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“Congestive Heart Failure: The Golden Rules”
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The Golden Rules of Diagnosing Left Heart Failure
(Cough + Murmur ≠ Heart Failure)

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Older, small breed dogs commonly present to a veterinarian with a left apical systolic heart murmur due to mitral regurgitation (MR) 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.

The Golden Rules

1. **A cough is not diagnostic of left heart failure.** Coughing is the hallmark 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 left heart failure!

   Similarly, just because a cough clears up or improves with furosemide therapy does not mean the dog has left heart failure. Furosemide is also a bronchodilator (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 pulmonary edema due to left heart failure.

2. **The dog has to have a heart murmur.** 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. Small breed dogs almost never get dilated cardiomyopathy (DCM), except for American cocker spaniels. 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. A soft murmur in a small-breed dog with MMVD is very rarely associated with left heart failure. Thus, a soft murmur (grade 1-2) tends to rule out left heart failure. The 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.

3. **The left atrium has to be at least moderately and usually severely enlarged in chronic heart failure.** Heart failure only occurs when severe, overwhelming heart disease is present. In the case of chronic mitral regurgitation that means the defect (hole) in the mitral valve has to be so large that >75% of the blood flow ejected with each beat by the left ventricle goes backward through the mitral valve into the left atrium and so <25% goes forward into the aorta. In other words, mild and moderate mitral regurgitation do not result in heart failure. In chronic mitral regurgitation, the size of the left atrium reflects the severity of the MR. Only when the left atrium is severely enlarged does the left atrial pressure finally increase and so pulmonary edema start to accumulate. Therefore, when left heart failure is present, the left atrium is usually severely and is at least moderately enlarged. In most dogs you can accurately assess left atrial size with thoracic radiographs. In one study, the vertebral heart score 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.

4. **The left atrium can be small if the leak is acute.** In dogs with acute chordal rupture and acute left heart failure, 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. And even then, most dogs that present as if they’re in acute heart failure have actually gradually gotten worse and have finally reached a point that the owner finally notices something is wrong.

5. **Pulmonary edema has to be present but it isn’t as easy to diagnose radiographically as you would like.** 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 (lung fields are 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 report 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.

6. **If the respiratory rate (RR) is normal, there is no pulmonary edema.** Almost 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 and clear-cut left heart failure, it’s time to have the owner count the sleeping respiratory rate (SRR) at home (see below).

7. **Do a furosemide trial if the RR is elevated.** If a dog is tachypneic or dyspneic in the exam room, and has radiographs consistent with left heart failure, administer furosemide at a dose of at least 2 mg/kg BID and going up as high as 8 mg/kg every 1-2 hours depending on the severity of the dyspnea. If the RR decreases following appropriate furosemide therapy, you can be reasonably certain the dog had left heart failure. Similarly, if the sleeping RR is elevated at home, it’s time to do a furosemide trial. A reduction in SRR within 24-48 hours of starting at least a 2 mg/kg BID trial of furosemide confirms your suspected diagnosis of left heart failure.

8. **Most dogs with mitral regurgitation do not need an echocardiogram.** 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; a 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. You need radiographs +/- the SRR to diagnose cardiogenic pulmonary edema.

9. **Dogs in left heart failure do not live for years on therapy and do not live months without therapy.** Dogs with left heart failure don’t even live for weeks without furosemide. The disease is progressive, especially once left 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 not due to left heart failure. Similarly, if you have a patient that has been on the same dose of furosemide for 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. And it kills within months, not years.

10. **Dogs with crackles usually have chronic bronchitis.** Loud (obvious), coarse crackles (the only ones we usually recognize) occur uncommonly with cardiogenic pulmonary edema and are much, much more common in chronic bronchitis. With chronic bronchitis they are due to the mucous in the airways popping with respiration. Therefore, if you hear obvious crackles, think lungs (chronic bronchitis), not heart failure. Also, if you hear crackles and the dog is not tachypneic/dyspneic, the crackles are not due to pulmonary edema.
**Sleeping Respiratory Rate (SRR)**

**Why monitor SRR?**

Owner participation in assessing and recording of SRR is:

- Probably the most sensitive indicator of developing pulmonary edema or pleural effusion.
- A very useful home-monitoring technique for owners to perform.
- Used once a diagnosis of CHF has been made (and is now controlled), or where substantial heart disease exists and is likely to result in CHF at some future stage.

**How is it done?**

Have the owner record the SRR and character on their pet (number of breaths/min). The recording should be done when the animal is comfortably resting or asleep, in a thermo-neutral environment (ie, not too cold, not too hot). This should be repeated daily for 2-3 days (to get a baseline variation), and then once or twice weekly. Keep a log.

If the SRR changes substantially between measurements, the owner should then measure daily to confirm the change, or to document a trend. If a trend is documented, the owner should contact the veterinarian for further evaluation.

You can also have your client download an SRR app for their iPhone or iPad

If you are uncertain that the owner is measuring the SRR accurately, or if the owner tells you he/she can't do it, have them record the pet while it is sleeping with a video camera and send you the video file

**Normal SRR** in dogs and cats is usually <30 breaths/min, often in the high-teens or low 20s. However, a few cats will have a normal rate in the 30s. Dogs and most cats with subclinical heart disease also have SRR <30 breaths/min. Consistent SRR >30 breaths/min in dogs with underlying heart disease is strongly suggestive of developing CHF. However, primary respiratory disease with concurrent subclinical heart disease needs to be ruled out. Consistent SRR >30-40 breaths/min in cats with pre-existent heart disease warrants further evaluation.

**What do I do if SRR is high?**

An elevated SRR is NOT diagnostic of CHF. It simply means that there is something causing a tachypnea in your patient, requiring additional evaluation. That can be pulmonary edema or it can be primary respiratory disease. Therefore, the SRR is good at ruling out CHF if it's normal, but not necessarily as good at ruling in CHF if it's elevated.

If the SRR is elevated, several options exist. If everything else in your physical examination and history points to CHF as the cause, then thoracic radiographs should be obtained. If there are no contraindications, a short diuretic trial can be employed. If the SRR decreases on furosemide, you then have made the diagnosis of left heart failure.

Presence of sinus arrhythmia or sinus bradycardia is inconsistent with a diagnosis of CHF - nearly all animals with CHF will have sinus tachycardia.

**What do I do if the patient is already receiving diuretics or other drugs for CHF?**

If the patient is already on medications for CHF, then SRR can be used to monitor for relapsing CHF. A patient with well controlled CHF will have a normal SRR (<30 breaths/min). If CHF starts to recur, the SRR will start to creep up over time, allowing you and the owner to intervene earlier in the disease process (e.g., increase the furosemide dose), before the situation becomes critical.

Sometimes, animals are placed on medications for CHF inappropriately, or at least questionably. In such a situation, where you are not sure that CHF was ever present, but the patient is receiving diuretics and other CHF medications (ACE-inhibitors, pimobendan, etc), a slow and gradual reduction in the diuretic dose can be implemented, while the owner records the SRR daily. If the SRR remains unchanged over a 1-2 week period, while the diuretic dose decreases toward zero, then the diagnosis of CHF is ruled out (i.e., the patient was originally misdiagnosed as having CHF). If, on the other hand, the SRR gradually increases as the diuretic dose decreases, then the diagnosis of CHF was most likely correct. In that case, the diuretic can be resumed at the original dose to prevent further development of CHF.