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A Closer Look at Urine Casts

Urine sediment analysis is frequently performed as a component of routine urinalysis in veterinary laboratories. While identification of cellular elements and crystals may become routine to most, urine casts can be challenging to discern. This article will discuss the mechanism of cast formation and provide guidelines for microscopic identification of casts.

Fundamentals of Cast Formation

Casts are the only formed element of urine that originate solely in the kidney.[1] Four criteria are necessary for cast formation: high salt concentration, acidic pH, reduced tubular flow rate, and a matrix mucoprotein. Tamm-Horsfall mucoprotein is an albuminous mucoprotein secreted in small amounts by normal tubular epithelial cells in the loop of Henle, distal tubule, and collecting ducts; these are the sites from which most casts form. Precipitation of Tamm-Horsfall mucoprotein is the first event in cast formation. It provides a meshwork of fibrils to which crystals, cells, or cellular elements surrounding the mucoprotein can adhere.[2,3]

Since the cast is formed by using the renal tubular lumen as its mold, it is cylindrical, with parallel sides and consistent diameter throughout the length. In general, the cast has rounded or tapered ends and is several times longer than it is wide, although the exact shape and length of the cast depend on the morphology of the renal tubular lumen from which it is formed.[3] Variation in cast size and shape may contribute to low precision for cast identification in some laboratories.[1]

Types of Urinary Casts

Hyaline Casts

Hyaline casts consist of Tamm-Horsfall mucoprotein and are colorless, semitransparent cylinders (unstained) with rounded ends; they contain no cells. These casts are best viewed microscopically with subdued light.

During prolonged stasis, the hyaline cast serves as the structural unit to which crystals, cells, or cellular elements attach, thus reflecting the malady of the nephron from which the cast is generated.[3] They are named according to the predominant element that they contain. The following are examples:

Erythrocyte Casts

Erythrocyte casts form when red cells are present in the tubular lumen (**Figure 1**). These casts indicate intrarenal bleeding but may also be seen in glomerulonephritis. They are very fragile and appear orange-red to yellow-orange in fresh urine.

Leukocyte Casts

Leukocyte casts connote a suppurative process in the kidney; usually the predominant cell type is the neutrophil (**Figure 2**).

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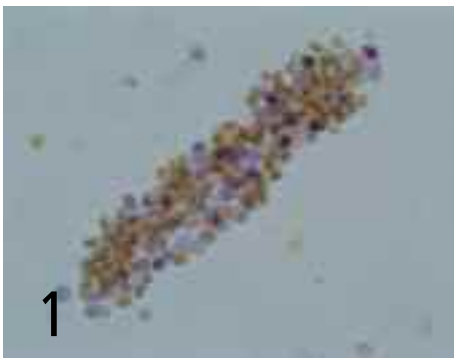
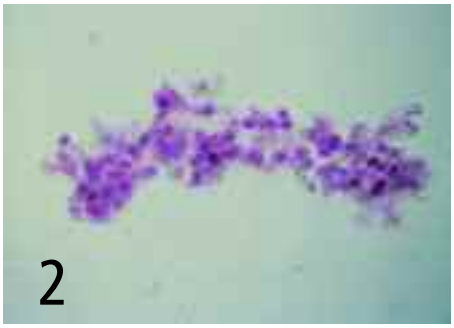
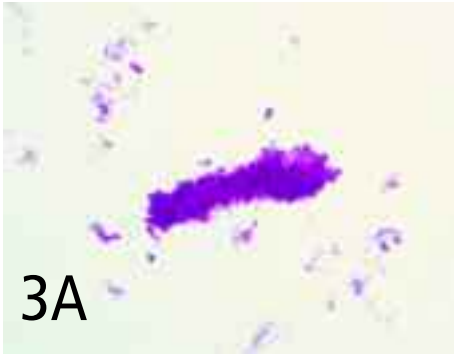


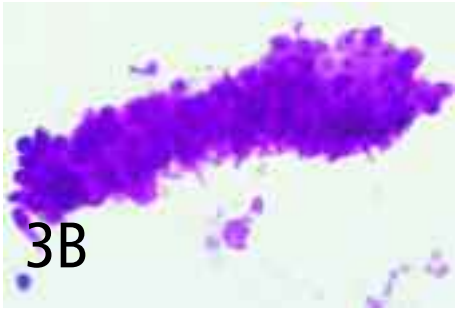
Erythrocyte cast in urine sediment. These casts are very fragile and are rarely observed in the urine of dogs and cats. (Sedi-Stain; 400×)



Leukocyte cast in urine sediment that is beginning to undergo fragmentation. Neutrophils and mononuclear cells are present and moderate numbers of bacteria also may be seen. Caution should be taken to avoid confusing clumps of white blood cells with WBC casts. (Sedi-Stain; 400×)



Epithelial cell cast in urine sediment. Occasional RBCs and free small epithelial cells are also present. (Sedi-Stain; 100×)



Higher magnification of epithelial cell cast depicted in Figure 3A. (Sedi-Stain; 400×)

Granular cast and mucus; both contain fat droplets. The urine cast has defined borders, while the mucus is irregular in shape and size. (400×)

Epithelial Cell Casts

Epithelial cell casts result from desquamation of tubular cells that have not disintegrated (**Figure 3**). They occur in any disease that damages the tubular epithelium. If the epithelial cells have degenerated, these casts may be difficult to distinguish from leukocyte casts.

Granular Casts

Granular casts form from degeneration of epithelial cell casts (**Figure 4**). Epithelial cell demise causes cell boundaries to diminish and disintegrate, giving rise to opaque granules that vary in size and shape. These casts are called *coarsely granular casts*. Further disintegration of coarse granules results in fine granules attached to casts, termed *finely granular casts*, although the distinction between these two types of casts is clinically insignificant.[3] Aside from size of granules, coarsely granular casts also are darker in color (often dark brown), and are typically shorter and more irregular in outline than finely granular casts; they frequently have broken ends. Finely granular casts contain granules that are grayish to pale yellow. In addition to tubular epithelial cell damage, granular casts can be associated with proteinuria of glomerular or tubular origin.

Fatty Casts

Fatty casts (lipid casts) are coarsely granular casts containing fat droplets that accumulate as a result of cell lipid degeneration (**Figure 5**). These droplets are highly refractile and may adhere to Tamm-Horsfall protein in degenerative tubular disease.

Waxy Casts

Waxy casts are colorless to gray and highly refractile (**Figure 6**). They are broader and appear denser than hyaline casts. The body of the cast may be convoluted or contain cracks or fissures; the ends are usually square. These casts are formed in the collecting tubule when the urine flow is decreased; considerable time and intrarenal stasis are necessary for formation of a waxy cast.

One popular theory is that granular, fatty, and waxy casts are products of epithelial cell casts in different stages of degeneration.[3]

Fatty cast. Highly refractile droplets that vary in size and shape appear on multiple planes of view. This type of cast may indicate degenerative tubular disease. (400×)

Waxy cast. The broad structure and blunt ends are hallmarks of waxy casts. (400×)

Frequently Asked Questions

Q. **What preanalytical variables help find urine casts?**

A. For sediment analysis, urine specimens should be centrifuged at 400g for 5 minutes.[2] Excessive centrifugation speed or time may disrupt casts. In addition, urine specimens more than 2 hours old or alkaline pH may contribute to disintegration of urine casts.[4]

Q. **What is the best microscopic magnification to use?**

A. Microscopic analysis should be performed at low magnification (10×) with subdued light to delineate casts in the urine. View 10 fields, average the number found per field, and report the number and type of casts per low-power field.[2]

Q. **Which casts are commonly seen?**

A. Up to 2 hyaline and 1 granular cast per low-power field are considered normal in urine that is moderately concentrated. Excessive numbers of casts imply a disease process in the kidney.[2]

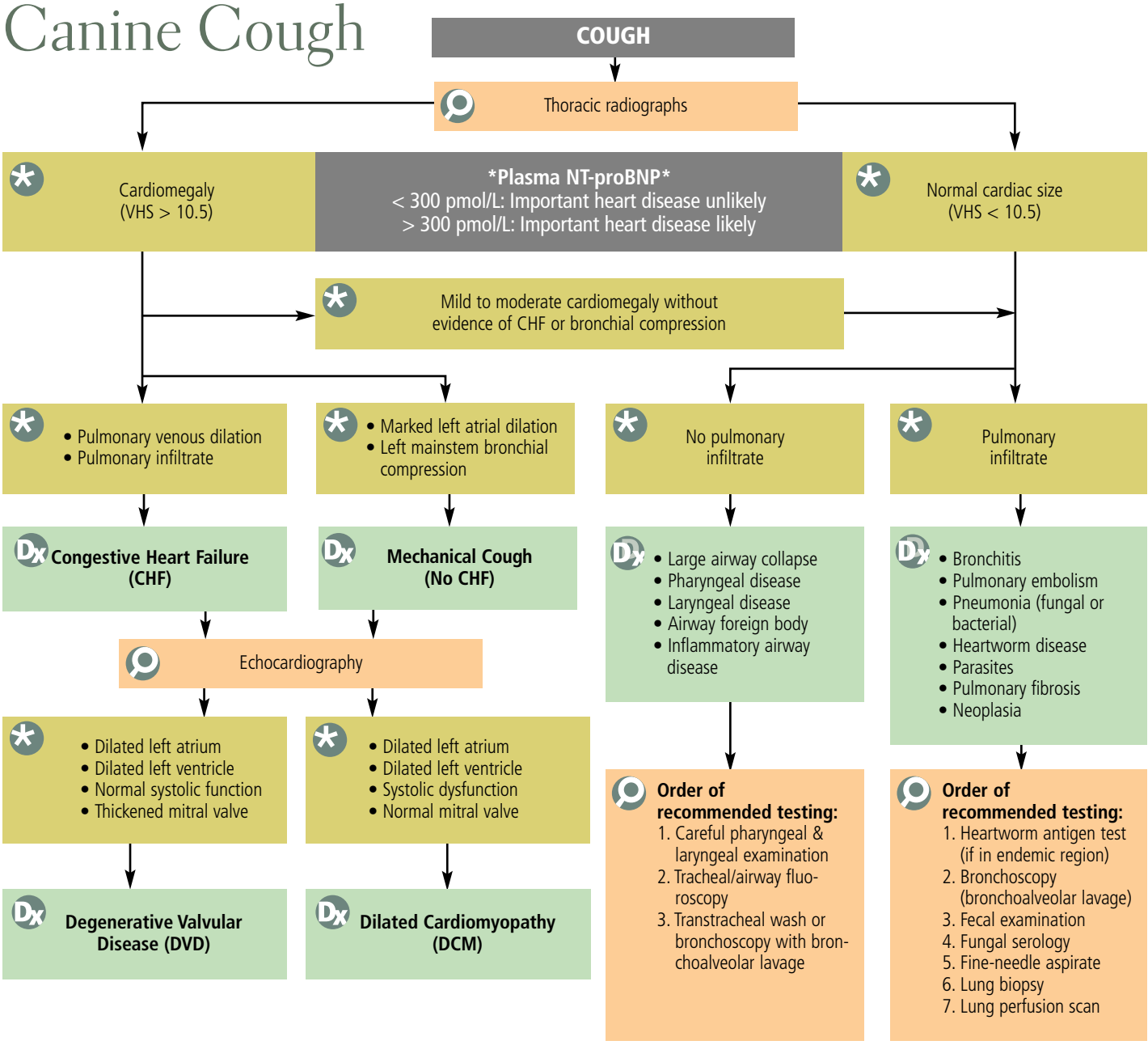
Acknowledgment

Figures 1, 2, 3A, 3B. Reprinted from *Interpretation of Canine and Feline Urinalysis.* Chew D, DiBartola SP—Wilmington, DE: The Gloyd Group Inc, for Ralston Purina, 1998, pp 26-28, with permission.

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

Matthew W. Miller, DVM, MS, Diplomate ACVIM (Cardiology), and Sonya G. Gordon, DVM, DVSc, Diplomate ACVIM (Cardiology), Texas A&M University

Canine Cough



* Plasma NT-proBNP values should serve only as an ancillary supportive diagnostic test, not as a definitive yes or no result. Ongoing research will most likely result in the current cutoff value of 300 pmol/L being adjusted higher, perhaps as high as 800 pmol/L.

- Patients can have cardiac disease and noncardiac cough simultaneously. Dogs with preclinical heart disease may have cardiomegaly but clinical signs due to airway disease. *Cardiomegaly does not automatically equate to heart failure.*
- Although there are numerous causes of canine cardiac disease, the most common diagnosis is DVD or DCM.
- Individual diagnoses are not mutually exclusive (ie, dogs with CHF as a result of CVD often cough due to both CHF and severe left atrial enlargement. This causes a mechanical cough or concurrent airway disease.)

CHF = congestive heart failure; CVD = cardiovascular disease; DCM = dilated cardiomyopathy; DVD = degenerative valvular disease; NT-proBNP = N-terminal prohormone brain natriuretic peptide; VHS = vertebral heart size

Dx Diagnosis
Dx Differential Diagnosis
Investigate
Tx Treatment
Result

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Canine Congestive Heart Failure

Profile

Definition

Congestive heart failure (CHF) is a clinical syndrome associated with a cardiac lesion that results in an increase in venous pressure sufficient to cause a build-up of fluid within the lungs and/or abdomen and/or pleural space.

Signalment

Species. Dogs of any age, sex, breed, or activity level can develop CHF.

Breed predilection. Breeds predisposed to degenerative valvular disease (DVD) and dilated cardiomyopathy are most commonly affected.

- DVD is most common in small breed, older dogs. The prevalence is highest in cavalier King Charles spaniels, dachshunds, toy poodles, miniature schnauzers, and Chihuahuas.
- DCM is most commonly seen in older, large breed dogs. The prevalence is greatest in Doberman pinschers, Great Danes, Irish wolfhounds, boxers, and cocker spaniels.

Causes

- CHF is caused by volume overload of one or both ventricles, resulting in an increase in filling pressures sufficient to cause fluid accumulation within the lungs and/or abdomen and/or pleural space. Any cardiac disease that causes volume overload can cause CHF.
- Although there are several documented causes of CHF in the dog, the majority of cases are secondary to either DVD, or less commonly, DCM.
- Most cases of DCM are thought to be hereditary.
- Nutritional deficiencies, including taurine and carnitine, have been implicated in the etiology of DCM in specific breeds (eg, cocker spaniels).

Risk Factors

The only established risk factor for CHF is development of either DVD or DCM.

Pathophysiology

- The basic pathophysiology of CHF is similar regardless of the underlying cause.
- Although signs of pulmonary congestion (cough, shortness of breath) frequently dominate the clinical picture, the primary abnormality is either myocardial systolic dysfunction (in DCM) or valvular incompetence (in DVD), leading initially to reduced cardiac output.
- Reduced cardiac output activates compensatory mechanisms, including the sympathetic nervous system (SNS) and renin-angiotensin-aldosterone (RAAS) system, which initially restore cardiac output to normal or near-normal levels.
- SNS activation:
  - Causes increased heart rate, increased contractility, and peripheral vasoconstriction
  - Excessive activation of the SNS can be associated with tachyarrhythmias and progressive myocardial dysfunction.
- RAAS activation:
  - Associated with plasma volume expansion from sodium and fluid retention, as well as further vasoconstriction
  - Plasma volume expansion secondary to RAAS activation ultimately leads to elevated venous pressures and edema formation.
- Although initially adaptive, profound and prolonged or chronic activation of these systems is ultimately maladaptive and contributes to disease progression.

Signs

- Common signs of CHF include cough, shortness of breath, exercise intolerance, and syncope.
- Less common signs include abdominal distension, positional respiratory compromise, and isolated nocturnal coughing.
- Patients with DCM, and especially DVD, are frequently identified in the preclinical stage of the disease.

Pain Index

CHF is not thought to be associated with significant pain. Patient anxiety, however, may be associated with development of pulmonary edema and respiratory compromise.

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ACE = angiotensin-converting enzyme; CHF = congestive heart failure; DCM = dilated cardiomyopathy; DVD= degenerative valvular disease; ECG = electrocardiogram; RAAS = renin-angiotensin-aldosterone system; SNS = sympathetic nervous system

Diagnosis

Definitive Diagnosis

- A definitive diagnosis of CHF is established based upon supportive historical findings (ie, tachypnea, cough, exercise intolerance, etc) and physical examination findings (ie, murmur, gallop rhythm, arrhythmia, jugular venous distension, etc) coupled with high-quality thoracic radiographs that identify both pulmonary edema and a cardiac lesion sufficient to cause CHF.
- Resolution of a radiographic infiltrate with a therapeutic challenge of furosemide is considered by many to be the gold standard for diagnosis of CHF (**Figure 1**.)

Other Diagnostic Testing

- Initial blood analysis may demonstrate evidence of renal or prerenal azotemia, mild elevations in liver enzymes, and electrolyte abnormalities.
- Blood abnormalities may become exaggerated following initiation of furosemide and an ACE inhibitor.
- Echocardiography should be performed to confirm the diagnosis. If the patient is *not stable* on room air, the echocardiogram can be delayed until the patient is stable. An echocardiogram cannot confirm a diagnosis of heart failure; it merely confirms the type and severity of structural heart disease.
- Systemic blood pressure should be evaluated to confirm the absence of hypertension, confirm presence and severity of hypotension, and establish a baseline for future reference.
- An ECG is indicated if an arrhythmia is ausculted or suspected based on history.
- Testing NT-proBNP, a circulating biomarker that is elevated in the serum of dogs with CHF, may be useful in dogs with clinical signs suggestive of heart failure, such as dyspnea. The clear clinical utility of this test awaits the outcome of ongoing clinical trials. Although currently published data suggest that values > 300 pmol/L are supportive of CHF, recent reports argue that a more appropriate value might be > 800 pmol/L.
- Other ancillary tests (eg, taurine concentration, *Trypanosoma cruzi* titer, troponin I) may sometimes be useful in dogs with suspected DCM.

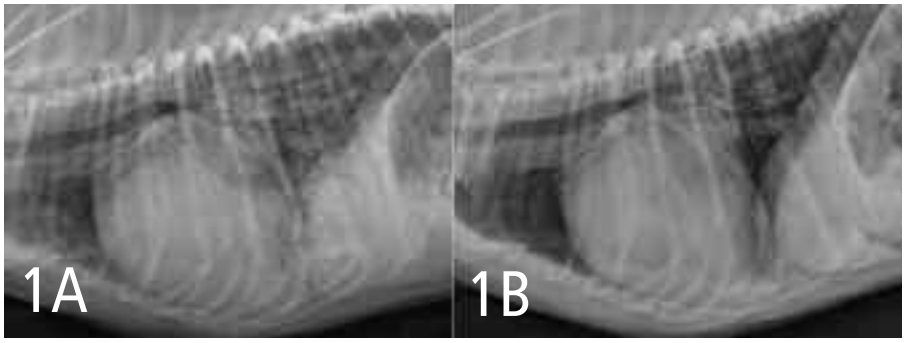
Differential Diagnosis

- Common differentials for cough and shortness of breath include acute primary respiratory diseases, such as infectious bronchitis (kennel cough), chronic small airway disease (chronic bronchitis), dynamic large airway collapse, pneumonia, pulmonary neoplasia, heartworm disease, and pleural/chylous pleural effusion.
- Differentials for collapse and exercise intolerance include diseases of the cardiovascular system in the absence of heart failure (sick sinus syndrome, atrioventricular block [AV block]) the musculoskeletal system (chronic degenerative joint disease, myasthenia gravis), nervous system (intracranial disease, spinal cord diseases), hematopoietic system (anemia), and metabolic derangements (hepatic encephalopathy).

Laboratory Findings/Imaging

Clinicians should be alert to the fact that in presence of cough or signs of respiratory distress, CHF should always be considered a differential diagnosis. Even if thoracic auscultation fails to reveal abnormal lung sounds, a thoracic radiograph should be performed because thoracic auscultation is not highly sensitive for detecting pulmonary edema.

- Radiographically, cardiogenic pulmonary edema in the dog is typically a symmetric, mixed interstitial pattern, worse in the caudal dorsal or perihilar region.
- Other patterns do not rule out cardiogenic pulmonary edema as a differential.
- Definitive diagnosis can be established by documentation of substantial radiographic cardiomegaly or important structural heart disease with an echocardiogram (**Figure 2**), coupled with radiographic clearing of the infiltrate following administration of furosemide (2 to 5 days).
- Resolution of clinical signs is not sufficient to confirm a diagnosis.
- Documentation of structural heart disease with an echocardiogram does not confirm the diagnosis of heart failure, it just means it is possible. Remember that many dogs have preclinical DCM and DVD and could have clinical signs related to other common noncardiogenic diseases (see **Differential Diagnosis**).



Thoracic radiographs from a dog with CHF secondary to DVD before (A) and after 3 days of furosemide therapy (B). Notice the resolution of the pulmonary parenchymal infiltrate but the persistence of the radiographic cardiomegaly.

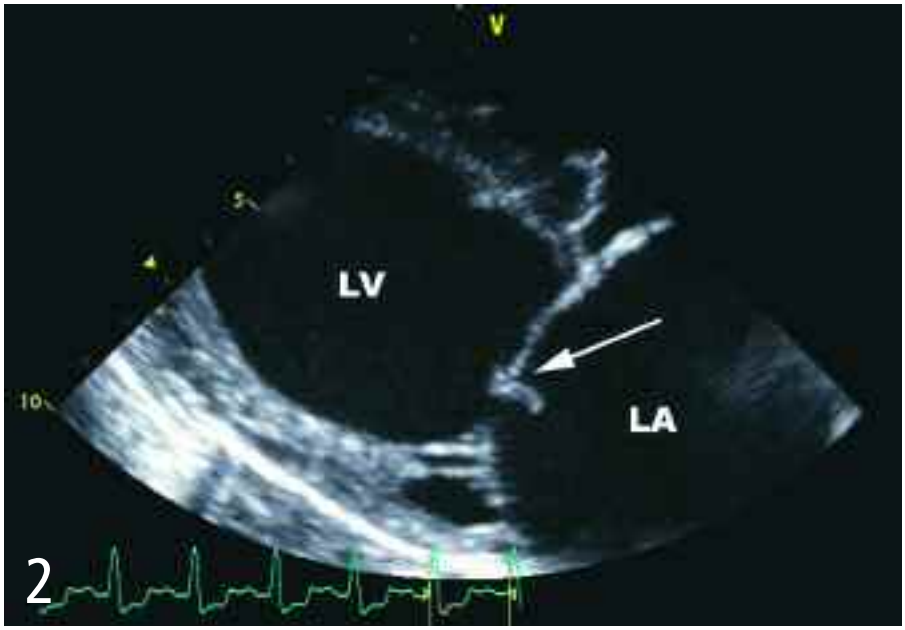
Postmortem Findings

- Gross postmortem findings of CHF are usually those commensurate with pulmonary edema, ascites, and sometimes pleural effusion.
- Generalized cardiomegaly is found, particularly in the left atrium and ventricle (**Figure 3**).
- The mitral valves are thickened, knobby, and sometimes irregular in the case of DVD (**Figure 3**), but typically normal in DCM.

Treatment

Inpatient or Outpatient

- Treatment focuses on the resolution of clinical signs and prolongation of life.
- Resolution of clinical signs of excess plasma volume with appropriate diuresis typically involves the use of furosemide and intermittent abdominocentesis as required, and an ACE inhibitor.
- Improvement and/or preservation of tissue perfusion may include a combination of inotropic support and afterload reduction. If myocardial failure exists, positive inotropic support may increase forward cardiac output and possibly decrease venous filling pressures. If DVD exists, afterload reduction may improve forward cardiac output and decrease volumetric valvular regurgitation. Pimobendan appears to be very effective for both of these applications. Antiarrhythmics are used as required for hemodynamically significant arrhythmias, which are uncommon in DVD but relatively common in DCM.
- Chronic heart failure
  - Conventional therapy for chronic heart failure due to both DCM and DVD includes oral furosemide as needed (1–4 mg/kg Q 8–12 H), an oral ACE inhibitor (enalapril or benazepril) at the label dose, and oral pimobendan (Vetmedin, Boehringer Ingelheim, www.vetmedin.co.uk; 0.25–0.3 mg/kg Q 12 H).
- Acute or decompensated heart failure
  - Management of acute decompensated heart failure can be facilitated by oxygen supplementation and the use of parenteral furosemide (IV or IM) at doses of 1–2 mg/kg Q 1–3 H until the respiration rate has reduced by approximately 50% from baseline.
  - Initiation of oral pimobendan may be beneficial acutely because effects occur within 1 to 3 hours of administration.
  - Addition of an ACE inhibitor can wait until the patient is receiving oral furosemide and is stable.



Right parasternal long axis view from a dog with severe DVD showing a dilated and spherical left atrium and left ventricle. Note the prolapse of the thickened anterior mitral valve *(arrow)* suggestive of chordal rupture. LA = left atrium; LV = left ventricle

Postmortem specimen (whole heart and opened left atrium) from a dog with severe DVD. Note the dilated left atrium and markedly thickened and irregular mitral valve leaflets. Ao= aorta; LA = left atrium

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ACE = angiotensin-converting enzyme; AV block = atrioventricular block; CHF = congestive heart failure; DCM = dilated cardiomyopathy; DVD= degenerative valvular disease; ECG = electrocardiogram; NT-proBNP = N-terminal prohormone brain natriuretic peptide; SNS = sympathetic nervous system

- Additional/alternative medications such as spironolactone, digoxin, beta-blockers (atenolol, carvedilol), and hydralazine may be indicated in acute and chronic heart failure management on a case-by-case basis.
- If azotemia develops, cautious IV administration of 1/2 to 1× maintenance low-sodium fluids should be coupled with temporary discontinuation or reduction in diuretic doses.

Contraindications & Precautions

- There are no absolute contraindications when treating a life-threatening disease such as CHF.
- Caution should be used when initiating heart failure therapy in dogs with significant preexisting azotemia or those receiving NSAIDs.

Activity

- Moderate to severe exercise restriction is warranted in patients with severe acute or decompensated CHF.
- Normal activity patterns can be resumed in dogs that become stable on appropriate medical management. Typically, the pet should be allowed to set its own pace.

Client Education

- Clients must be familiar with the clinical signs of impending or overt congestive heart failure, such as cough, shortness of breath, exercise intolerance, and collapse.
- Having clients monitor resting respiratory rate at home may allow them to anticipate impending decompensation, which is frequently preceded by a gradually increasing resting respiratory rate.
- These patients are invariably on diuretics, so it is imperative that they have free access to fresh water at all times.

Nutritional Aspects

- Moderately sodium-restricted diets may be beneficial if tolerated, but diet should be changed only in dogs that are stable on medical management.
- Administration of taurine and carnitine may be of clinical benefit in cocker spaniels with DCM or other nontraditional DCM breeds (any non-Doberman).
- Administration of fish oil has been associated with reductions in some arrhythmias and circulating levels of inflammatory cytokines in dogs with CHF.

Follow-Up

Patient Monitoring

- Once a definitive diagnosis is established, patient follow-up consists of diagnostic tests directed at determining whether CHF is controlled and if complications associated with disease progression and medications are occurring.
- Diagnostic tests typically include a complete medical history to include drug dosing, thoracic radiographs, routine serum biochemical testing, and measurement of systemic arterial blood pressure.

Prevention

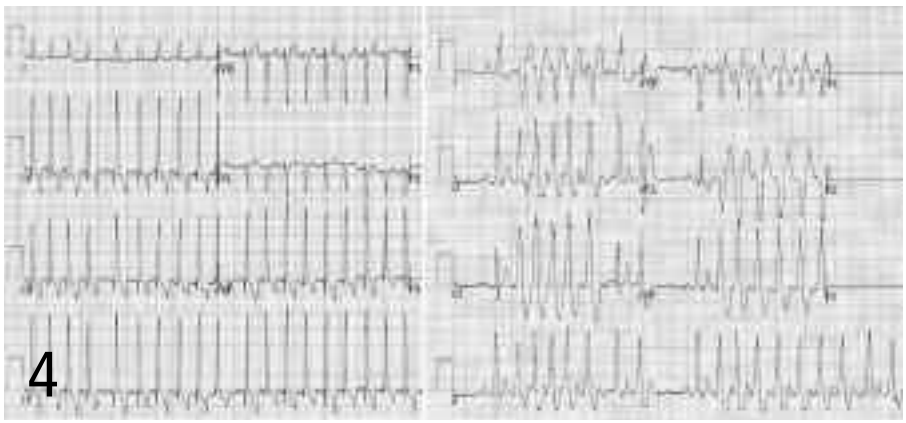
- There are no data to suggest that either of these diseases can be prevented.
- Initiation of therapy in the preclinical stages of DVD is of questionable benefit.
- Identification of DCM in its preclinical stage and initiation of therapy with ACE inhibitors may prolong the preclinical interval.[1]

Complications

- Complications are typically associated with disease progression and heart failure therapy and lead to rapid decompensation.
- Common complications in DCM include development of important arrhythmias such as atrial fibrillation and paroxysmal or sustained ventricular tachycardia (**Figure 4**).
- Complications associated with disease progression in DVD include chordal rupture, left atrial rupture, progressive myocardial dysfunction, and pulmonary hypertension.
- Clinically significant complications associated with standard CHF medications include hypokalemia and azotemia.

Course

- Both these diseases may have a prolonged preclinical stage.
- Once CHF has developed, patients typically have progressively more profound clinical signs that eventually fail to be palliated by medications, or sudden death occurs.



ECGs from a dog with DVD and atrial fibrillation (left) and a dog with DCM complicated by paroxysmal ventricular tachycardia (right).

At-Home Treatment & Monitoring

- Daily administration of multiple medications is necessary for optimal patient management. Having owners monitor resting respiratory rate is helpful. Progressive increases in respiratory rate usually predict clinical decompensation.
- Periodic monitoring of thoracic radiographs, systemic blood pressure, and routine biochemical tests emphasizing renal function and electrolytes help optimize patient care and limit episodes and/or severity of decompensation episodes.

In General

Relative Cost

- Inpatient stabilization of patients with CHF can be costly depending on duration of hospitalization ($$$$–$$$$$).
- Monthly costs associated with outpatient management are relatively modest ($$).

Cost Key

$ = < $100	$$$$ = $500-$1000
$$ = $100-$250	$$$$$ = > $1000
$$$ = $250-$500	

Prognosis

- With very few exceptions, diseases causing CHF in dogs are fatal.
- With appropriate management, median survival times in dogs with CHF secondary to DCM approach 1 year, while dogs with DVD may survive longer than 2 years.
- Good response to short-term/acute management often predicts good long-term survival.
- Many dogs receiving appropriate standard heart failure therapy are completely free of clinical signs and enjoy excellent quality of life for a period of time and clients are typically happy with the outcome of therapy.

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

Article archived on www.cliniciansbrief.com.

CHF = congestive heart failure; DCM = dilated cardiomyopathy; DVD= degenerative valvular disease; ECG = electrocardiogram; NSAID = nonsteroidal antiinflammatory drug

sounding board CONTINUED FROM PAGE 9

faucet." Assuming that this use of the paper towel is to prevent clean hands from picking up germs left when the faucet was turned on, isn't it irresponsible to leave germs behind for someone else to pick up? Shouldn't you recommend that the faucet be washed with soap and water before being turned off? Along this same line, I recently read a study that in the human field, stethoscopes are a significant fomite—and probably pens are as well. How many of us sanitize those after working with a possibly contagious animal?

Raymond J. Schuerger, DVM
Pittsburgh, Pennsylvania

Editors Respond

Thank you for catching our short-sighted commentary. Of course, we didn't intentionally make recommendations to leave the next person in the bathroom at peril; they were based on use of toilets where you yourself are not the offending person and, as such, do not go far enough. Your remarks are insightful and certainly will benefit readers and give them pause. Thus, we are passing them on.

Section archived on www.cliniciansbrief.com

Debra Horwitz, DVM, Diplomate ACVB, Veterinary Behavior Consultations, St. Louis, Missouri

The Great Escape: Dogs That Roam

My client has a 2-year-old male dog that jumps the fences and roams the neighborhood. What advice can I give her to stop this unwanted behavior?

Dogs may escape and roam for many reasons, including exploration, searching for mates (if intact), searching for food, or for social contact. Roaming can be the result of separation distress or a response to frightening situations such as storms or noises. It can be a predatory behavior or motivated by inter-dog aggression. Escape behaviors are self-fulfilling; leaving the house or yard and being free to roam is a great reward. In some cases, the dog is just taking advantage of the opportunity provided by an open door or gate. Intact males are most commonly presented for escape and roaming behaviors, but neutered animals will escape and roam as well.

Investigate the Problem

The first step is to determine why and how the dog escapes. Asking the owner simple questions about the household routine and recent changes may provide some insight. Animals that have recently relocated may be trying to return to their old home territory. Intact animals may be seeking mates. Does the pet show signs of anxiety at owner departure or prior to escape, such as whining, pacing, destruction, or salivation? Where does the pet go and what does it do once it escapes? Does the pet just bolt out the door when it is opened?

Dogs that escape and roam put themselves at risk. They may be hit by cars, encounter toxic materials, or be injured by other animals. The animal may present a risk by aggressive behaviors directed toward other animals or people. Without proper identification, it may never be returned home and could potentially be euthanized by a shelter.

Contain the Situation

Treatment begins with management. Proper containment and a good routine are essential to preventing escaping and roaming. The animal's confinement must be made secure, because each time the dog is able to overcome a repair and escape, its behavior is reinforced and it will likely work harder and longer to get out the next time. Install secure fencing outdoors. For dogs that tend to escape and roam, underground electric fencing is usually not a sufficient deterrent. Gates should latch securely and, if necessary, be locked. If the dog charges out the door when it is opened, secure the animal before opening the door. If visitors or young children commonly enter and exit the house without warning, it may be necessary to use gates and confinement indoors to prevent the dog's escaping.

Anxiety or Phobias

If the animal suffers from an anxiety-related condition such as separation anxiety, or noise or storm phobias, that condition that must be treated concurrently or escape is still likely. While identifying and treating underlying conditions is beyond the scope of this article, many resources for treatment and pharmacologic intervention are available.[1-4] If the dog is intact and searching for mates, then neutering or spaying is important. A 60% to 90% reduction in roaming behavior can be achieved by castrating intact male dogs.[5]

No Place Like Home

Identifying the principal motivation for roaming may help determine a treatment plan. The goal is to make staying home more attractive than leaving. For example, enriching the home environment with food-dispensing toys or puzzles, increasing social contact time with people, and offering adequate outlets for play and exploration can be helpful. When possible, include daily leashed walks that provide contact time with other dogs, if dog-compatible, and games. If the dog cannot be walked due to unruly behavior or pulling, address this with training and perhaps a head collar or no-pull harness. Regular mealtimes and random treats, especially

continues