Left-Sided Congestive Heart Failure in Dogs: Pathophysiology and Diagnosis*

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ABSTRACT: Congestive heart failure (CHF) is a relatively common condition in dogs. Diagnostic and treatment strategies continue to evolve as researchers explore the pathophysiology and develop a better understanding of this syndrome. Unfortunately, however, the morbidity and mortality associated with heart failure remain high. This article focuses on the pathophysiology of CHF and the diagnosis of dogs that present either with initial clinical signs or with decompensation despite therapy.

Heart failure is not a disease per se. Rather, it is a complex pathophysiologic syndrome resulting in the clinical manifestation known as heart failure.

Traditionally, congestive heart failure (CHF) is defined as systolic and/or diastolic failure of the left heart and activation of neurohormonal mechanisms resulting in cardiogenic pulmonary edema. Pulmonary edema is not observed in dogs with right-sided failure. Veterinarians must rely on characteristic clinical signs and presenting complaints such as a moist cough, exercise intolerance, respiratory distress, tachypnea, and syncope to help make a diagnosis of pulmonary edema secondary to CHF. However, these clinical signs can also be observed in dogs with tracheal collapse, heartworm disease, pulmonary parenchymal disease, or pulmonary hypertension.

Unlike human medicine, in veterinary medicine, a failing heart and decreased forward flow cannot be attributed solely to myocardial failure. Other causes of cardiac injury and myocardial dysfunction can lead to abnormal function resulting in heart failure.

PATHOPHYSIOLOGY
The pathophysiology of heart failure can be divided into three separate, yet intertwined, mechanisms: (1) myocardial failure, (2) volume or pressure overload, and (3) compliance failure.

Myocardial failure typically results when the myocardium loses normal systolic contractile and diastolic relaxation functions. Dysfunction of the myocardial sarcoplasmic reticulum results in decreased calcium binding and release, thereby making less calcium available to the cardiac myocyte during contraction (systole).
During diastole, elevations in intracellular calcium in the diseased sarcomere cause decreased myocardial relaxation. Conditions that tend to be associated with myocardial failure include dilated cardiomyopathy, ventricular septal defects, and aortic stenosis.

Volume overload is a common reason that dogs develop left-sided CHF. Volume overload can cause the left ventricle to develop myocardial damage, hyperfunction, and exhaustion. These responses tend to be a sequential progression of events leading to eccentric hypertrophy, in which the left ventricular chamber becomes enlarged to accommodate a greater volume of blood. This change allows the impaired heart to eject a more normal stroke volume despite decreased myocardial contractility. The Frank-Starling law of the heart states that the greater the stretch of the myocardium before contraction, the greater the force of contraction will be. Therefore, if the ventricular end-diastolic pressure (preload) is decreased, the ventricular stroke volume will also be decreased. A diseased heart has a decrease in myocardial stretch and, as a result, a shift in the Frank-Starling curve. The changes associated with eccentric hypertrophy are often subtle and incremental until the body can no longer maintain cardiovascular homeostasis. A patient’s inability to compensate for intravascular volume overload can be secondary to mitral valve disease, a ventricular septal defect, or a patent ductus arteriosus (PDA).

Ventricular compliance is determined by the volume, geometry, thickness, and tissue composition of the chamber walls. Compliance failure results when the ventricles cannot distend enough to allow adequate filling during diastole. Compliance failure can be secondary to fibrosis of the myocardium or endocardium, hypertrophic or restrictive cardiomyopathy, or pericardial effusion. Hypertrophic and restrictive cardiomyopathies are extremely rare in dogs.

Regardless of the inciting cause, at the onset of CHF, compensatory neurohormonal mechanisms are activated in an effort to maintain cardiovascular homeostasis. While not all aspects of the neuroendocrine changes in patients with heart failure are well understood, the overall objective of the compensatory mechanisms is to normalize cardiac output and arterial blood pressure in the presence of a potentially failing heart. The compensatory mechanisms typically follow a predictable sequence. First, a decrease in cardiac output results in reduced arterial blood pressure, which is sensed by baroreceptors located in the carotid body and aortic sinus. Baroreceptor stimulation activates the sympathetic nervous system and causes reciprocal inhibition of the parasympathetic nervous system. This results in an increase in heart rate and peripheral vasoconstriction. Within 30 seconds, cardiac output normally increases to a level that is adequate to sustain circulatory stability.

During this altered state of cardiovascular dynamics, renal blood flow decreases due to the drop in systemic blood pressure and compensatory vasoconstriction. This decrease in blood flow can have detrimental effects on renal function that manifest as prerenal azotemia. The decrease in arterial blood pressure stimulates the juxtaglomerular cells in the kidneys to release prorenin. Prorenin is later cleaved to renin in the first of a number of compensatory mechanisms to maintain normal systemic blood pressure in the presence of decreased cardiac output. These mechanisms make up the renin-angiotensin-aldosterone system (RAAS), which begins when renin acts on angiotensinogen (also called renin substrate) to release angiotensin I, primarily from the liver. Angiotensin-converting enzyme, situated mainly in the lungs, converts angiotensin I to angiotensin II. Angiotensin II has significant vasoconstrictive properties that cause an increase in total peripheral vascular resistance, thereby raising arterial pressure (Figure 1). Angiotensin II also triggers the adrenal gland to release aldosterone to promote sodium and water retention in an attempt to conserve circulating intravascular fluid volume. Concurrently, the pituitary releases antidiuretic hormone, which causes urine concentration. The water retained from the renal collecting ducts further contributes to the increase in blood volume.

Although this cascade of events initially has positive effects, it has potentially deleterious consequences for the cardiovascular and renal systems when it is sustained over the long term. Part of the body’s response to its own

Pulmonary crackles and coughing, commonly caused by pulmonary edema, can also be attributed to many other causes, such as pulmonary fibrosis or chronic bronchitis.
signs of left-sided CHF include respiratory distress, exercise intolerance, lethargy, anorexia, tachypnea, and a moist, possibly productive, sometimes nocturnal cough. Syncope is more rarely described. Pulmonary crackles can be associated with pulmonary edema but can also be ausculted in patients with pulmonary fibrosis, pneumonia, chronic bronchitis, or pulmonary hypertension. Likewise, coughing can be caused by CHF but is also frequently described as a primary presenting complaint in animals with a variety of conditions, including pulmonary parenchymal disease, pulmonary thromboembolism, heartworm disease, fungal disease, pneumonia, and tracheal collapse. Clinical signs common to a variety of cardiac and pulmonary diseases are listed in Table 1. Making the correct diagnosis is important so that treatment can be initiated rapidly without exacerbating an already stressed patient.

Initial thoracic and cardiac auscultation is sensitive in making a diagnosis of cardiac disease, but it is very insensitive for diagnosing CHF. Some dependable signs of cardiac disease include a systolic murmur that is at least grade 3/6 in the absence of anemia or severe hematocrit concentration; any diastolic murmur; gallop sounds/dysrhythmias; a palpable precordial thrill; and absence of arterial pulses.12

A pathologic murmur is typically the result of an underlying disease of the heart or vessels.13 Some important causes of pathologic murmurs include congenital or acquired valvular diseases, stenosis, and vascular shunt anomalies.14 Murmurs resulting from mitral valve insufficiency are loudest over the mitral valve region and left atrium and can be early systolic to holosystolic (Figure 2). Mitral and aortic murmurs are normally ausculted ventrally in the left hemithorax but can radiate dorsally and to the right hemithorax, depending on intensity.14 The correlation between the intensity or loudness of a murmur and the size of the valvular insufficiency is controversial because loud murmurs can result from a small orifice. Likewise, a large orifice, arrhythmia, and high left atrial pressures can result in a less intense murmur.14

The most common cause of mitral insufficiency is chronic degenerative valvular disease.15 Stretching of the annulus secondary to dilated cardiomyopathy and congenital defects such as PDA can also cause a mitral murmur.14 Other heart-base (left-sided) systolic murmurs are associated with pulmonic or subaortic stenosis. An aortic or subaortic murmur is most commonly ausculted over the left fourth intercostal space. It is also possible to aus-
cultate aortic or subaortic murmurs from the right cranioventral third intercostal space. The pulmonic valve is typically auscultated on the left hemithorax, approximately over the third intercostal space. The most common congenital vascular anomaly is PDA. The murmur that accompanies a PDA has been described as “machinery-like,” with continuous systolic and diastolic components that are best heard dorsally and cranially over the aortic and pulmonic valves.

Simultaneous cardiac auscultation and femoral pulse palpation can increase the likelihood of detecting a dysrhythmia. A gallop rhythm with an accentuated third heart sound (S3) may be ausculted in dogs with left ventricular disease. An audible S3 is most commonly heard with conditions that result in ventricular overloading during diastole, including mitral insufficiency, dilated cardiomyopathy, congenital septal defects, and PDA.

Pulse quality or variation can be used as part of the physical examination to assist in the diagnosis of heart disease. Pulse palpation is palpation of the “pulse pressure”—the difference between systolic and diastolic blood pressure. A palpable femoral pulse indicates a mean arterial blood pressure of at least 50 mm Hg. A palpable dorsal pedal pulse indicates a systolic pressure of at least 80 mm Hg. Palpation of pulse quality can help detect regional or generalized perfusion problems and should be used as an adjunct to the physical examination; however, no absolute values of blood pressure should be deduced from pulse palpation alone.

Severe left ventricular failure can result in decreased stroke volume and increased total peripheral vascular resistance, leading to small, weak pulses of poor quality. A bounding or “water hammer”-type pulse is most often associated with PDA or aortic insufficiency. In dogs with mitral insufficiency, most of the stroke volume is ejected early in systole, resulting in a shorter, brisker pulse.

Pulsus alternans is a phenomenon in which strong and weak arterial pulses alternate during a normal sinus rhythm. True pulsus alternans is a sign of severe myocardial depression, typically secondary to dilated cardiomyopathy. Pulsus paradoxus results when systemic arterial pressure falls during inspiration. This is most commonly associated with pericardial effusion and cardiac tamponade. In patients with this condition, pulses are often absent for a number of beats during inspiration.

The complete absence of pulses may result from a partial or complete obstruction of a peripheral artery secondary to thromboembolic disease.

Under normal conditions, a dog’s mucous membranes are pink with a 1- to 2-second capillary refill time (CRT). A CRT that is greater than 2 seconds may indicate an underlying cardiac problem. A rapid CRT in a cardiac patient is more characteristic of hyperdynamic shock or possibly sepsis. Pale mucous membranes are associated with anemia or hypovolemia or may be indicative of severely reduced cardiac output due to heart disease. Cyanosis indicates hypoxemia of cardiac or severe pulmonary etiology. Central cyanosis is caused by hypoxemia of arterial blood secondary to abnormal pulmonary function. Peripheral cyanosis is caused by excessive oxygen extraction and desaturation of capillary

<table>
<thead>
<tr>
<th>Disease/Condition</th>
<th>Coughing</th>
<th>Exercise intolerance</th>
<th>Syncope</th>
<th>Cyanosis</th>
<th>Pulmonary crackles</th>
<th>Poor pulse quality</th>
<th>Palpable dysrhythmia</th>
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<tr>
<td>CHF</td>
<td>Yes</td>
<td>Yes</td>
<td>±</td>
<td>±</td>
<td>Yes</td>
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<td>±</td>
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*These signs depend on where the tumor is located (e.g., near the heart, inside the pericardium, near a major vessel).*
and venous blood during states of poor oxygen delivery. The presence of cyanosis does not alone distinguish cardiac from noncardiac dysfunction.

The cardiac apex beat should be palpated during the physical examination. A decrease in or loss of the apex beat may be secondary to obesity or any condition that displaces the heart within the thorax (e.g., intrathoracic mass, pericardial or pleural effusion, cardiomegaly). By definition, a palpable thrill or vibration caused by a cardiac murmur marks the point of maximal intensity of the murmur. The most severe murmurs (i.e., grade 5/6 to 6/6) are associated with a palpable thrill. With rare exceptions, a palpable thrill indicates that the murmur is of hemodynamic significance. Combining a thorough history with the patient’s signalment and physical examination findings should be able to refine the suspicion of heart failure.

**DIAGNOSTIC TESTS**

If a definitive diagnosis is not possible based on the physical examination, history, and clinical signs, other diagnostic tests must be conducted. Often, it is better to conduct the initial assessment and initiate treatment than to stress the patient in order to obtain diagnostic information such as radiographs. The diagnosis of CHF can be confirmed once the patient is clinically able to withstand the stress of diagnostic testing.

**Radiography**

Radiography is perhaps the best way to document cardiogenic pulmonary edema. However, animal positioning and restraint often cause distress that may be fatal. If thoracic radiographs are deemed essential, prolonged restraint should be avoided. Ventrodorsal views should also be avoided because positioning can cause severe patient distress. When possible, dorsoventral views are preferred.

Early detection of pulmonary edema is challenging for most clinicians. Assessing pulmonary vasculature can help in evaluating the patient before the onset of heart failure. In a patient with imminent CHF, the pulmonary veins may appear larger than the pulmonary arteries due to venous congestion. On the lateral view, the cranial pulmonary vessels are located dorsally to ventrally in the following order: lobar artery, bronchus, and lobar vein. On the dorsoventral (or ventrodorsal, if obtainable) view, the artery is craniolateral to the vein. The mnemonic “Veins are central and ventral” can be used to remember the positions of the vessels.

In patients with early left-sided heart failure, the characteristic radiographic pattern is a hilar to perihilar interstitial infiltrate that diffuses caudodorsally (Figures 3 and 4). This pattern can progress to include an alveolar infiltrate starting at the cardiac hilum and progressing into the periphery. On the lateral thoracic view, the loss of the caudal cardiac waist suggests enlargement of the left atrium. Left atrial enlargement often
causes a dorsal deviation of the left mainstem bronchus. Ventricular enlargement can result in dorsal displacement of the cardiac base, causing a dorsal deviation of the trachea. In certain breeds, such as Doberman pinschers with dilated cardiomyopathy, the pulmonary infiltrates may appear more generalized throughout the lung fields.22

Vertebral Heart Sum

The vertebral heart sum is a technique that evaluates heart size in relation to the length of the thoracic vertebrae. The vertebral heart sum can be calculated in dogs by using the following steps22:

1. Obtain the long-axis measurement.
   • Measure the long axis of the heart from the carina to the apex on a lateral thoracic radiograph.
   • Superimpose this length on the spine, starting at the cranial aspect of the fourth thoracic vertebra, and count caudally the number of vertebral bodies that it covers.
2. Obtain the short-axis measurement.
   • Measure the short axis of the heart at the level of the caudal vena cava to the cranial waist, perpendicular to the long axis of the heart.
   • Repeat the technique used to obtain the long axis, starting at the cranial edge of the fourth thoracic vertebral body.
3. Add the long- and short-axis measurements to yield the vertebral heart sum (Figure 5).

The normal range of the vertebral heart sum in dogs is 8.5 to 10.5. Vertebral heart sums greater than 10.5

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Figure 3. Lateral radiograph demonstrating a perihilar to caudodorsal interstitial pattern consistent with CHF.

Figure 4. Ventrodorsal view of the patient in Figure 3 demonstrating a perihilar to caudodorsal interstitial pattern consistent with CHF.

Figure 5. Vertebral heart sum measurements. $A =$ Distance from carina to apex. $B =$ Distance at the widest point perpendicular to line $A$. For each line, count the number of vertebral bodies covered, starting from the fourth thoracic vertebral body and moving caudally. Add the total number of vertebral bodies: $A + B =$ vertebral heart sum.
may indicate cardiomegaly due to cardiac disease. An enlarged silhouette warrants further investigation with the use of echocardiography.

**Electrocardiography**

Electrocardiography is not a sensitive or specific test for CHF. While CHF patients may have an abnormal electrocardiogram (ECG), a normal or equivocal ECG does not rule out CHF. However, an ECG often helps in generating a differential diagnosis and making a diagnosis. Dysrhythmias are typically due to an underlying cardiac disease such as dilated cardiomyopathy or chronic valvular disease. Factors that contribute to a rhythm disturbance include myocardial hypertrophy, atrial stretch, myocardial ischemia, electrolyte disturbances, aberrant conduction through the His-Purkinje system, myocardial fibrosis, and increased circulating catecholamines or vagal tone. Dysrhythmias can also occur in the absence of underlying cardiac disease. Rapid, sustained, and untreated dysrhythmias may precipitate the development of CHF when underlying cardiac disease is present.

**Echocardiography**

Cardiac ultrasonography, or echocardiography, is an important, noninvasive imaging modality that can help determine the type and degree of cardiac dysfunction. Echocardiography can also be used to rapidly detect pericardial effusion, pleural effusion, and cardiac or intrapleural mass lesions. However, the emergency treatment of heart failure rarely changes based on echocardiographic findings; therefore, it is possible to delay this test until the patient is clinically more stable.

**M-Mode echocardiography** is also known as motion mode and is a one-dimensional, real-time display of the structures of the heart. This mode provides the most accurate measurement of cardiac size and function. The most common measurement of ventricular function is left ventricular fractional shortening, which, in essence, measures the change in the diameter of the left ventricle between contractile and relaxation points. It is very important to remember that fractional shortening is not a direct measure of contractility but is a measure of function. Fractional shortening is affected by preload, afterload, and contractility, individually or together. Most ultrasonography machines automatically calculate the fractional shortening once the necessary points have been plotted on a frozen image. Algorithms for normal fractional shortening based on weight and body surface area are useful in determining normal values for each patient. A fractional shortening above 30% is considered normal. The following equation is used to calculate fractional shortening:

\[
\text{Fractional shortening} = \frac{(LVD - LVS)}{LVD} \times 100
\]

In this equation, \(LVD\) stands for left ventricular diastolic dimension and \(LVS\) stands for left ventricular systolic dimension.

Typically, the reduced left ventricular afterload in dogs with mitral regurgitation artificially increases the fractional shortening to within normal limits, despite a failing myocardium. In other words, a normal fractional shortening in a dog with mitral regurgitation does not translate into normal function. Therefore, some cardiologists believe that measurement of ventricular end-systolic diameter may prove to be a better or more accurate evaluation of function than fractional shortening. This has not been validated or published in the veterinary literature. The characteristic echocardiographic findings in cases of endocardiosis include left-sided (atrial and ventricular) dilation, mitral valve leaflet thickening (Figure 6), and wall and septal hypertrophy. The more common echocardiographic findings with dilated cardiomyopathy (Figure 7) include ventricular and atrial dilation, normal to thick wall and septal thickness, poor fractional shortening, and hypokinesis.

**Color-flow Doppler** provides a simple, accurate method of qualitatively assessing jets from insufficient cardiac valves (Figures 8 and 9). Regurgitation of blood through the mitral valve into the left atrium during systole typically produces a high-velocity jet of blood that can be detected with color-flow Doppler. In most severe cases of mitral regurgitation, flow continues throughout systole at a velocity of 5 to 6 m/sec.
Cardiac Troponin Assay

Troponins are thin myofilibrillar proteins that regulate the interaction between actin and myosin in skeletal and cardiac muscle. There are three distinct troponins (I, C, and T); the cardiac isoforms (cTnC, cTnI, cTnT) show a high degree of cardiac muscle specificity.26–28

A number of studies have demonstrated high circulating cardiac troponin levels in human and animal patients with heart disease; thus, these proteins are a potential marker of heart disease.27,29,30 Cardiac troponin I (cTnI) has been shown to be a highly sensitive and specific indicator of myocardial damage in both humans and animals.28 Cardiac troponin assays are routinely used in human medicine and are regarded as a diagnostic standard of care in patients suspected of having acute myocardial infarction.31 Plasma cTnI concentrations have been demonstrated to be elevated in dogs with cardiac disease.28 This fact may have great clinical potential if a bedside cTnI assay is validated for use in the veterinary emergency and critical care setting.

Brain Natriuretic Peptide Assay

Brain natriuretic peptide (BNP) is a hormone that is synthesized in the atria and ventricles.12 The plasma BNP
concentration is high in human patients with cardiac disease and higher in those with CHF; it is also a sensitive prognostic indicator of premature death from cardiovascular disease. A new canine BNP ELISA (Biosite Inc, San Diego, CA) was shown to be helpful and accurate in diagnosing CHF in dogs presenting with cough or respiratory distress. Cough/dyspnea was secondary to cardiac causes in 93 dogs (54%) and to noncardiac causes in 80 dogs (46%). In the former group, the median BNP concentration was 24.46 ± 48.98 pg/ml, whereas in the latter group, it was 2.66 ± 10.11 pg/ml. This difference was statistically significant (P < 0.05). The median BNP concentration in dogs with subclinical heart disease was 3.03 ± 7.59 pg/ml. Healthy dogs with no evidence of heart disease had a median BNP concentration of 0.96 ± 1.53 pg/ml. Most dogs with noncardiac disease have a normal to only mildly elevated BNP concentration. The new assay may prove to be of great help and importance in veterinary patients with CHF.

**CONCLUSION**

CHF is a complex physiologic syndrome. Although the basic mechanisms are well understood, new diagnostic methods are evolving. Further investigation of the neuroendocrine cascade has enabled the use of blood-based testing as an adjunct tool in the diagnosis of canine cardiac disease, although it is not yet routinely used in clinical practice.

**REFERENCES**

1. Which of the following is not a mechanism in the pathophysiology of heart failure?
   a. myocardial failure
   b. volume or pressure overload
   c. valvular pressure
   d. compliance failure

2. Compensatory mechanisms that are geared toward normalizing cardiac output and arterial blood pressure in the presence of a failing myocardium include
   a. the renin-angiotensin-aldosterone system.
   b. elevated BNP.
   c. peripheral vasoconstriction.
   d. a and c

3. _______ is not a potential presenting complaint for a patient with CHF.
   a. Respiratory distress  c. Tachypnea
   b. Exercise intolerance  d. Polyphagia

4. What is the characteristic radiographic pattern for a dog with CHF?
   a. diffuse bronchial
   b. ventral interstitial
   c. perihilar to caudodorsal interstitial
   d. cranioventral alveolar

5. _______ with left-sided CHF may present with a more generalized pulmonary infiltrate.
   a. Doberman pinschers  c. Toy poodles
   b. Bull terriers  d. Pomeranians

6. Clinical signs of poor tissue perfusion do not include
   a. cool extremities.
   b. weakness.
   c. increased urine output.
   d. poor pulse quality.

7. Which diagnostic test should be avoided in patients with suspected CHF?
   a. radiography (ventrodorsal view)
   b. radiography (lateral view)
   c. echocardiography
   d. electrocardiography

8. The vertebral heart sum provides information about
   a. heart position.
   b. heart size.
   c. ventricular compliance.
   d. pulmonary edema.

9. Pale mucous membranes may be due to
   a. anemia.
   b. hypovolemia.
   c. an increase in cardiac output.
   d. a and b

10. CHF may be ruled out by a
    a. normal ECG.
    b. normal fractional shortening.
    c. vertebral heart sum less than 10.5.
    d. none of the above