Congestive Heart Failure in the Dog

Daniel F. Hogan, DVM, Diplomate ACVIM-Cardiology, Purdue University

Profile

DEFINITION

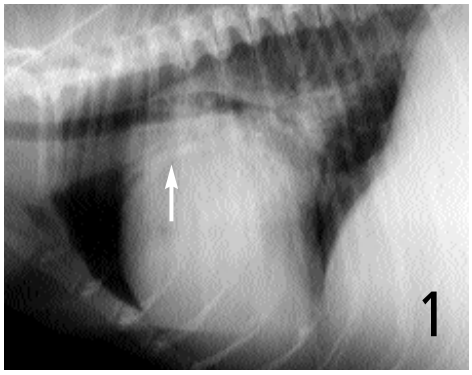
Presence of pulmonary edema caused by an increase in left atrial and pulmonary venous pressures resulting from underlying cardiac disease. (Right-sided CHF often has different causes and therapies may differ.)

SIGNALMENT

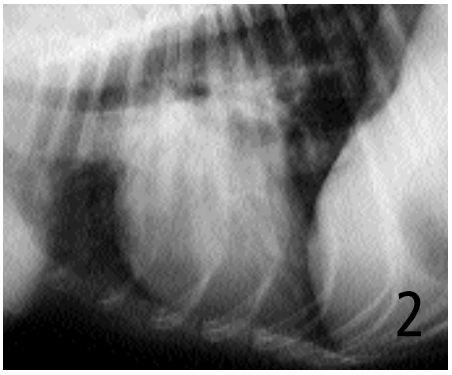
- Certain breeds have been shown to be predisposed to cardiac disease that leads to CHF (see **Breed Predispositions to Cardiac Diseases**).
- CHF resulting from congenital heart disease is most commonly seen in young dogs, although it occurs occasionally in older dogs (**Figure 1**). CHF resulting from acquired cardiac disease is typically seen in middle-aged and older dogs (**Figure 2**).
- Certain congenital diseases have gender differences and DCM appears to have a more rapid progression in male dogs.[1]

CAUSES/RISK FACTORS/ PATHOPHYSIOLOGY

- Congenital heart disease: The most common congenital diseases that can result in CHF include PDA, VSD, and MVD with mitral insufficiency. The reported frequencies of these defects in dogs with CHD are 27.7%, 9.8%, and 1.7% for PDA, VSD, and MVD, respectively.[2]
- Acquired cardiac disease: The most common acquired cardiac diseases that can result in CHF include chronic valvular disease of the mitral valve and DCM. The reported frequencies for chronic valvular disease and DCM in dogs with heart disease are 40% and 11%, respectively.[1]
- Risk factors for acute decompensation include ingestion of a high-salt meal, arrhythmias, and sudden progression of cardiac disease (ruptured mitral valve chordae tendineae, left atrial tear)



Lateral thoracic radiograph from a 10-year-old dog with PDA, chronic valvular disease, and secondary systolic dysfunction. Note the enlarged pulmonary vein of the cranial lung fields.



Lateral thoracic radiograph showing cardiomegaly, left atrial enlargement, pulmonary venous congestion, and pulmonary edema in a dog with chronic valvular disease

Breed Predispositions to Cardiac Diseases

CONGENITAL HEART DISEASE	
Patent ductus arteriosus:	Maltese, toy poodle, miniature poodle, bichon frise, Pomeranian, Yorkshire terrier
Ventricular septal defect:	West Highland white terrier, English springer spaniel, basset hound, English bulldog
Mitral valve disease:	Great Dane, bull terrier, Newfoundland, German shepherd, golden retriever, dalmatian
ACQUIRED HEART DISEASE	
Chronic valvular disease:	Cavalier King Charles spaniel, Chihuahua, miniature schnauzer, Maltese, toy poodle
Dilated cardiomyopathy:	Doberman pinscher, Irish wolfhound, Great Dane, boxer, Saint Bernard, Newfoundland

PAIN INDEX

CHF significantly affects quality of life. It can result in profound discomfort and anxiety and may affect the owner's perception of the pet, resulting in euthanasia.[3]

CHD = congenital heart disease; CHF = congestive heart failure; CVD = chronic valvular disease; DCM = dilated cardiomyopathy; MVD = mitral valve dysplasia; PDA = patent ductus arteriosus; VSD = ventricular septal defect

Diagnosis

The history, physical examination findings, and therapeutic responses are helpful in providing supportive evidence for the diagnosis of CHF. Additional evidence is acquired through imaging.

HISTORY

- Typically includes some degree of coughing and dyspnea
- Exercise intolerance may be seen.
- The owner should be questioned on when the clinical signs were first noted and whether they have improved, worsened, or stayed the same.

PHYSICAL EXAMINATION

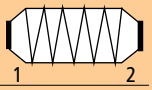
- Pulmonary edema usually manifests as increased respiratory effort and increased sounds on thoracic auscultation.
- Cardiac murmurs are very common (see **Location and Characterization for Cardiac Murmurs**). The most common cause of CHF is chronic valvular disease, and most of these patients have an audible murmur.
- Arterial pulse quality can be affected by the presence of DCM (weak); PDA (bounding); or arrhythmias, such as atrial fibrillation (irregular).
- Mucous membrane color can be altered with CHF because of poor cardiac output (pallor) or severe hypoxemia (cyanosis).

Location and Characterization for Cardiac Murmurs

Mitral regurgitation (MVD, CVD)

left apex

systolic, plateau shaped



Patent Ductus Arteriosus

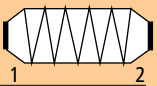
left heart base

continuous

Ventricular Septal Defect

right cranial sternal border

systolic, plateau shaped



1 = first heart sound, 2 = second heart sound

DEFINITIVE DIAGNOSIS

- CHF can only be diagnosed by thoracic radiography as fluid seen within the pulmonary parenchyma.
- There are many causes of noncardiogenic pulmonary edema.
- Definitive diagnosis of CHF can be accomplished through measuring pulmonary capillary wedge pressure. However, this is invasive and rarely done by cardiologists, who usually make a presumptive diagnosis by means of the history, physical examination findings, and supportive evidence obtained through imaging.

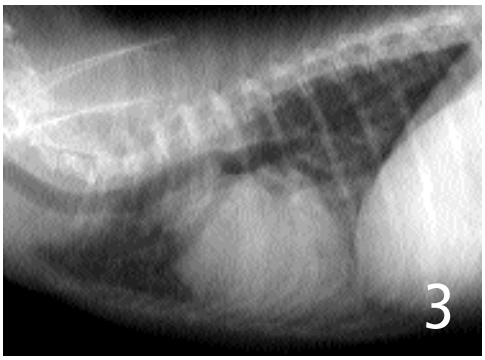
IMAGING

Thoracic radiography

- The classic pattern of cardiogenic pulmonary edema is perihilar, but a patchy or multifocal distribution can be seen.
- Pulmonary venous congestion should always be seen with cardiogenic pulmonary edema. The caudal pulmonary vasculature is best visualized from a dorsoventral projection and may demonstrate the greatest changes.[4] The cranial pulmonary vasculature is best visualized in the lateral projection (**Figures 1** and **2**).
- Comorbid cardiomegaly is supportive of CHF.

Echocardiography

- Left atrial dilatation, left ventricular dilatation, systolic dysfunction, or thickened mitral valve is supportive of organic heart disease and CHF.
- Cardiac defects may be seen with congenital heart disease.
- Cardiogenic pulmonary edema cannot be diagnosed by echocardiography alone.



Lateral thoracic radiograph from a dog with severe respiratory disease showing cardiomegaly, a pulmonary interstitial pattern, and bronchiectasis

DIFFERENTIAL DIAGNOSIS

Respiratory disease

- Coughing or dyspnea arising from respiratory disease is the most common and important differential diagnosis for CHF (**Figure 3**).[5]
- Concurrent respiratory diseases, such as chronic bronchitis and tracheal collapse, are very common in dogs with underlying cardiac conditions.
- Pulmonary edema with cardiomegaly and pulmonary venous congestion should allow an accurate diagnosis of CHF.

Acute respiratory distress syndrome (ARDS)

- Uncommon but serious condition in which pulmonary edema results from damaged capillary walls.[6]
- Closely resembles CHF.
- Estimation of left atrial pressures has been used to differentiate ARDS from CHF.

Pulmonary infiltrative disease

Many possible diseases (fungal, neoplastic, parasitic, inflammatory) can result in coughing and dyspnea. The radiographic patterns of infiltrates are often different from CHF, and cardiomegaly is usually absent. Evidence of systemic disease (lymphadenopathy, fever, inflammatory leukograms) is often present.

continues

Treatment

CHF can be treated in an outpatient or inpatient setting depending on severity. Patients with severe disease that are successfully treated as inpatients can then be managed as outpatients.

- **Activity**—Excessive activity should be avoided. Modest, low-impact activity is generally recognized as beneficial.
- **Client education**—Clients should be made aware that CHF almost always requires life-long medical therapy (see **Tx at a Glance**). Many dogs do well, however, and therapy should be strongly considered.
- **Surgery**—Correction of certain congenital defects, such as PDA, can be accomplished through interventional procedures, such as coil embolization or surgical ligation.[7, 8] Open-heart surgery for the management of chronic valvular disease has been performed in a small number of dogs.[9] Mitral valve repair is being performed at Colorado State and Texas A&M Universities.

Medications[10] & Cost

EMERGENCY THERAPY

Therapy for life-threatening CHF requires aggressive therapy that may need to be adjusted during the acute period.

FOND protocol ($$$$-$$$$$)

Furosemide (50 mg/ml, 2.2–4.4 mg/kg IV Q 1–8 H, CRI: 0.66 mg/kg IV bolus, then 0.66 mg/kg/hr)

- Rapid onset of action; lasts for up to 2 hours. If intravenous access cannot be obtained, intramuscular administration is acceptable but time of onset will be delayed.
- Plasma volume is reduced, decreasing left atrial and pulmonary venous pressures.
- Can be given frequently (up to once every hour) or by CRI but can result in electrolyte derangements.

Oxygen (via nasal catheters, oxygen cage, hood, or mask at 40%–60%)

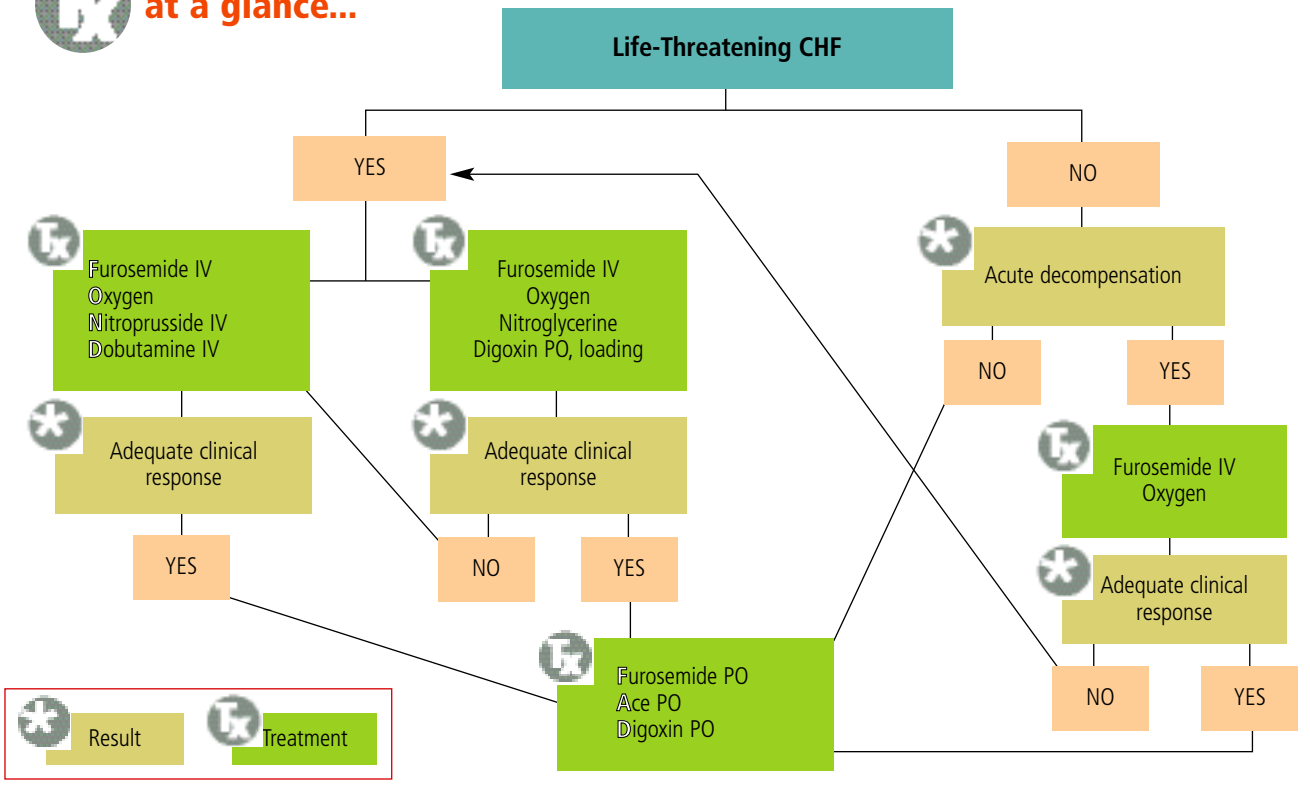
- Pulmonary edema results in hypoxemia. By increasing the fractional percentage of inspired oxygen, hypoxemia can be reduced.

Nitroprusside sodium (2 mg/ml, CRI: 1.0–10 µg/kg/min)

- *Potent* mixed (arterial and venous) vasodilator.
- Venous vasodilatation results in redistribution of the blood volume, reducing pulmonary venous and left atrial pressures.
- Arterial vasodilatation increases cardiac output and reduces the regurgitant

Cost Key:	
$ = <$100	$$$ = $250-500
$$ = $100-250	$$$$ = $500-1000
	$$$$$ = >$1000

at a glance...



volume in dogs with chronic valvular disease.
- Rapid onset of action but short half-life, thereby requiring a constant-rate infusion.
- Dilute in 5% dextrose and protect from light.
- Most significant deleterious effect is hypotension and tachycardia.
- Blood pressure must be monitored during infusion with an ideal goal of reducing mean arterial pressure by 10%–15% (not < 70 mm Hg).
- Use lowest infusion rate and titrate up to effect.
- Hypotension can be corrected by temporarily stopping or slowing infusion.

Dobutamine (12.5 mg/ml, CRI: 5–20 µg/kg/min)
- Inotropic support in severe CHF is critical in dogs with DCM and can be helpful in those with decompensated chronic valvular disease.
- Rapid onset of action and short half-life, thereby requiring a constant-rate infusion once diluted in 5% dextrose.
- Tolerance occurs after 48–72 hours of therapy, although beneficial effects may be sustained for much longer periods.
- Arrhythmias can be seen; electrocardiography should be done.
- Use lowest infusion rate and titrate up to effect.
- Arrhythmias can be corrected by temporarily stopping or slowing infusion.

Modified FOND protocol ($$$-$$$$)

Furosemide (according to the FOND protocol, above)

Oxygen (according to the FOND protocol, above)

Nitroglycerine ointment (2%, 1/2–1 inch on skin Q 8–12 H)
- Organic nitrate that induces venous vasodilatation and decreases pulmonary venous and left atrial pressures.
- Tolerance can develop rapidly, of little concern in emergency setting.

Digoxin (0.003–0.005 mg/kg PO Q 8 H for 48 hr, then Q 12 H)
- Positive inotrope that reduces atrioventricular nodal conduction, decreasing heart rate with atrial fibrillation.
- Therapeutic drug levels can be obtained by an oral loading protocol.

ACUTE, NON–LIFE-THREATENING ($$)

Treat with intravenous furosemide and supplemental oxygen. If clinical response is inadequate within 2 to 3 hours, use FOND or modified FOND protocol.

NONEMERGENCY THERAPY ($/MO)

Chronic CHF can be managed with FAD protocol:

Furosemide—1.1–4.4 mg/kg PO Q 8–24 H

ACE inhibitor
- Enalapril—0.5 mg/kg PO Q 12 H
- Benazepril—0.25–0.5 mg/kg PO Q 12–24 H
- Lisinopril—0.5 mg/kg PO Q 12–24 H
- Ramipril—0.5 mg/kg PO Q 12–24 H

Digoxin—Can be used early even in dogs that do not have systolic failure for neurohumoral-modulating effects.
- 0.003–0.005 mg/kg PO Q 12 H
- 0.22 mg/m^2 PO Q 12 H

Follow-Up

PATIENT MONITORING

- Check renal and electrolyte values 3 to 5 days after beginning furosemide and ACE inhibitor therapy.
- Measure digoxin levels after 5 to 7 days of therapy (6–8 hr after dosing); 1–2 ng/ml is considered therapeutic, but lower levels may be beneficial.
- Evaluate parameters every 6 months or if inappetence, vomiting, or diarrhea develops.[11]

COMPLICATIONS

- Dogs on long-term FAD therapy may present with vomiting, dehydration, and azotemia.
- Measure digoxin levels and discontinue digoxin and ACE inhibitor therapy.
- Reduce the furosemide dose by half and initiate minimal fluid therapy (0.25–0.5 times maintenance requirements).
- After recovery, start drugs back at 50% of the previous dose.
- Monitor renal values and titrate digoxin dose based on serum levels.

COURSE

Many if not all CHF dogs decompensate as the disease progresses. Counteracting decompensation may require increasing doses of furosemide and adding other diuretics or vasodilators.

In General

PROGNOSIS

The length of survival depends on the cause and severity of the underlying cardiac disease and the response to therapy. Six months to 2 years is reported in the literature, and although this seems vague, it accurately reflects the heterogeneity of CHF in dogs.

See Aids & Resources, back page, for references, contacts, and appendices.

ACE = angiotensin-converting enzyme inhibitor; CHF = congestive heart failure; CRI = constant-rate infusion; DCM = dilated cardiomyopathy; MO = month; PDA = patent ductus arteriosus