Heart Disease: Diagnosis & Treatment

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Background

Clinical heart disease is the stage of disease when a patient has or had signs attributable to cardiovascular disease. However, similar to human heart disease, determining when veterinary heart disease becomes clinical or progresses to heart *failure* is variable; it is often unclear exactly when preclinical heart disease evolves to clinical heart disease (see **Definitions**). Congestive heart failure (CHF) is easily recognized when there is radiographic evidence of pulmonary edema or cardiogenic effusion. Subtle signs of CHF (eg, pulmonary venous enlargement, exercise intolerance, weakness, lethargy) may be noted on thoracic radiographs or patient history.

Therapeutic planning for heart failure patients includes diagnosing the underlying heart disease and identifying the stage or class of heart disease.

Heart Disease Classification

In veterinary medicine, a modified version of the American College of Cardiology and American Heart Association (my.americanheart.org) classification system for humans is commonly used; the author follows a modified version for hypertrophic cardiomyopathy (HCM) and dilated

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Definitions

Heart failure is failure of the heart to pump and distribute blood appropriately and results in tissue hypoxia.

Congestive heart failure is cardiac dysfunction resulting in increased venous/capillary pressures that lead to edema or effusions.

How I Treat Congestive Heart Failure

- ❏ Reduce excessive preload.
- ❏ Reduce afterload.
- ❏ Improve myocardial contractility.
- ❏ Consider the specifics for CVD.
- ❏ Consider the specifics for DCM.
- ❏ Consider the specifics for HCM.
- ❏ Enact follow-up measures.

CHF = congestive heart failure, CVD = canine valvular disease, DCM = dilated cardiomyopathy, HCM = hypertrophic cardiomyopathy

5 Primary Determinants of Stroke Volume & Cardiac Output

Preload
Preload depends on venous return, total blood volume, and blood distribution within the vascular system. Increased diastolic stretching (ie, preload) results in more forceful cardiac contraction (ie, Frank-Starling mechanism). If diastolic myocardial function is normal, increased end-diastolic volume induces a more forceful contraction with only modest increases in end-diastolic pressure. Myocardial fibrosis and hypertrophy impede diastolic filling because they prevent optimal stretch by the myofibers even when filling pressures are increased. With many cardiac diseases, excessive preload can result in pleural effusion, ascites, pulmonary edema, or peripheral edema.

Afterload
Afterload, the intraventricular systolic tension experienced during ejection, is determined by peripheral vascular resistance, physical properties (compliance) of the arterial tree, and volume of blood in the ventricle at onset of systole. Increased afterload leads to reduced rate or amount of ejection at preload. Reducing the afterload in patients with CHF may improve forward cardiac output, reduce regurgitant jet size, and speed resolution of CHF signs.

Myocardial Contractility and/or Inotropy
Myocardial contractility, the innate property of the myocardium that defines force of contraction, is affected by sympathetic nerve activity, concentration of circulating catecholamines, and, to some extent, heart rate. Anoxia, ischemia, acidosis, and disease processes (eg, DCM, chronic mitral insufficiency with severe volume overload) can reduce contractility and inotropic state. Reduced myocardial contractility can be assessed by echocardiography and more indirectly via systemic blood pressure measurement. Drugs that can increase myocardial contractility (ie, positive inotropes) can help alleviate acute and chronic clinical signs of CHF.

Heart Rate
Heart rate is determined by automaticity of the sinoatrial (SA) node, which is subject to autonomic regulation and other environmental (eg, temperature) and metabolic (eg, thyroid levels) factors. Cardiac output increases linearly with heart rate when stroke volume is constant; however, at extremely rapid heart rates, ventricular filling, stroke volume, and cardiac output are reduced. Patients can present with clinical signs attributed to bradyarrhythmias (eg, third-degree atrioventricular [AV] block, high-grade second degree AV block) and tachyarrhythmias (eg, ventricular or supraventricular tachycardia). Arrhythmias can contribute to clinical signs in patients with structural heart disease (eg, atrial fibrillation in patient with severe mitral insufficiency and CHF, ventricular tachycardia in patient with dilated cardiomyopathy). Addressing arrhythmias in a patient with CHF may help speed resolution of signs.

Synergy
Ventricular synergy is orderly synchronized contraction of the ventricles. Dyssynergy can lead to a reduction of stroke volume and cardiac output. Resynchronization therapy, fairly common in human cardiology, is starting to be investigated in veterinary cardiology (ie, in patients with a pacemaker-induced myocardial dysfunction).

Although the stage of CHF often determines treatment, it is essential to assess and therapeutically support the 5 primary determinants of stroke volume and cardiac output.

CHF = congestive heart failure, CVD = canine valvular disease, DCM = dilated cardiomyopathy, HCM = hypertrophic cardiomyopathy, LAE = left atrial enlargement

cardiomyopathy (DCM; see **Stages of Heart Disease**). This classification can help veterinarians effectively identify and treat heart disease, but there is no perfect standard for treating all heart disease patients as they are expected to advance through the stages unless progression is altered by treatment. Other classification schemes include modified NYHA (New York Heart Association) classes, ISACHC (International Small Animal Cardiac Health Council) classes, and ACVIM (American College of Veterinary Internal Medicine) Consensus Statement Classes for canine valvular disease (CVD).

Diagnostics

One of the most important tools for assessing heart disease is a complete cardiovascular examination (see **How I Diagnose**, next page), including assessment of peripheral perfusion, femoral pulse quality, body condition, respiratory rate and character, and auscultation of the heart sounds, rhythm, and rate. Assessment of systolic blood pressure via Doppler sphygmomanometry can be part of a thorough cardiovascular examination in patients with suspected underlying heart disease (ie, patients at stage B1 and greater). Assessment of blood pressure provides information about afterload, making it an important part of the cardiovascular examination and therapeutic planning. Ultrasound skills to assess left atrial size, ventricular contractility, and presence or absence of effusion are useful. Echocardiographic assessment by a veterinary cardiologist is ideal. In patients requiring diuretic therapy, baseline assessment of renal values and electrolytes is recommended.

Therapy

Although the stage of CHF often determines treatment, it is essential to assess and therapeutically support the 5 primary determinants of stroke volume and cardiac output: preload, afterload, inotropic state (myocardial contractility), heart rate, and synergy (see **5 Primary Determinants of Stroke Volume & Cardiac Output**). These determinants help guide the measures necessary for patients with cardiac disease, regardless of the underlying disease process.

Stages of Heart Disease: CVD, DCM, & HCM Cases

Stage A
Patients at high risk for developing heart disease with no identifiable structural heart disorders (eg, Cavalier King Charles spaniel, Maine coon cat without heart murmurs)

Stage B
Patients with structural heart disease (eg, mitral insufficiency murmur) but without signs associated with CHF; because of important clinical implications for prognosis and treatment, patients are subdivided into B1 and B2 for many diseases

- **Stage B1:** Asymptomatic patients with structural abnormalities, minimal cardiac remodeling, and no radiographic or echocardiographic evidence of cardiac remodeling (eg, Cavalier King Charles spaniel with mitral insufficiency but no significant left atrial enlargement [LAE], Maine coon cat with mild hypertrophy and systolic anterior motion of the mitral valve but little to no LAE)

- **Stage B2:** Asymptomatic patients with structural abnormalities and echocardiographic or radiographic evidence of cardiac remodeling

Stage C
Patients with past or current clinical signs of heart failure associated with structural heart disease

Stage D
Patients with end-stage disease and clinical signs of heart failure refractory to standard therapy and requiring advanced or specialized treatment to remain clinically comfortable

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How I Diagnose Heart Disease

Consider the specifics for CVD.

- Stage A
  - Auscultation
- Stage B
  - Auscultation
  - Thoracic radiographs
  - Blood pressure
  - Echocardiogram
- Stages C & D
  - Auscultation
  - Thoracic radiographs
  - Blood pressure
  - Serum biochemistry profile
  - Echocardiogram

Consider the specifics for canine DCM.

- Stages A & B1
  - Auscultation
  - Echocardiogram
  - 24-hour Holter
  - Electrocardiogram
  - ± NT proBNP
  - ± Genetic test (ie, Doberman pinscher, the PDK4 mutation; boxer and other breed tests[1-3])
- Stage B2
  - Auscultation
  - Echocardiogram
  - 24-hour Holter
  - Electrocardiogram
- Stages C & D
  - Auscultation
  - Echocardiogram
  - Thoracic radiographs
  - Electrocardiogram
  - Serum biochemistry profile
  - 24-hour Holter

Consider the specifics for feline HCM.

- Stage A
  - Auscultation
  - Echocardiogram
  - ± Genetic test (ie, Maine coon[4,5] and ragdoll cats[6-8])
  - ± NT proBNP
- Stages B1 & B2
  - Auscultation
  - Echocardiogram
  - Total thyroxine (TT_4) test
  - Blood pressure
  - ± Genetic test
  - ± NT proBNP
- Stages C & D
  - Auscultation
  - Echocardiogram
  - TT_4 test
  - Blood pressure
  - Thoracic radiographs
  - Serum biochemistry profile
  - ± NT proBNP[9]

How I Treat Congestive Heart Failure

Reduce excessive preload.

- Diuretics
  - Emergency situations and long-term maintenance
    - Thiazide diuretics (long-term maintenance)
    - Spironolactone (long-term maintenance)
    - Furosemide for long- and short-term maintenance
- Venodilators
  - Nitroglycerin ointment
  - Nitroprusside infusions
  - Isosorbide compounds
  - ACE inhibitors
- Thoracentesis or abdominocentesis for effusions
  - With third-space effusion, mobilizing fluid with diuretics alone is often difficult; manual removal of fluid results in faster resolution without excessive diuretic dosing and volume depletion.

Reduce afterload.

- Nitroprusside (most common) and hydralazine can be used in emergencies.
- Amlodipine and ACE inhibitors are more effective and used more commonly for chronic CHF.
- Pimobendan (Vetmedin, vetmedin.com), although an inotropic agent, also has vasodilatory capacity and thus can reduce afterload while providing inotropic support.

Improve myocardial contractility.

- Pimobendan for emergency and maintenance therapy
- β-adrenergic agonists (eg, dobutamine) for emergent patients and those with DCM
- Cardiac glycosides (eg, digoxin, digitoxin) rarely for maintenance therapy and never for emergent cases

- Phosphodiesterase inhibitors (eg, amrinone, milrinone for emergent cases), but only available in costly IV formulations

☑ Consider the specifics for CVD.

- Stages A & B1
  - No drug or dietary therapy
- Stage B2
  - ACE inhibitor if blood pressure >140 mm Hg or if moderate–severe LAE
  - ± Pimobendan
- Stage C
  - Furosemide
  - Pimobendan
  - ACE inhibitors
  - Spironolactone
  - Moderate sodium restriction but adequate protein diet
- Stage D
  - Furosemide
  - Pimobendan (off label at increased dose)
  - ACE inhibitors
  - Spironolactone
  - Amlodipine
  - ± Hydralazine
  - ± Thiazide
  - Moderate sodium restriction but adequate protein diet
  - Oxygen therapy
  - Nitroprusside and/or dobutamine

☑ Consider the specifics for DCM.

- Stage A
  - No drug or dietary therapy
- Stage B1
  - No drug or dietary therapy
  - Antiarrhythmic agents if needed
- Stage B2
  - Pimobendan
  - ACE inhibitor
  - Moderate sodium restriction but adequate protein diet
- Stage C
  - Pimobendan
  - ACE inhibitors
  - Furosemide
  - Spironolactone
  - Moderate sodium restriction but adequate protein diet
  - Antiarrhythmic agents if needed
- Stage D
  - Furosemide (switch to torsemide if indicated)
  - Pimobendan
  - ACE inhibitors
  - Spironolactone
  - ± Thiazide
  - Moderate sodium restriction but adequate protein diet
  - Antiarrhythmic agents if needed

☑ Consider the specifics for feline HCM.

- Stages A & B1
  - No drug or dietary therapy
- Stage B2
  - Clopidogrel (off label) ± aspirin
  - ± Atenolol
  - Enalapril
- Stage C
  - Furosemide
  - ACE inhibitor
  - Clopidogrel (off label) ± aspirin
  - ± Atenolol
- Stage D
  - Furosemide
  - ACE inhibitor
  - Clopidogrel (off label) ± aspirin
  - Pimobendan (off label) if myocardial failure present

☑ Enact follow-up measures

- Recheck blood work (eg, renal values, electrolytes) 5–7 days following any changes in or initiation of diuretic or ACE inhibitor therapy.
- When no therapeutic changes are made, recheck patients in stage B1 or B2 q6–12mo.
- For patients in stage C or D (regardless of disease), recheck assessments generally consist of a thorough cardiovascular examination with assessment of systolic blood pressure q6mo.
- Repeat thoracic radiographs ± echocardiogram are not absolutely necessary at every recheck unless clinical examination or history suggest decompensation.
- To prevent owners from abandoning therapy because of cost, diagnostic testing may be best reserved for when it is most necessary. ■ cb

See **Aids & Resources**, back page, for references & suggested reading.

ACE = angiotensin-converting enzyme, CHF = congestive heart failure, CVD = canine valvular disease, DCM = dilated cardiomyopathy, HCM = hypertrophic cardiomyopathy, NT proBNP = N terminal prohormone of brain natriuretic peptide, TT_4 = total thyroxine