Management of Congestive Heart Failure in Dogs

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Introduction

Heart failure represents the end result of a variety of conditions in dogs. With few exceptions, such as congenital disease (e.g., PDA) or certain acquired diseases like taurine deficiency and idiopathic pericarditis, there is no cure for the underlying cause of heart failure and therefore treatment is focused on control of clinical signs and improvement in quality of life. While as clinicians, we enjoy classifying the specific type of heart disease (e.g., dilated cardiomyopathy versus chronic valvular disease), it may be in fact more important to treat the body's response to the failing heart, rather than the appearance of the heart itself.

Most drugs associated with an improved survival have a mechanism of action that, at least in part, interferes with the neurohumoral stimulation that causes signs of CHF (e.g., angiotensin-converting enzyme inhibitors, beta-blockers, and spironolactone). There has been progressive support for use of therapies that interrupt these compensatory responses. Studies into the effects of neurohumoral activation at the cellular (myocardial) level have identified that progressive myocardial hypertrophy, cardiac remodeling, myocardial fibrosis, and progressive cardiac enlargement all are directly impacted by these seemingly exuberant compensatory reactions. Pimobendan is the exception to this rule, and this newest cardiac drug both improves survival and improves quality of life by reducing the clinical signs associated with heart disease. Since most other drugs that act as positive inotropes have been associated with shortened survival in people with chronic CHF, it seems that the profession's prior efforts to target drugs based on the mechanism of action of the drug (e.g., heart not contracting well >>> give positive inotrope) may be misguided, and instead the first-choice approach should be to employ therapies that have been studied and demonstrated to improve clinical signs and/or survival, regardless of their mechanism of action.

Angiotensin-Converting Enzyme Inhibitors

Angiotensin-converting enzyme (ACE) inhibitors are commonly used in the management of CHF. Their use is predicated on the knowledge that interfering with the activation of the renin angiotensin system leads to diminished plasma levels of angiotensin II and reduced stimulation of aldosterone. As a result, fluid retention and vasoconstriction are blunted. Newer information documents the benefit of ACE inhibition with respect to altering the progressive cardiac enlargement and remodeling known to attend most forms of heart failure, although this might not be true in situations of isolated mitral regurgitation. This effect on cardiac remodeling seems to be mediated (at least in part) via an increase in bradykinin. The beneficial effects of ACE inhibition likely result from both the vasodilation and the drug's

effects to reduce cardiac remodeling. If the progressive ventricular enlargement and progressive replacement of myocytes with non-distensible fibrous tissue can be avoided, survival might be prolonged, and symptoms of heart failure can be reduced.

Angiotensin-converting enzyme inhibitors have proved to be useful in a variety of settings. In a large number of human heart failure trials, ACE inhibitors have been proven to prolong survival. In addition, these drugs have been documented to slow the progressive cardiac enlargement, and delay the onset of CHF in humans with left ventricular dysfunction. In well-designed canine heart failure trials, ACE inhibitors resulted in improved clinical signs and prolonged the time until an animal dropped out of the study (equivalent to improved survival). Which animals should be treated with ACE inhibitors? There is very good experimental evidence to support the use of ACE inhibitors in dogs with congestive heart failure due to either dilated cardiomyopathy or chronic valvular disease (endocardiosis). Most veterinary cardiologists would agree that ACE inhibitors are probably indicated in dogs with congestive heart failure due to endocarditis, congenital heart diseases associated with volume overload, and other forms of left-sided congestive heart failure in dogs. The role of ACE inhibitors in the treatment of animals with asymptomatic heart disease remains a hotly debated topic; however, at best the increase in survival time in dogs with asymptomatic chronic valvular disease is modest, and therefore use of ACE inhibitors in dogs with minimal atrial enlargement is probably of marginal benefit. Alternatively, in dogs with mitral regurgitation and concurrent systemic hypertension, as well as in dogs with mitral regurgitation and concurrent significant proteinuria, administration of an ACE inhibitor is currently recommended by the author. Twice-a-day therapy is recommended, and ACE inhibitors are usually added into furosemide and pimobendan for the management of CHF in dogs.

Furosemide (Lasix)

It has been documented that asymptomatic heart disease is usually not associated with measurable activation of certain neuroendocrine systems; however, these neuroendocrine responses are stimulated following therapy with a diuretic. This stimulation of the neuroendocrine system is generally thought to be counterproductive, especially if one is to believe the prevailing attitudes regarding the progression of heart failure. Many cardiologists would not recommend single agent use of furosemide for treatment of CHF. This does not mean that appropriate diuretics should not be used when congestion develops! Still, it is quite difficult to define the exact dose of diuretic required by any individual dog or cat with CHF. The dose required to clear significant edema accumulations and cause the animal to be minimally symptomatic (the desired dose) is often close to a dose that might result in electrolyte disturbance, dehydration and the development of prerenal azotemia. The combined use of ACE inhibitors and diuretics compromises one of the kidneys' normal compensatory mechanisms (vasoconstriction of the efferent arteriole) and can lead to elevation of BUN and creatinine when 1) excessive diuretic dose is initiated or 2) significant preexisting renal disease is present. Most cardiologists now concurrently use ACE inhibitors and diuretics for animals with CHF, and the prevailing recommendation is to measure renal function prior to starting therapy and then repeat the BUN, creatinine and electrolytes 5 to 10 days after starting drugs to treat congestive heart failure.

Owing to concerns about azotemia, and in response to certain studies in people with CHF which document adverse outcomes when high doses of diuretics are employed, the author currently recommends use of the "lowest possible dose of furosemide" in animals with CHF. This often means a degree of experimentation must be performed to best evaluate an individual animal's needs. Giving an owner upper and lower limits for acceptable furosemide dose, and carefully explaining to them that they should "give more for difficulty breathing or rapid respirations, and give less if the animal seems weak, lethargic, anorexic, or depressed" has worked successfully for the author. In most instances, canine patients are given less than 2 mg/kg q 12 h, and in most cats I initially try to use 6.25 mg/cat/day for chronic therapy. Some cats require higher doses of furosemide, but many can be treated with 6.25 mg/cat every other day. When a dose of 2.2 mg/kg twice a day is exceeded during chronic therapy, the author usually thinks that diuretic resistance has been reached and adds in either spironolactone alone or a combination hydrochlorothiazide with spironolactone instead or going to higher and higher doses of furosemide. Alternatively, in refractory cases, use of injectable furosemide can restore a diuresis in some animals, and torsemide can also be tried at approximately 0.2 to 0.3 mg/kg q 12–24 hours when other diuretics have become ineffective

Pimobendan

Pimobendan is a calcium-sensitizing drug that is useful as a positive inotrope in addition to having properties as a phosphodiesterase inhibitor with vasodilating effects. It has been studied in dogs with chronic valvular disease and in dogs with dilated cardiomyopathy. In dogs with dilated cardiomyopathy many authors indicate that there is a notable clinical benefit to addition of pimobendan to background therapies for heart failure, including improved clinical signs and improved survival. In most veterinary studies, pimobendan-treated dogs have fared as well or better than dogs treated with ACE inhibitors. Pimobendan also seems to be associated with a low side effect profile and the author has not recognized repeatable side effects in dogs, other than perhaps GI upset, and the side effects do not appear to be associated with any negative impact on the animal's well being. The drug has only been studied in dogs with active CHF, and the role for this drug in pre-CHF situations is unclear - currently the author is withholding pimobendan until CHF is present or at least some signs of cardiac disease (e.g., cough with marked left atrial enlargement) are evident. Some studies suggest that use in dogs with mitral valve disease before the onset of congestive heart failure could be detrimental, but these small studies require additional review. The usual dose for pimobendan is 0.25 to 0.3 mg/kg q 12 hours. We sometimes use higher doses (off label) for advanced, refractory CHF. There is now evidence of prolonged survival in Doberman pinschers if pimobendan is administered in the occult phase, once moderate cardiac enlargement is evident on echocardiography.

Beta-Blockers and Sotalol

Beta blockade has gained favor recently as a therapeutic modality for treatment of CHF. Several studies on the use of metoprolol and carvedilol have documented benefits that accrue from chronic treatment with beta-blockers in people, but the role of beta-blocker therapy in dogs with congestive heart failure is unclear. These effects in people are often not seen for several months after initiation of beta blockade. Such benefits include upregulation of previously downregulated beta-receptors, improved cardiac performance (improved stroke volume), and improved survival. These clinical benefits appear to have sound theoretical basis, and are

currently being evaluated in veterinary patients with naturally occurring CHF. Beta-blockers are best employed in animals that are minimally symptomatic with early/mild heart failure, or in animals in later stages of CHF that are already **well controlled** on a stable cardiac drug regimen. Metoprolol has been used at 0.2 mg/kg BID, with slow titration upward q 2–3 weeks up to 0.4–6.6 mg/kg TID. Carvedilol has been used by some cardiologists with doses of 0.2 mg/kg BID and slow titration upward toward a dose of 0.8 mg/kg BID; however, many dogs with CHF will not tolerate this upward dose titration with either drug.

Another drug with some beta-blocking action which is probably better classified as a Class III antiarrhythmic drug is sotalol. Administration of sotalol results in prolongation of action potential duration in canine Purkinje and ventricular muscle fibers. This drug may terminate reentrant arrhythmias by prolonging refractoriness. Electrocardiographic effects include a slowing of the sinus node firing rate and prolongation of the P-R and Q-T intervals (dog). Like other beta-blockers, sotalol has negative inotropic effects and this causes the drug to be poorly tolerated by dogs with advanced myocardial failure and congestive signs. The drug is used for both ventricular and supraventricular arrhythmias. It is infrequently associated with conversion of atrial fibrillation to sinus rhythm. Side effects can include the development of congestive heart failure, bradycardia or AV block, hypotension, syncope, and GI side effects.

Sodium Nitroprusside for Severe CHF

Dogs and cats with severe pulmonary edema that is unresponsive to 1 or 2 doses of furosemide at 4 mg/kg can be very difficult to manage successfully. The author has had some success with dobutamine; however, the drug that most reliably controls life-threatening CHF is sodium nitroprusside. A continuous rate infusion is required in order to give sodium nitroprusside, but this drug can be very effective in this setting. Measurement of blood pressure is essential; however, the author has become more permissive about how low a blood pressure can be tolerated for 4 to 12 hours in order to control severe CHF. Many dogs and cats with severe pulmonary edema and a systolic blood pressure of 70 mm Hg can still tolerate an infusion of sodium nitroprusside for several hours without apparent long-term renal damage resulting from the presumed renal hypoperfusion. Close observation of the animal, skilled technicians, and frequent reevaluation of the animal's condition are needed to find an effective dose. Doses ranging between 2 and 10 mcg/kg/min are often successful in controlling severe pulmonary edema in both dogs and cats. The drug is usually administered for 12 to 48 hours until severe edema is resolved and other cardiac medications can be added into the drug regimen.

Potential Treatment Options or Interventions

Angiotensin-Converting Enzyme Inhibitors

Enalapril: 0.5 mg/kg q 12–24 hours, lisinopril: 0.5 mg/kg q 12–24 hours.

Benazepril: 0.25 to 0.5 mg/kg q 12–24 hours (Use BID if 0.25 mg/kg dosing).

Indications

Congestive heart failure, systemic hypertension, protein-losing nephropathy.

Side Effects

Azotemia, hypotension, gastrointestinal side effects; side effects most commonly seen when used concurrently with diuretics.

Follow-up

Recheck renal function and electrolytes in 5 to 14 days. Serial blood pressure measurement.

Diuretics

Furosemide: Highly variable dosing schedule based on degree of CHF.

Cats: 1 mg/kg q 48–72 hours may suffice in some cases. 1–2 mg/kg q 12–24 hours; use the higher dose for certain cases, especially those with pleural effusion.

Dogs: 2 mg/kg q 8–24 hours for chronic management of CHF. For emergency management of CHF doses up to 4 mg/kg q 1 hour for 2–5 doses may be required. Furosemide can also be used as a CRI at 0.1–1 mg/kg/hour.

Indications

Congestive heart failure

Side Effects

Azotemia (usually prerenal), hypokalemia, hyponatremia, hypochloremia, hypomagnesemia, metabolic alkalosis, hypotension, dehydration.

Follow-up

Recheck renal values and electrolytes in 5 to 14 days after starting and after each dose adjustment.

Spironolactone

1 mg/kg q 24 hours to 2 mg/kg q 12 hours. Can be combined with hydrochlorothiazide and dosed in a similar fashion.

Indications

Some clinicians are using spironolactone earlier in the management of CHF due to the improved survival noted from clinical trials of human patients when spironolactone was added to background therapies. In these cases a lower dose may be appropriate. When CHF is refractory and/or a chronic furosemide dose is required in excess of 2 mg/kg q 12 hours, then addition of these drugs may be appropriate.

Side Effects

Gastrointestinal side effects, anorexia, hyperkalemia.

Follow-up

Recheck electrolytes in 5 to 14 days and q 2 months thereafter.

Digoxin

Difficult to dose and therefore a number of dosing schemes are available. In dogs, the author most frequently starts at 0.005 mg/kg q 12 hours and makes further dose reductions based on cachexia, renal insufficiency, large volume effusions, and certain breed-specific limitations (Starting dose no more than 0.125 mg BID for Doberman regardless of size; no more than 0.25 mg BID as a starting dose in any dog). The author avoids use of digoxin in cats unless CHF is accompanied by rapid atrial fibrillation.

Indications

Atrial fibrillation, repetitive supraventricular arrhythmias in conjunction with CHF, small-breed dogs with syncope and no clear arrhythmic etiology, refractory CHF.

Side Effects

Anorexia, gastrointestinal side effects, neurologic side effects (depression or dull mentation), cardiac arrhythmias

Follow-up

Digoxin serum levels should probably be maintained 0.8–1.2 ng/ml range in a 6 to 8 hour post-pill blood sample, obtained 5 to 8 days after starting digoxin, which is toward the lower end of the therapeutic range for most laboratories.

Beta-Blockers

Metoprolol extended release: 0.2 mg/kg BID, with slow titration upward q 2–3 weeks up to 0.4–0.6 mg/kg BID.

Carvedilol: Initial doses of 0.2 mg/kg BID and slow titration upward to 0.8 or 1.0 mg/kg q 12 h.

Indications

Certain supraventricular or ventricular arrhythmias. As an adjunct for management of CHF aimed at improving survival (extrapolating from human trials).

Side Effects

Weakness or lethargy due to reduced cardiac output, bradycardias, AV block, bronchoconstriction, worsening signs of CHF, syncope, hypotension. Beta-blockers are best initiated in animals that are minimally symptomatic for CHF.

Follow-up

Serial exams are often required, usually q 2 weeks, in order to assess response to therapy and assist in uptitration of the drug.

Pimobendan

Pimobendan: 0.25–0.3 mg/kg q 12 hours

Indications

A calcium-sensitizing drug used as a positive inotrope with vasodilator effects in animals with CHF. Pimobendan might also prove to be useful in ICU situations.

Side Effects

Gastrointestinal effects, possibly arrhythmias.

Follow-up

Routine follow-up is used after initiation of this medication. Higher doses might help in refractory CHF (off label).

Dietary Modifications

A variety of diets are reduced in sodium; some have more specific modifications which are desirable for heart disease.

Indications

Moderate sodium restriction early in heart disease; more severe sodium restriction might be better as CHF advances. Protein restriction should be avoided, many renal diets have inadequate protein, despite the fact that they might be sodium restricted.

Side Effects

Uncommon; diet acceptance can be challenging if a sudden dietary switch is made when CHF is active or new drugs which affect appetite are being introduced.

Follow-up

Routine follow-up is indicated, with evaluation of dietary compliance and body condition score at recheck exams.

For access to diet handouts and other helpful information for owners of pets with heart disease, see:
www.tufts.edu/vet/heartsmart/ (http://www.tufts.edu/vet/heartsmart/) or
http://www.tufts.edu/vet/heartsmart/resources/treats_for_dogs_with_heart_disease.pdf
(VIN editor: the link was updated on 11-02-16 http://vet.tufts.edu/wp-content/uploads/treats_for_dogs_with_heart_disease.pdf (http://vet.tufts.edu/wp-content/uploads/treats_for_dogs_with_heart_disease.pdf))

Avoiding Common Pitfalls

1. Discuss the diet - avoid salty treats and foods
2. Is more drugs better than fewer drugs?
3. Limit TID and QID medications
4. Give cat owners the option to have drugs compounded (liquid)
5. Avoid drugs with high side effect profile
6. Check and follow renal function
7. Advise owners that "some changes in medication doses or types might be needed"
8. Discuss exercise moderation
9. Judicious management of cardiac arrhythmias
10. Try to prevent thrombus formation in cats with significant atrial enlargement.

When Should I Recheck Him (or Her), and What Should I Do?

The author routinely recommends re-evaluation of the patient with a chemistry profile to check renal function and electrolytes 7 to 10 days after initiation or alteration of cardiac medications. Serum digoxin levels should be obtained, ideally 8 hours post-pill, an examination 7 to 10 days after initiation of the medication. Physical examination, packed cell volume, total proteins, blood pressure, follow-up thoracic radiographs, follow-up electrocardiography, and historical reports from the owner are all useful in trying to assess response to therapies. In many instances, the doses or types of medications need to be adjusted at the time of this initial recheck and a subsequent visit 7 to 10 days later should be scheduled.

The next recheck visit should be scheduled for 2 to 3 months and at that time a physical examination with chemistry profile should be performed. Finally, 6 months after initial diagnosis the author recommends a follow-up examination with echocardiogram to search for changes in the appearance of the heart or other alterations which might dictate a need for change in therapy.

Speaker Information

(click the speaker's name to view other papers and abstracts submitted by this speaker)

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URL: http://www.vin.com/doc/?id=7672007 (http://www.vin.com/doc/?id=7672007)

Diagnosing CHF in dogs — The Golden Rules

MEDICAL FAQS

Mark Kittleson

Introduction

Older, small breed dogs commonly present to a veterinarian with a left apical systolic heart murmur due to mitral regurgitation (MR; secopndary to myxomatous mitral valve degeneration) and respiratory abnormalities (e.g., cough, tachypnea, dyspnea) where the veterinarian must try to figure out if the respiratory abnormality is due to primary lung disease or left heart failure (pulmonary edema). This is particularly challenging for most veterinarians when the presenting complaint is a cough. In numerous instances these dogs are diagnosed with left heart failure when instead they have lung disease, most commonly chronic bronchitis. The following "rules" have been devised to help veterinarians make the distinction between left-sided congestive failure and primary lung disease in old, small breed dogs with a heart murmur.

References **Related Info** (/members/search4/Default.aspx?rows=25&sort=1&pg=1&snip=0&rnw=0&IS=0&q=Diagnosing+CHF+in+dogs&hdlr=vinmost&lyr=-1&specs=-1&

Clinical Use Information

Do the Golden Rules apply to all dogs with all forms of heart disease?

No. These rules apply primarily to adult (usually older) small-breed dogs with left heart disease.

Do the Golden Rules apply to cats?

Not necessarily. Only rules 1, 5, 8, and 9 are directly applicable.

Are the Golden Rules infallible?

No. Nothing is infallible! The Golden Rules work in most cases when applied correctly. If you follow the rules, you will increase or decrease the probability that an adult small-breed dog has left heart failure (cardiogenic pulmonary edema). However, they also require correct interpretation of things such as thoracic radiographs and clinical findings.

The Golden Rules

1. **It takes severe, overwhelming heart disease to cause heart failure.** Mild, mopderate and even failry severe heart disease does not result in heart failure. Heart failure only occurs when severe, overwhelming heart disease is present. In the case of chronic mitral regurgitation (MR) that means the defect (hole) in the mitral valve has to be so large that >75% of the blood flow ejected with each beat goes backward through the mitral valve into the left atrium and so <25% goes forward into the aorta. In chronic MR, the size of the left atrium reflects the severity of the MR. Only when the left atrium is severely enlarged is the MR severe enough for the left atrial pressure to increase high enough that pulmonary edema accumulates. Therefore, when chronic left heart failure is present, the left atrium is usually severely and is always at least moderately enlarged. The caveat is that this is not true when acute left heart failure due to a ruptured chorda tendineae is present (see rule #2 below). In most dogs you can accurately assess left atrial size with thoracic radiographs. In one study, the VHS (/doc/?id=3854772) of Cavalier King Charles Spaniels with pulmonary edema was always >12 vertebrae (Lord *et al* 2012). Whether this applies to other breeds is not known. Alternatively, you can assess LA size echocardiographically, although in most cases, this is not required.
2. **If the leak is acute, the LA can be small.** In dogs with acute chordal rupture and acute L-CHF, the LA may not be enlarged because it might not have had time to do so. These are uncommon (<5% of cases?). Chordal rupture should only be suspected when the onset of clinical signs is acute.
3. **A soft murmur is not heard with severe MR.** Almost all older, small-breed dogs have myxomatous mitral valve degeneration (MMVD) with severe, overwhelming mitral regurgitation (MR) as the cause of their left heart failure (cardiogenic pulmonary edema). Small breed dogs almost never get DCM, except for cocker spaniels. In almost all instances a soft (grade 1-2) systolic murmur is due to mild mitral regurgitaiton and almost no dogs with severe MR have a soft murmur. Therefore, in almost all instances, a moderately loud to loud (grade 3 or more) left apical systolic murmur has to be present for pulmonary edema to be present and a soft murmur (grade 1-2) tends to rule out left heart failure (cardiogenic pulmonary edema). A lack of a murmur essentially excludes pulmonary edema as a cause of the clinical signs. However, a loud murmur is quite frequently heard in dogs with mild to moderate mitral regurgitation so hearing one does not mean that the patient has severe disease or left heart failure (cardiogenic pulmonary edema).
4. **Diagnosing cardiogenic pulmonary edema radiographically is nowhere as easy as it's made out to be.** If there is **clear evidence** of severe LA enlargement and pulmonary edema radiographically (and a loud heart murmur) then you're pretty much home free. However, there are numerous instances when you cannot accurately diagnose pulmonary edema radiographically. Digital radiography has made it more difficult (too busy). Expiratory-phase films result in over-interpretation of the pulmonary parenchymal pathology. Old fat dogs have whiter lungs than young skinny dogs. This common finding (old dog lungs on expiration) leads to the misdiagnosis of "pulmonary edema". The ever-so-common finding of "mild perihilar edema", in the vast majority of cases, is a false positive finding. If you don't have **clear and obvious** marked (severe) left atrial enlargement and **clear** pulmonary edema (especially in the dorso-caudal lung fields), further

work-up is necessary, unless the dog is dyspneic. If the dog is dyspneic, prompt attention is needed and may require your best estimate of what is occurring along with response to drug therapy.

5. **Rely on the sleeping respiratory rate.** Any dog that has pulmonary edema will have tachypnea. Consequently, if the RR in the exam room is normal (<30 breaths/minute), pulmonary edema is not present. Conversely, if a dog is clearly dyspneic due to pulmonary edema in the exam room, thoracic radiographs will usually show clear evidence of the pulmonary edema. The primary problem is that many dogs are excited or stressed and so pant or have an increased RR in the exam room, making it difficult to impossible to get a respiratory rate that is meaningful. In that case, if you don't believe the dog has fulminant clear-cut L-CHF, it's time to have the owner count the SLEEPING (/doc/?id=3853832) (click on this link for instructions) respiratory rate (SRR) at home.
6. **If the dog is has a loud heart murmur and you've documented tachypnea/dyspnea, it's time to do a Lasix trial.** Cardiogenic pulmonary edema is not the only thing that causes tachypnea. Consequently, once you've documented that the SRR is increased or the RR and effort are clearly increased in the exam room and the thoracic radiographs are at least consistent with pulmonary edema, administer furosemide (furosemide, Salix) (/doc/?id=5224182) at a dose of at least 2 mg/kg BID to see if the SRR decreases. If the SRR decreases following appropriate furosemide therapy, you can be reasonably certain the dog has left heart failure (cardiogenic pulmonary edema). If it does not, the Lasix dose may be too low or the dog may have primary lung disease instead.
7. **Most dogs with a cough and a heart murmur are not in heart failure.** Coughing is the hallmark clinical sign of chronic bronchitis. Tachypnea/dyspnea are the hallmarks of pulmonary edema. Dogs with pulmonary edema can cough but coughing is much more common with primary lung disease, like chronic bronchitis. Remember: Murmur + cough does not equal L-CHF!

   Similarly, just because a cough improves with furosemide therapy does not mean the dog has left heart failure (cardiogenic pulonary edema). Furosemide is a bronchodilator (/doc/?id=5224182#two) (and probably does a couple of other things with regard to the respiratory tract) so dogs with chronic bronchitis often improve on furosemide with reduced or resolved coughing. This is not evidence that the dog has left heart failure (pulmonary edema).
8. **An echocardiogram can only provide findings consistent with, not diagnose, heart failure.** You cannot diagnose left heart failure using an echocardiogram (cannot see pulmonary edema with it). All you can do is identify findings that are compatible with left heart failure (e.g., a severely enlarged LA; ruptured chorda tendineae). A veterinary cardiologist can calculate an estimate of left atrial (LA) pressure that might increase the probability of a dog having pulmonary edema, but even that's not proof.
9. **Heart failure is a terminal disease.** Dogs with L-CHF don't live for months without furosemide and don't live for years with heart failure, especially on the same dose of furosemide. The disease is progressive, especially once heart failure begins. So if you have a dog that presents with a cough that has been

present for months and unchanged and the dog is not receiving furosemide, the cough is very unlikely to be due to heart failure. Similarly, if you have a patient that has been on the same dose of furosemide for many months (maybe even years) and is still alive, it's not in heart failure. Heart failure inexorably progresses, requiring a higher and higher dose of furosemide.

10. **Crackles? Think lungs, not heart.** Loud (obvious), coarse crackles (the only ones we usually recognize) occur rarely with cardiogenic pulmonary edema, are much, much more common in chronic bronchitis, and are due to the mucous in the airways popping with respiration. Therefore, if you hear obvious crackles, especially in a dog that is not obviously dyspneic, think lungs, not heart.

Can I figure this out interactively?

Yes. The CHF Helper (/Link.plx?ID=4558932) we (Rishniw) have developed on VIN allows you to go through the diagnosis for a patient step-by-step until you arrive at a possible solution.

[Top]

References

Journal Articles

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1. Kittleson MD and Kienle RD: Small Animal Cardiovascular Medicine - Chapter 19 - Diagnosis (/doc/?id=5496367&pid=5928)

Proceedings

1. Rishniw M. It Used To Be Simple: The Basics of Diagnosing CHF (/doc/?id=5742439). BSAVA Congress 2013

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SPEAKER INFORMATION

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