breathing at rest. Oxygen supplementation is given immediately by face mask or “flow by” during initial examination and treatment. An intravenous catheter is placed and furosemide is administered. A dosage of 4-8 mg/kg q 1-4 h is used for fulminant pulmonary edema, and 2-4 mg/kg q 8-12 h for maintenance. Try to avoid excessive doses that can result in dehydration, hypovolemia, hypokalemia, and alkalosis. Cats are more sensitive to these side effects, and an initial dosage of 2-4 mg/kg IM, IV or SQ q 8-12 hours is recommended. Topical nitroglycerine (1 inch q 4-6 h) is applied to a shaved or hairless area to induce venuodilation. If the ears are warm, indicating good perfusion, the paste can be applied there. Otherwise, a shaved area on the flank or the inguinal or axillary region should be used. This effect is very rapid. As venous capacitance is increased through vasodilation, pulmonary venous congestion and preload are decreased resulting in less pulmonary edema.

If significant improvement is not seen within 30 minutes following initiation of therapy with oxygen, furosemide, and nitroglycerine, a nitroprusside drip should be initiated. Sodium nitroprusside (Elkins-Sinn, Inc., Cherry Hill, NJ 08003) is a balanced vasodilator which dilates both arteries and veins through direct action on the vessel wall. It also produces pulmonary arterial dilation and produces an increase in stroke volume with no change in heart rate. Because it decreases both afterload and preload, SNP can have very beneficial effects on patients with acute decompensating heart failure and pulmonary edema. Sodium nitroprusside is a very potent vasodilator which has almost immediate effects when delivered as a constant rate infusion. The adverse effects of SNP include hypotension and cyanide toxicity. Hypotension can be avoided by not administering the drug to patients which are hypovolemic or dehydrated, and by beginning at a low dose and gradually increasing the dosage to effective levels while monitoring blood pressure. Cyanide toxicity can occur because SNP is metabolized to cyanide in the body. To prevent toxic levels from developing, the lowest effective dose should be used and the infusion should not be administered past 72 hours. Administration of vitamin B₁₂ may help to promote excretion. Because of these limitations, SNP should be used only for the short term management of acute heart failure patients while they adjust to long term management with digoxin, furosemide, and vasodilators.

The recommended dosage for SNP in the dog is 2-10 µg/kg/min. The following formula can be used to formulate an initial solution at a dosage of 2 µg/kg/min.

\[
M = \frac{(D)(W)(V)}{R \times 16.67}
\]

M = #mg drug needed (SNP is available in 50 mg vials at 10 mg/ml)
D = the dosage in µg/kg/min (in this case 2 µg/kg/min)
W = the body weight in kg (for this example assume our patient weighs 5 kg)
V = volume in ml of base solution (for this example use 100 ml of 5% dextrose solution)
R = rate in ml/hr (typically use a low rate – eg. 5 ml/hr - to avoid volume overload)

Eg. \[ M = \frac{2 \mu g/kg/min (5 kg) (100 ml)}{(5 ml/hr) (16.67)} \]

\[ M = 12 \text{ mg@10 mg/ml} = 1.2 \text{ ml SNP} \]
To make the SNP drip, 1.2 ml SNP is added to 100 ml 5% Dextrose in water at a rate of 5 ml/hr. The patient is monitored q 30 minutes for changes in mucous membrane color, capillary refill time, heart rate, blood pressure, and pulmonary crackles and wheezes. If no improvement is seen, the dosage is increased by 1 µg/kg/min by adding 6 mg (0.6 ml) SNP to the base solution. This is done every 30 minutes until an improvement in pulmonary auscultation and decrease in respiratory rate is noted. The typical effective dosage in dogs is usually between 3-8 µg/kg/min. Therapy with nitroglycerine should be discontinued if a nitroprusside drip is administered. Blood pressure should be monitored during initial therapy to avoid hypotension.

The infusion is usually continued for 24-48 hours until there is no longer radiographic evidence of pulmonary edema and the patient is becoming stabilized on oral medications. During the SNP infusion, furosemide can be administered as needed to control pulmonary edema (2-4 mg/kg IV q 2-8 hr) and oral maintenance doses of digoxin can be initiated if indicated. After the patient is stable, long acting vasodilators such as enalapril can be initiated and the SNP infusion is gradually discontinued. The nitroprusside CRI can be discontinued 30 minutes after oral dosing with enalapril. (Dog: 0.5 mg/kg PO q 12-24 h; Cat: 0.25-0.5 mg/kg PO q 12-24 h). Sodium nitroprusside is sensitive to light and the infusion system must therefore be covered during administration.

Upon stabilization and resolution of pulmonary edema, chronic managemnt strategies should be initiated or continued and consist of diuretic therapy, an angiotensin converting enzyme inhibitor such as enalapril, digoxin therapy if indicated, exercise restriction, and a low sodium diet. Diuretic therapy is provided at the lowest dose to control pulmonary congestion and edema. Furosemide (1-2 mg/kg q 8-48 h ) is the most common diuretic used in the chronic management of congestive heart failure. Spironolactone (2 mg/kg PO q 24 h) is a potassium sparing diuretic that can improve long term response to treatment if given early in the course of therapy by limiting the neurohumeral compensatory responses that lead to volume overload (primarily through the inhibition of aldosterone). Digoxin is indicated if there is marked left ventricular enlargement, tachycardia, atrial arrhythmias, systolic dysfunction as determined by echocardiography, or persistence of clinical signs despite diuretic and vasodilator therapy.

Digoxin reduces sympathetic stimulation of the heart by stabilizing baroreceptor function. It also improves contractility and slows the heart rate by decreasing AV conduction. The Dosage in small breed dogs is 0.0055-0.011 mg/kg q 12 h PO (The large breed dog dosage is 0.22 mg/m² q 12 h PO). The dosage should be based on lean body weight. Digoxin elixir is absorbed more readily than the tablet form. If the elixir is used, 80% of the calculated dose should be administered to avoid toxicity. Generic formulations of digoxin are not recommended. Prior to initiating therapy, blood work should be performed to identify azotemia, hypokalemia, and hypothyroidism and the dosage should be lowered if renal disease, thyroid dysfunction, ascites, or cachexia is present or if concurrent quinidine therapy is used.

Serum digoxin levels should be checked 7 days after initiating therapy 8 hours after the last dose. The therapeutic range for most labs is 0.8-2.2 mg/ml. If values are not in the therapeutic range, the dosage should be adjusted accordingly. Many dogs are sensitive to digoxin toxicity. Signs of digoxin toxicity include anorexia, vomiting, diarrhea, lethargy, and arrhythmias. If these signs occur in a patient receiving digoxin, a blood sample should be taken and digoxin discontinued until the results are available.
**Canine dilated cardiomyopathy** occurs primarily in Doberman Pinchers, “Giant” breeds and other large dogs but can also be seen in Springer Spaniels and American Cocker Spaniels. Boxers have their own breed specific form of cardiomyopathy characterized primarily by severe ventricular arrhythmias and sudden death. If arrhythmias are controlled, the dogs can live 1-3 years before classic signs of dilated cardiomyopathy (cardiomegaly, systolic dysfunction, congestive heart failure) become evident.

Clinical signs of cardiomyopathy include dyspnea, tachypnea, exercise intolerance, weight loss, cachexia, abdominal distension (ascites), and syncope. These signs, even weight loss, can be dramatic and sudden, occurring over a 2-4 week period. Physical examination findings include tachycardia, weak pulses, pulse deficits, soft mitral or tricuspid murmurs secondary to stretching of the annular ring and inability of valve leaflets to close. Muffled heart and lung sounds are noted with pleural effusion, and inspiratory crackles are ausculted with pulmonary edema. Hepatomegaly, splenomegaly, ascites, peripheral edema (“stocking up” of rear limbs), and jugular venous distension are signs of right sided heart failure.

Thoracic radiographs usually reveal severe cardiomegaly with marked dilation of all 4 chambers and diffuse pulmonary edema. Sometimes cardiomegaly is so prominent that it must be differentiated from the globoid appearance of the heart shadow seen with pericardial effusion. Cardiomegaly is not always seen in Boxers with cardiomyopathy. In Dobermans, left atrial enlargement is a prominent finding. Common electrocardiographic findings include tachycardia with tall, wide QRS complexes. Atrial fibrillation is a common arrhythmia seen in large breed dogs with cardiomyopathy while Dobermans and Boxers often have ventricular arrhythmias (eg. premature ventricular contractions or paroxysmal ventricular tachycardia). Echocardiography reveals dilation of atrial and ventricular chambers, thinning of the myocardium, and decreased contractility evidenced by poor wall motion and decreased fractional shortening.

Treatment for the dyspneic patient with cardiomyopathy is similar to the initial management strategy described above. Dogs should receive supplemental oxygen during initial examination and hospitalization. An intravenous catheter is placed and if there is evidence of congestive heart failure (tachypnea, inspiratory crackles, tachycardia, heart murmur), furosemide should be given immediately (2-4 mg/kg IV). The initial database should include blood sampling, ECG, thoracic radiographs, and echocardiography as soon as the patient has stabilized.

Dogs with severe systolic dysfunction and cardiogenic shock may benefit from immediate positive inotropic support.

Dobutamine is the drug of choice for providing immediate positive inotropic effects for short term management of severe myocardial failure. There is evidence in humans with congestive heart failure that dobutamine infusion can result in a sustained improvement in myocardial function even after the drip is discontinued because of replacement of myocardial catecholemines. The dosage is 2.5-20 ug/kg/min and the drip rate should be minimized to avoid exacerbation of volume overload (see CRI formula above). Infusions are initiated at 2.5 ug/kg/min. If tachycardia or exacerbation of atrial fibrillation occurs, the dosage can be decreased. If there is no improvement in pulse quality, mucous membrane color, or capillary refill time, the dosage can be increased. Dopamine is also a positive inotrope, and it is less expensive than dobutamine. Unfortunately, it is more likely to cause arrhythmias and tachycardia and should be monitored closely during infusion. The recommended dosage of dopamine for positive inotropic effects is 5-7 ug/kg/min. Higher dosages can cause marked peripheral vasoconstriction resulting
in increased afterload. Lower dosages (2-3 ug/kg/min) have little effect on the heart, and primarily act to
dilate renal and mesenteric arteries. Amrinone is a positive inotrope that has not been heavily
investigated for veterinary use. It is both a positive inotrope and a vasodilator. The dosage is 1-3 mg/kg
IV bolus followed by 10-100 ug/kg/min CRI titrated to effect. At high dosages it can cause tachycardia
and hypotension. It can also exacerbate atrial fibrillation. Amrinone is incompatible with solutions
containing dextrose.

Digoxin should be started at an oral maintenance dose concurrently with the intravenous positive
inotrope because 3-5 days are required for digoxin to take effect. Large breed dogs generally require less
digoxin on a per weight basis than small breed dogs. It should be dosed according to body surface area:
0.22 mg/m² q 12 h. In general, the dosage should not exceed 0.015 mg/kg/day for large dogs. Doberman
Pinchers are particularly sensitive to digitalis toxicity. A maximum dose of 0.375 mg/day is
recommended for this breed. For cases of rapid atrial fibrillation a double dose can be given for the first
24-48 hours. If ventricular arrhythmias are severe, avoid digoxin until they are controlled. If quinidine is
used, reduce the digoxin dose by 50%. Serum digoxin levels should be checked 5-7 days after initiating
treatment. The therapeutic range is 0.8-2.2 mg/ml 8 h after dosing.

In emergency situations, intravenous nitroprusside should be considered only after positive
inotropic therapy has been implemented, and only in animals that cannot be stabilized with oxygen,
diuretics, and positive inotropic therapy (see above discussion).

Procainamide is the drug of choice for ventricular arrhythmias. Procainamide can cause severe
hypotension if given rapidly IV. In emergency situations, administer 2 mg/kg IV over 3-5 minutes and
follow with 20-50 ug/kg/min CRI. For most other situations, procainamide can be given IM or PO, 8-20
mg/kg q 6 h. The sustained release form is given q 8 h. Other drugs that have been effective in managing
chronic ventricular tachycardia include tocanide (10-20 mg/kg q 8 h PO), mixiletine (5-8 mg/kg q 8 h PO)
or sotalol (2 mg/kg q 12 h PO).

If atrial fibrillation is present and not responding to digoxin, a beta blocker (atenolol, 0.25-1.0
mg/kg PO q 12-24 h) or diltiazem (1.0-1.5 mg/kg q 8 h) can be added 72-96 h after beginning digoxin.
Beta blockers and calcium channel blockers are also negative inotropic agents, thus, lowest possible
doages should be initiated and titrated upward until the resting heart rate is 100-150 bpm.
Once stabilized, patients are managed with chronic therapeutic recommendations including diuretic,
digoxin, low sodium diet, exercise restriction, antiarrhythmics if indicated, and an angiotensin converting
enzyme inhibitor if tolerated. Other potential therapies include Coenzyme Q-10 (2-5 mg/kg PO q 8-12
hr), carnitine (50 mg/kg PO q 8 hr), or taurine (20-25 mg/kg PO q 12 hr).

**Feline hypertrophic cardiomyopathy** is the most common heart disease in cats. Other forms of
feline heart disease include dilated and restrictive cardiomyopathy. Dilated cardiomyopathy (DCM) has
been associated with taurine deficiency. It is fairly uncommon because commercial cat foods have been
adequately supplemented with taurine since 1989 and is usually only seen in cats that eat dog food or
human food. Cats with DCM have generalized cardiomegaly and dilated left atrial and ventricular
chambers on echocardiography. Fractional shortening is decreased (<25-30%). Hypertrophic
cardiomyopathy (HCM) occurs in middle aged cats in good body flesh. A familial form has been
reported in Maine Coon cats. Concentric hypertrophy results in myocardial stiffness and decreased
ventricular lumen size. Radiographically, the heart may appear “valentine” shaped because of left and
right atrial enlargement. Echocardiography reveals thickening of the interventricular septum and/or left ventricular free wall (>6 mm) and left atrial enlargement with decreased ventricular lumen size. Fractional shortening is increased (>45%). In cats ≥10 years of age, hyperthyroidism is a common cause of a potentially reversible form of HCM. In restrictive cardiomyopathy, the ventricle becomes stiff and non-compliant because of myocardial fibrosis. Echocardiography reveals normal ventricular lumen size, normal to decreased shortening fraction, left atrial enlargement and a bright hyperechoic endocardium secondary to fibrosis.

Cardiomyopathy should be suspected in acutely dyspneic cats that have heart murmurs, gallop rhythm, or other arrhythmias. Pleural effusion is common in cats with heart failure and is evidenced by muffled heart and lung sounds accompanied by rapid, shallow respirations. Emergency treatment includes O₂, cage rest, thoracocentesis and diuretics (furosemide 0.5 - 2.2 mg/kg s.i.d. to t.i.d., IV). Cats are sensitive to furosemide and are more prone to developing hypotension, dehydration, and hypokalemia than dogs. Initially a dose of 1 mg/kg can be given q 8 h, but the frequency should be decreased to 1-2 times daily. Nitroglycerine ointment (1/4-1/2" q 6 h) is effective in relieving pulmonary edema in the first 48 hours. Enalapril (0.25 mg/kg SID PO) has shown beneficial results in cats with congestive heart failure. If possible, cats with cardiomyopathy should have an ultrasound examination of the heart to determine whether the underlying condition is hypertrophic or dilated. Plasma taurine levels and serum thyroid hormone levels can be checked in cats with cardiomyopathy. Cats with dilated CM should be given taurine (250-500 mg b.i.d. to t.i.d.). Digitalis is contraindicated in hypertrophic CM, but improves contractility in cats with dilated or restrictive CM. The dosage is 0.0312 mg (1/4 of a 0.125 mg tablet) per cat q 24-48 h. Cats with hypertrophic CM may benefit from atenolol (6.25 - 12.5 mg SID/cat) or diltiazem (1/4 of a 30mg tablet t.i.d.). A sustained release preparation of diltiazem is now available which allows for SID-BID dosing. Each capsule contains four 60 mg tablets. A cat should receive 1/2 tablet twice daily P.O. Aspirin (25 mg/kg every third day) has been recommended to prevent thromboembolism.

Thromboembolism occurs in 25-48% of cats with cardiomyopathy. Aggressive treatment of a recent thrombus is intravenous streptokinase (90,000 IU over 30 minutes, followed by 45,000 IU per hour for 3 hours). Reperfusion injury and life threatening hyperkalemia may occur. Conservative management includes pain control (Butorphanol, 0.2 mg/kg IM in cranial lumber muscles q 8 h and acepromazine 0.1 mg/kg SC q 8 h), heparin (200 IV/kg IV followed by 150-200 IU/kg SC q 8 h) and management of heart failure. Gradual return of function may occur over 7-14 days, and is complete by 6-8 weeks. Prognosis is poor in cats with thrombosis to renal or mesenteric arteries, or with hard, painful limb muscles.