

COMPLICATIONS

CHF Treatment in Dogs

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CHF is a common outcome of degenerative valve disease or dilated cardiomyopathy in dogs. It is characterized by accumulation of fluid in the dependent venous circulation (pulmonary circulation for left-heart disease; systemic circulation for right-heart disease). Treatment of CHF relies on pharmacologic reduction of venous pressure in the dependent (affected) circulation or physical removal of third-space (pleural space, peritoneal space, pericardial space) fluid. In many cases, the clinical condition stabilizes once therapy is optimized; however, the disease inevitably progresses and, in some cases, complications occur.

Complications of CHF can broadly be divided into 2 major categories: (1) progression or relapse of previously stable CHF and (2) treatment-associated (iatrogenic) complications. Relapses are usually associated with progressive pathologic conditions, while treatment-associated complications are the result of adverse drug reactions, toxicity, or inappropriate management.

Prevention

CDVD and DCM are progressive diseases. The rate of progression varies and is not always predictable. As with cancer, treatment of CHF can result in a remission of clinical signs, but relapses are to be expected. Several measures can be taken to help reduce the likelihood or rate of relapse.

Exercise & Stress

Dogs with CHF commonly have exercise intolerance. Strenuous exercise increases circulating catecholamines, which has a negative effect on the diseased cardiovascular system. However, modest, controlled exercise can help reduce afterload by vasodilating the peripheral vascular beds in exercising muscles. Thus, dogs with well-controlled CHF should be encouraged to engage in a mild to moderate level of controlled exercise.

Thermal and psychological stresses also increase cardiovascular workload. Dogs with CHF should be kept in a comfortable (thermoneutral) environment, and situations that induce anxiety or severe excitement should be avoided.

Diet

“Thanksgiving syndrome” or “Superbowl syndrome” is a phenomenon that many emergency clinicians recognize. Dogs with preexistent, controlled, heart disease may present with CHF during these weekends because they have been given access to large quantities of salty food (e.g., turkey skins, chips, ketchup, hot dogs) that create a sudden increase in preload (water retention), which destabilizes the patient. Maintaining a diet with a consistent amount of sodium is more important in managing CHF than a low-salt diet, since the level of sodium affects the amount of diuretic therapy necessary to keep the CHF under control. Sudden increases in sodium intake can precipitate decompensation.



Iatrogenic or Therapeutic Complications

Fluid therapy is generally contraindicated in management of CHF patients. Giving even small volumes of parenteral fluids to a critically balanced, compensated patient can cause a relapse. It is impossible to selectively hydrate certain tissues or systems—if you hydrate the limbs or kidneys, you also hydrate the lungs. Clinicians should resist administering fluids during short anesthetic procedures or during treatment of CHF. Diseases that require fluid therapy (e.g., pancreatitis, severe renal failure) are extremely difficult to treat in the face of CHF and may be grounds for euthanasia.

Drug reactions are best minimized by using the smallest effective dose of medication, closely monitoring the patient at home and in the clinic, and avoiding excessive polypharmacy. Owners should be educated to observe for signs of complications, such as anorexia, adipsia, vomiting, anuria, seizures, and weakness.

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CDVD = chronic degenerative valvular disease; CHF = congestive heart failure; DCM = dilated cardiomyopathy

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Azotemia and uremia secondary to administration of diuretics and ACE inhibitors are among the most common drug-related complications. Because ACE inhibitors decrease glomerular filtration rate, many animals with CHF that are placed on diuretics and ACE inhibitors develop mild azotemia but remain clinically stable. Occasionally, glomerular filtration rate will drop precipitously, resulting in uremia. Evaluation of renal indices (BUN, creatinine) 4 to 7 days after ACE inhibitors and diuretics are started or after doses of these drugs are increased, as well as owner observations of clinical signs, can help alert clinicians to developing complications. Clinicians should be wary of prescribing NSAIDs with ACE inhibitor–diuretic combinations because prostaglandin inhibition of NSAIDs can substantially increase the likelihood of renal complications.

Digoxin Toxicity

Digoxin toxicity is uncommon today because the drug is less popular in managing CHF and is used at lower doses. Monitoring serum digoxin concentrations 6 to 8 hours after administration can help confirm that a dose is appropriate. Keeping the serum concentration at the lower end of the reference range (e.g., 0.5 to 1.0 ng/ml) has been shown in humans to be as effective as higher doses in managing CHF but is associated with less risk for toxicity. It is likely that similar serum concentrations in dogs have suitably beneficial effects and that higher concentrations do not add benefits. Clinicians should carefully evaluate potential drug interactions between digoxin and other drugs, since many agents (e.g., cyclosporine, itraconazole, diltiazem, spironolactone) are known to increase digoxin concentrations.

Beta-blockers have been advocated in treatment of DCM in humans, with documented improvement in cardiac function and survival. Although these observations have not been duplicated in veterinary patients, several groups are evaluating

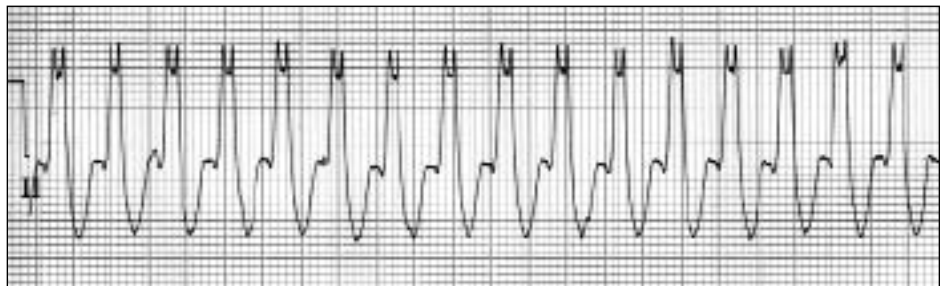


Atrial fibrillation with rapid ventricular response. The rapid response is typically seen with atrial fibrillation secondary to severe cardiac disease and CHF. It results in diminished cardiac output and elevated pulmonary venous pressures and can cause relapse of previously controlled CHF.

this promising treatment. However, beta-blockers may cause decompensation in patients with important cardiac disease and CHF, so clinicians should avoid beta-blockers in this population.

Pulmonary Artery Hypertension

Dogs with chronic left-sided CHF sometimes develop reactive pulmonary artery hypertension; this occurs sporadically without obvious predictors. Pulmonary arterioles constrict excessively, eventually causing hypertrophy of the arterial intima and media. This results in very high pulmonary artery pressure, leading to right-heart failure (although left-heart failure commonly resolves), syncope, and dyspnea.



Ventricular tachycardia. Like atrial fibrillation, ventricular tachycardia can cause decompensation of previously controlled CHF.

Treatment

Some complications cannot easily be prevented and must be addressed when they occur. In most cases, more aggressive diuretic therapy should accompany specific therapy to regain control of CHF.

Arrhythmias

Arrhythmias, most commonly atrial fibrillation, can destabilize previously well-controlled CHF. The decrease in cardiac function accompanied by rapid, unsynchronized, haphazard cardiac contractions often causes relapse. Controlling arrhythmia helps control CHF. Digoxin is usually combined with either beta-blockers or calcium-

ACE = angiotensin-converting enzyme; BPM = beats per minute; BUN = blood urea nitrogen; CDVD = chronic degenerative valvular disease; CHF = congestive heart failure; DCM = dilated cardiomyopathy; NSAIDs = nonsteroidal antiinflammatory drugs

channel blockers to control ventricular response rate (and heart rate). Beta-blockers should be avoided in patients with overt signs of CHF, especially those with DCM. A ventricular rate of 120 to 160 BPM is a reasonable clinical goal, but the ideal ventricular response rate must be established on an individual basis. Although cardioversion to sinus rhythm is usually unrewarding and in most situations impractical, recent reports have suggested that therapy with amiodarone may be effective at converting as many as 35% of dogs to normal sinus rhythm.

Ventricular or atrial tachycardia can also destabilize a patient. Antiarrhythmic selection is dic-

tated by the underlying disease and risk for complications with any particular agent. Combinations of antiarrhythmic drugs might be required in some patients to control the arrhythmia. Atrial tachycardias often respond to diltiazem, while ventricular tachycardias frequently respond in the short-term to lidocaine. Long-term therapy of ventricular tachycardia is more problematic. Mexiletine can be considered but may best be combined with atenolol. Sotalol is also effective, but can have negative inotropic consequences. Amiodarone shows promise—especially for refractory ventricular arrhythmias—but has several potential side effects.

Atrial Rupture

Rarely, a dog with severe CDVD will rupture the left atrium, resulting in acute cardiac tamponade. In many cases, this is an acutely fatal event. Although some dogs survive the initial event, subsequent ruptures are inevitable. Heroic surgical repair of the tear can be attempted but is usually unrewarding.

Relapsing CHF

If no complicating cause can be identified (such as dietary indiscretion, arrhythmias), clinicians should consider the relapse a function of disease progression. Often, simply increasing the

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Selected Drugs Used for Long-Term Management of Canine CHF

<i>Drug</i>	<i>Dose</i>	<i>Potential Side Effects or Toxicity</i>	<i>Monitoring</i>
Lasix	2–4 mg/kg PO Q 8–12 H	Hypokalemia, dehydration, prerenal azotemia	BUN/creatinine levels, appetite, urine output, water consumption
ACE inhibitors	0.25–0.5 mg/kg Q 12 H	Renal insufficiency, GI upset (rare), coughing (rare), hyperkalemia (mild, rare)	BUN/creatinine levels, serum potassium, appetite, urine output, water consumption
Digoxin	0.22 mg/m ² PO Q 12 H	GI upset (common), arrhythmias (various)	Serum digoxin levels, appetite, auscultation; ECG as necessary
Thiazide diuretics	1–2 mg/kg Q 12 H	Hypokalemia, especially if used with digoxin	Serum potassium
Pimobendan	0.25 mg/kg Q 12 H	None reported so far	
Spirolactone	1–2 mg/kg Q 12 H	Hyperkalemia (uncommon)	Serum potassium
Hydralazine	1–3 mg/kg PO Q 12 H	GI upset (common), reflex tachycardia (common), hypotension (uncommon)	Heart rate, blood pressure
Nitrates	Various	Hypotension (theoretical)	

ACE = angiotensin-converting enzyme; BUN = blood urea nitrogen; CHF = congestive heart failure; ECG = electrocardiography; GI = gastrointestinal; PO = orally

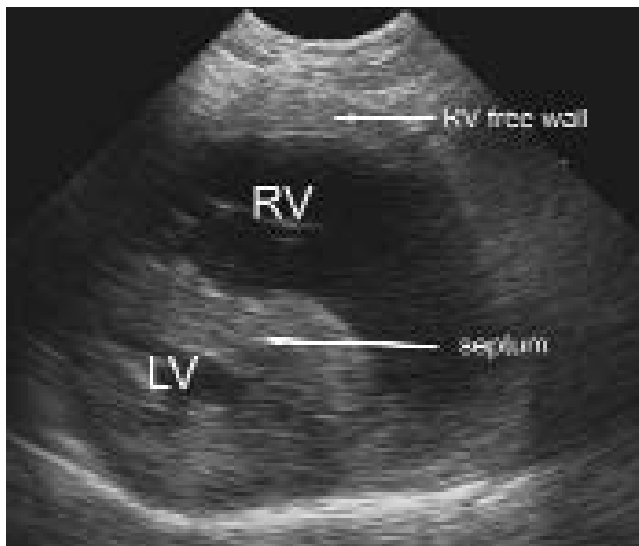
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diuretic dose or dosage is all that is required to control signs of CHF. However, relying simply on ever-increasing doses of diuretics does not constitute optimal therapy. Improving cardiac function by optimizing filling, addressing arrhythmias, improving contractility, and more aggressively offloading the ventricle (arterial vasodilatation) will more likely result in improved survival. If the patient has sharply increased dietary salt intake, the adjustment can be transient, and once stabilized, the patient can often be maintained on the original doses of medications.

Treatment of relapsing CHF should be fairly aggressive—the sooner the condition is resolved, the better. Once controlled, tapering the diuretics can be attempted to find the lowest effective dose.

Combination diuretic therapy (different classes of diuretics) may be more beneficial than therapy with a single diuretic. Recent studies suggest that spironolactone, alone or in conjunction with furosemide, does not noticeably alter urine production in normal dogs. Whether there is an additive effect in dogs with CHF is not known. Thiazide diuretics can also be combined with furosemide to treat relapsing CHF. Clinicians using this combination should watch for hypokalemia.

Pimobendan is an inodilator that has been shown to substantially improve survival in Doberman pinschers with DCM. It also appears to improve quality of life in dogs with CDVD. It is currently unavailable in the United States but can be obtained by petitioning the FDA for permission to import; however, there is a 1- to 2-month delay in processing of petitions due to the high demand for the drug. Pimobendan should be considered in cases of CDVD in which disease progression has resulted in relapse of



Short-axis view of a heart from a dog with severe pulmonary hypertension. The right ventricle (RV) has both volume overload (big chamber) and pressure overload (thick walls), the septum is thick and flattened, and the left ventricle (LV) is small because of volume underloading.

CHF and as a first-line treatment in Dobermans with DCM and CHF.

Diuretic/ACE Inhibitor–Induced Renal Insufficiency

Uremic renal insufficiency caused by diuretic/ACE inhibitor therapy requires cessation of these drugs and careful parenteral rehydration to restore renal perfusion. It is important to note that uremia and severe azotemia are serious complications, not to be confused with the mild azotemia we expect to see with most patients on diuretic/ACE inhibitor therapy. Once the azotemia is controlled, diuretics should be reintroduced (as these are most effective in controlling CHF). ACE inhibitors can be cautiously reintroduced, but some patients cannot tolerate even small doses of these drugs. In such cases, diuretics should be considered the drug of choice in managing the CHF.

Digoxin Toxicity

Digoxin toxicity is treated by stopping the drug. Because digoxin is eliminated by the kidneys, maintaining renal perfusion is important. Antiarrhythmics may need to be administered to control digoxin-induced arrhythmias. In severe cases, specific antidigoxin antibodies can be administered parenterally; however, these agents are very expensive.

Pulmonary Hypertension

Pulmonary hypertension in dogs has no known effective therapy. If caught early enough, left-heart disease treated aggressively can

reduce pulmonary artery pressure (if the vessels are still responsive). If chronic and fixed, pulmonary vasodilators can be used, but anecdotal evidence is not encouraging. Recently, sildenafil (Viagra) has become popular in treating pulmonary artery hypertension, but no trials have documented any clinical benefit. Left-heart failure often resolves, but right-heart failure ensues when pulmonary hypertension develops, necessitating manual removal of fluids (ascites, pleural effusion). ■