Congestive heart failure in the dog

KEY POINTS
- Thoracic radiographs are essential to definitively diagnose congestive heart failure (CHF) (12).
- For CHF, high doses of furosemide, either as a bolus or as a CRI, are often effective.
- In severe CHF, either sodium nitroprusside or intubation and intermittent positive pressure ventilation may be successful.

DEFINITION / OVERVIEW
CHF may be defined as the presence of fluid accumulation in the lungs (pulmonary edema), pleural space (pleural effusion), abdominal cavity (ascites), or pericardial sac (pericardial effusion) due to cardiac dysfunction. Cardiogenic fluid accumulation only occurs in dogs with elevated diastolic ventricular and/or atrial filling pressures. Left-sided CHF develops in dogs with elevated left heart filling pressures and leads to pulmonary edema with or without small-volume pleural effusion. Right-sided CHF occurs in dogs with elevated right heart filling pressures and is manifest as ascites with or without pleural effusion. Most dogs with large-volume pleural effusion have biventricular heart failure with elevated right and left heart filling pressures.

ETIOLOGY AND RISK FACTORS
The most common causes of CHF in the dog are acquired chronic valvular disease and dilated cardiomyopathy. Acquired chronic valvular disease, or endocardiosis, primarily affects the mitral valve, although up to one third of affected dogs have both mitral and tricuspid regurgitation. Pericardial effusion is also a cause of CHF, usually in large-breed dogs. Dogs with congenital heart disease may also develop CHF, as can dogs with bacterial endocarditis and a variety of uncommon cardiac disorders.

PATHOPHYSIOLOGY
CHF develops after ventricular diastolic filling pressures rise to abnormally high levels. This elevated pressure is transmitted back to the venous system and the elevated capillary pressures lead to fluid exudation into the interstitium and edema formation. Left-sided CHF, most commonly observed as pulmonary edema in dogs (13), often develops after the left ventricular or left atrial filling pressures rise above 15–20 mmHg. Right-sided CHF, recognized as ascites and/or pleural effusion, develops in most dogs once right heart filling pressures rise above 10–12 mmHg. In some dogs, chronic left heart failure leads to elevations in pulmonary arterial pressures and biventricular heart failure develops. Biventricular heart failure is identified as combined pulmonary edema, pleural effusion, and ascites.

Large- and giant-breed dogs are predisposed to dilated cardiomyopathy. The condition has also been recognized in Cocker Spaniels. Small and medium-size breeds of dogs are predisposed to chronic valvular disease. CHF is more usual in middle-aged to older dogs, but can develop as a result of congenital disease; dilated cardiomyopathy can be seen within the first few years of life. In general, there is a slight predisposition for the development of CHF in male dogs.

Algorithm for congestive heart failure.

12 A Shih-tzu with severe pulmonary edema due to heart failure. Note the expectorated pulmonary edema.
CLINICAL PRESENTATION

Historical signs
Cough is the most common presenting complaint for dogs with CHF. Additional historical complaints for dogs with CHF may include tachypnea or dyspnea, syncope, lethargy or exercise intolerance, abdominal distention, anorexia, and weight loss. While some dogs have slow development of clinical signs, it is common for clinical signs to appear more acutely.

Physical examination findings
Dyspnea, cough, and ascites may be noted. Femoral arterial pulses are often weak and the jugular vein is typically distended above the bottom third of the neck in dogs with right-sided or biventricular heart failure. Pulmonary crackles are often present on auscultation in dogs with pulmonary edema, and dogs with pleural effusion may have dull lung sounds ventrally. CHF in the dog is often associated with an S3 gallop (14). A murmur of mitral or tricuspid valve regurgitation is the most frequent murmur noted on auscultation, and the murmur is often loud in dogs with chronic valvular disease and soft in dogs with dilated cardiomyopathy. Arrhythmias with pulse deficits, mucous membrane pallor, or delayed capillary refill time may also be noted. Some dogs with CHF have a recent unplanned weight loss.

DIFFERENTIAL DIAGNOSES
Differential diagnoses include collapsing trachea, pneumonia, chylothorax, various forms of primary or metastatic neoplasia, diaphragmatic hernia, and bronchitis. In the author’s practice, bronchitis is an infrequent diagnosis in mature to older large-breed dogs, and dilated cardiomyopathy with mild CHF should be a key differential in this setting.

DIAGNOSIS
CHF can be reliably diagnosed based on a few key clinical findings. It is worth noting that echocardiography alone is generally not sufficient to diagnose CHF, and auscultation of the lungs for pulmonary crackles is also an unreliable method for diagnosing CHF.

Radiography
The key findings on thoracic radiographs (15) that can lead to a diagnosis of CHF are:
- Cardiomegaly.
- Pulmonary venous distention.
- Caudal vena cava distention.
- Perihilar pulmonary infiltration.
In dogs, the first radiographic evidence of left-sided CHF is an interstitial pattern, which can be difficult to distinguish from the aging pulmonary interstitial changes that are seen in many dogs. Resolution of this interstitial pattern following furosemide administration can be a method for distinguishing the two clinical entities. As CHF progresses, a bronchial pattern may be noted in many medium- to large-breed dogs; this is followed by overt alveolar flooding, which results in radiographic air bronchograms. Pleural effusion or ascites is usually evident in dogs with biventricular or right-sided CHF.

Additional testing
Additional diagnostic testing for dogs suspected of having CHF includes an ECG and an echocardiogram. Baseline laboratory testing, including a CBC and serum biochemistry profile with electrolytes, is also recommended. NT-pro BNP measurement may be useful in evaluating animals for suspected CHF.

Electrocardiogram
Findings from the ECG are not specific for CHF but can include a left atrial or left ventricular enlargement pattern, conduction disturbances, such as bundle branch block, and cardiac arrhythmias are common (16). Supraventricular arrhythmias are often present in dogs with chronic valvular disease, while atrial fibrillation and/or ventricular arrhythmias are more common in dogs with dilated cardiomyopathy.

Serum biochemistry
Modest elevations of BUN or creatinine may result from prerenal azotemia due to inadequate cardiac output or prior diuretic administration, elevated liver enzymes may be noted due to chronic passive hepatic congestion, and mild hypoproteinemia is common in dogs with ascites.
**Echocardiography**

The key echocardiographic finding to confirm a diagnosis of cardiogenic pulmonary edema in dogs is dilation of the left atrium. In dogs suspected to have right-sided CHF, dilation of the right atrium should be easily visualized, except in dogs with pericardial effusion. A variety of additional findings may be present and are usually specific to the type of heart disease that has led to CHF (17, 18).

**MANAGEMENT / TREATMENT**

**Initial emergency management**

Emergency management of CHF usually comprises oxygen therapy, high doses of diuretics, and nitrates. Theracventosens should be performed in dogs with pleural effusion that is of sufficient volume that it likely contributes to dyspnea. Dogs with large-volume ascites may benefit from abdominocentesis, especially if the ascitic fluid is limiting respiratory effort or lung volume. Caffeine is indicated, and supplemental oxygen can be administered through a number of methods.

**Furosemide**

Furosemide is the most commonly used diuretic in dogs and it can be administered in high doses, up to 4 mg/kg IV every hour, until relief of dyspnea is evident. There is recent enthusiasm for administration of furosemide via a CRI. A CRI of furosemide can be dosed at 0.1–1 mg/kg/hr. Injectable furosemide is diluted to a concentration of 10 mg/ml in either 5% dextrose in water or 0.9% NaCl.

**Nitroglycerine**

Nitroglycerine (glyceryl trinitrate) can be administered transcutaneously to the inner surface of the ear pinna, the inguinal region, or even smeared directly onto the oral mucous membranes. The 2% paste formulation can be dosed at 6–12 mm (0.25–0.5 inch) of paste for every 5 kg (11 lb) body weight.

**Additional treatment**

When these initial measures for emergency management of CHF are ineffective then sodium nitroprusside, dobutamine, or mechanical ventilation can be used.

Sodium nitroprusside

The most effective drug for dogs with severe pulmonary edema refractory to standard treatment is sodium nitroprusside. This drug is used for 1–3 days while other therapies for management of CHF are being initiated. Sodium nitroprusside is administered as a CRI at 1–5 µg/kg/min in 5% dextrose in water. Blood pressure measurement is desirable as a dramatic drop in blood pressure is possible and systolic blood pressure <70–90 mmHg should be avoided.

Dobutamine

Dobutamine at 1–10 µg/kg/min is recommended for dogs with refractory CHF associated with decreased cardiac function, such as with dilated cardiomyopathy. An initial CRI at 1–2 µg/kg/min is titrated upwards every 30 min until adequate clinical response or side effects are noted. Side effects can include sinus tachycardia, supraventricular or ventricular tachyarrhythmias, and GI side effects.

**Mechanical ventilation**

In selected cases, mechanical ventilation can be a very successful adjunct to the other therapies. Mechanical ventilation with use of positive end-expiratory pressure should be considered in any dog judged to be at imminent risk for CPA. It allows for control of the airway and avoidance of respiratory failure leading to respiratory arrest while other therapies are being performed. Positive end-expiratory pressure is useful in helping to clear pulmonary edema. Mechanical ventilation requires adequate equipment in addition to ventilator skills and a significant commitment of time and resources.

**Long-term management**

For chronic management of CHF, exercise limitation, dietary sodium restriction, diuretic therapy, and ACE inhibitors usually form the backbone of therapy. Cough, often due to left atrial enlargement, may be a troublesome long-term management concern (19, 20).

**PROGNOSIS**

With the exception of easily correctable congenital defects, the prognosis for dogs with CHF is always guarded. Many dogs respond well to initiation of medications and dietary recommendations, while others fail to respond or encounter repeated side effects or bouts of CHF. To some degree, the dedication of the owner and the owner’s financial means can play a big role in the outcome for dogs with CHF. While a 2-year survival is possible for some dogs with chronic valvular disease, a 6-month to 1-year survival might be more typical once CHF has developed.

In dogs with dilated cardiomyopathy the survival is often shorter than that of dogs with mitral regurgitation due to chronic valvular disease. In one study, the median survival was only 2 months, and several studies have identified a particularly short survival time for Doberman Pinschers. Still, with dedicated owners who are willing to make several adjustments to therapy, survival beyond 6 months is possible for many dogs with dilated cardiomyopathy. For dogs with CHF due to uncorrectable congenital heart disease the long-term survival is often only a few months with medical therapy alone.
Congestive heart failure in the cat

KEY POINTS
- Cats with severe pulmonary edema due to CHF are unstable and prone to stress. Stressful maneuvers, such as phlebotomy and catheter placement, should be delayed for several hours.
- The radiographic location of pulmonary edema can be variable in cats with CHF.
- Many cats with CHF can survive well beyond 1 year following initiation of successful management.
- ACE inhibitors are indicated for cats with CHF.
- Ultrasonography in the emergency room can be useful to identify the presence of pleural effusion or left atrial enlargement.

DEFINITION / OVERVIEW
CHF can be defined as the presence of fluid accumulation in the lungs (pulmonary edema), pleural space (pleural effusion), abdominal cavity (ascites), or pericardial sac (pericardial effusion), due to cardiac failure. Cardiogenic fluid accumulation occurs in cats with elevated diastolic ventricular and/or atrial filling pressures. Left-sided CHF develops in cats with elevated left heart filling pressures and leads to pulmonary edema with or without a small to moderate volume of pleural effusion and, rarely, pericardial effusion. Right-sided CHF occurs in cats with elevated right heart filling pressures. Most cats with large-volume pleural effusion have biventricular heart failure with elevated right and left heart filling pressures.

ETIOLOGY AND RISK FACTORS
Common causes of CHF in the cat are hypertrophic (21), restrictive, or dilated cardiomyopathy and, less frequently, congenital heart disease, such as ventricular septal defect, patent ductus arteriosus, and mitral or tricuspid valve dysplasia. Endocarditis is an uncommon cause of CHF. Some cats with heartworm disease may develop CHF.

Breed at increased risk for hypertrophic cardiomyopathy include Maine Coon cat, Norwegian Forest cat, Persian, and American Short Hair cat. Middle-aged, male cats are predisposed to hypertrophic cardiomyopathy, and surveys of cats with hypertrophic cardiomyopathy indicate that affected cats have a higher median weight than unaffected cats. Recent corticosteroid administration, especially long-acting formulations of prednisone (prednisolone), can precipitate CHF in otherwise compensated individuals. Trauma, intravenous fluids, and recent surgery or anesthesia with ketamine can also predispose to the development of CHF.

PATHOPHYSIOLOGY
The pathophysiology of CHF in cats is similar to that described for CHF in the dog. CHF develops following a rise in ventricular diastolic filling pressures, which is transmitted back to the pulmonary veins or systemic veins. This leads to elevated capillary pressures and edema formation. Left-sided CHF is most commonly seen as pulmonary edema in cats, although a small to moderate volume of pleural effusion develops in some cats. Right-sided CHF is clinically observed as ascites and/or pleural effusion in cats. Biventricular heart failure leads to pleural effusion with or without pulmonary edema and small-volume ascites. Large-volume ascites of cardiogenic origin in cats is uncommon.

CLINICAL PRESENTATION
History
The history provided in most cats with CHF is often an indicator of congenital heart disease. The history provided in most cats with CHF is acute illness of 1–3 days’ duration. Dyspnea is recognized by some owners; however, lethargy, reduced food intake, and limited interaction with family members (e.g. hiding in the closet) are the predominant abnormalities noted in many cases. Overt dyspnea in some cats is not apparent until the travel to or visit at the veterinarian. Additional possible historical complaints for cats with CHF are syncope, intermittent open-mouth breathing, reduced exercise tolerance before open-mouth breathing, abdominal distention, and, occasionally, weight loss in cats with pleural effusion or ascites.

Cough is an uncommon presenting complaint for cats with CHF. Feline asthma, heartworm, or lungworm should be considered as more likely diagnoses in cats with cough.

Physical examination findings
Dyspnea is often the most prominent initial finding on initial evaluation of cats with CHF. Harsh lung sounds with pulmonary crackles are typically present in cats with pulmonary edema. Cats with pleural effusion of cardiogenic origin often have muffled lung sounds on the ventral thorax, hepatomegaly, and jugular vein distention. Femoral arterial pulses are often weak and mucous membranes can be pale or cyanotic. Rectal temperature is often low.

Auscultation of the heart can be an important contributor to the diagnosis of CHF as a soft murmur, a gallop, or arrhythmia is often present (22). Cardiac murmurs in most feline heart disease are best transmitted to the sternal border. A loud (IV/VI or louder) murmur in a young cat with CHF is often an indicator of congenital heart disease.

DIAGNOSTIC MANEUVERS
- Auscultation of the heart can be an important contributor to the diagnosis of CHF as a soft murmur, a gallop, or arrhythmia is often present.

21 Necropsy specimen (21a) and two-dimensional echocardiogram (21b) demonstrating a short-axis cross-section of the heart from a cat with CHF due to hypertrophic cardiomyopathy. There is marked hypertrophy of the interventricular septum (b) and left ventricular free wall (d), and the left ventricular internal lumen (c) is decreased in size. Right ventricle = a.

22 Schematic representation of typical auscultation findings from a cat with cardiomyopathy. The first and second heart sounds are normal (S1 and S2), with S1 occurring shortly after the onset of the QRS complex and S2 occurring near the end of the T wave. The S4 gallop is present in late diastole, after the P wave, and is the result of atrial contraction of blood into a stiff and hypertrophied ventricle. The gallop is often heard best using the bell of the stethoscope. Many cats have a soft, systolic cardiac murmur near the left or right sternal border and the murmur is depicted by the blue band between S1 and S2.