Diagnosis and Staging of Canine Heart Disease:
Deciding When & What to Treat
Rebecca L. Stepieın DVM, MS, DACVIM (Cardiology)

I. Prevalence and types of canine heart disease
   A. Valvular disease affects 70% of canine patients presented for cardiac evaluation
   B. Myocardial disease (DCM) is dramatic, but affects only about 8% of canine heart patients
   C. Large dogs may have anything, but miniature breeds seldom have DCM

II. Heart disease vs. heart failure
   A. The purpose of the cardiovascular system is to maintain adequate blood pressure (BP) to supply organ perfusion.
      1. **Heart disease**: physical or functional abnormality of component(s) of the CV system
      2. **Heart failure**: CO is inadequate to support BP despite normal hydration
         a. Low cardiac output = signs of low BP (hypotension)
         b. Sodium/water retention = congestion/edema/fluid accumulation
      3. **Compensatory mechanisms**: renin-angiotensin-aldosterone, sympathetic nervous system, antidiuretic hormone
   B. *Why bother?*
      1. HEART DISEASE----(usually)-------→ MONITOR
      2. HEART FAILURE-----(almost always)---→ TREAT

III. Identifying heart failure based on history
   A. Heart disease (HD):
      1. someone (maybe you) informed owners of abnormal physical findings consistent with heart disease OR the animal was screened based on breed
      2. the owner detects no abnormalities in the dog (appetite, activity etc.)
      3. abnormal physical findings: typically murmur, gallop or arrhythmia
   B. Heart failure (HF):
      1. physical findings consistent with heart disease are present
      2. owner reports outward clinical signs of cardiovascular dysfunction
a. signs of low output: inability to maintain an exercising state, FATIGUE, (decreased activity in general)
b. signs of water retention: shortness of breath (pulmonary edema or pleural effusion), abdominal fluid accumulation

C. Common historical reports to listen for (or directly ask about)
   1. unexplained weight loss
   2. difficulty sleeping (dog “can’t get comfortable at night”)
   3. cough: usually a sign of cardiac enlargement, may be worse in some positions

IV. Identifying heart failure based on physical examination
   A. General appearance
      1. HD: no outward abnormalities related to CV system
      2. HF: weakness, fatigue, abdominal fluid, dyspnea at rest, cyanosis
         a. left sided: dyspnea, cyanosis, cold extremities
         b. right-sided: ascites, inability to lie sternally or laterally
   B. Heart rate
      1. HD: usually normal (70-80 \(\rightarrow\) 120-140 bpm) - not breed related
      2. HF: often elevated at rest (>120 bpm)
   C. Rhythm (extra heart sounds [gallop] may be present in HD or HF)
      1. HD: usually regular or SINUS ARRHYTHMIA at normal heart rate
      2. HF: regular or arrhythmia (irregular rhythm with pulse deficits)
   D. Heart murmurs: intensity
      1. cannot differentiate HD vs. HF
      2. louder murmurs are worse in animals with valvular disease
      3. softer murmurs are worse in myocardial disease
   E. Heart murmur: point of maximal intensity and timing (systolic/diastolic etc.)
      1. cannot differentiate HD vs. HF
      2. useful to diagnose exact disease
      3. descriptive modifiers are not helpful and should be avoided (“holosystolic”)
   F. Pulse strength: reflects cardiac function but not HD vs. HF
G. Jugular examination:
   1. HD: may be jugular pulsation if severe tricuspid insufficiency or arrhythmia
   2. HF: jugular distension indicates elevated RA pressure → HF (± pulsation)

H. Pulmonary auscultation:
   1. HD: breath sounds audible all fields, may be accentuated if animal is breathing heavily, cough may be present if LA impinges on bronchus or airway collapse is present
   2. HF: crackles and wheezes may be present if pulmonary edema, especially caudal dorsal lung fields on deep inspiration. Muffled sounds ventrally → pleural effusion

I. Biochemical findings:
   1. HD: biochemical findings reflect concurrent disease states
   2. HF: azotemia, decreased PaO2 responsive to oxygen, mild ALT elevations

V. Thoracic radiographs: best documentation of fluid retention
   A. HD: changes in cardiac size and shape, no changes in vessels size, no infiltrates
   B. HF: HD findings PLUS infiltrates and vascular congestion OR pleural effusion
      1. infiltrates consistent with HF: vascular, interstitial and alveolar patterns
      2. NOT consistent: bronchial (unless pre-existing), nodular, mass lesions
      3. Things to watch for:
         a. pulmonary edema is usually bilateral
         b. predictable pattern: LA enlargement → pulmonary vascular congestion → interstitial leading to alveolar infiltrates (dorsal caudal, then ventral caudal, then ventral cranial)
         c. hepatomegaly with enlarged caudal vena cava (± ascites) = right heart failure

VI. Staging heart failure: a guide
   A. Once HF is present – how severe is it?
   B. Clinical guide for treatment
      1. New York Heart Association (NYHA): based on FUNCTION (difficult in dogs)
      2. ACC/AHA: based on radiographs and clinical signs (most useful)
### American College of Cardiology/American Heart Assoc (ACC/AHA) Classification System

#### At risk for heart failure

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>STAGE A</td>
<td>No structural disease, but high risk for developing heart disease</td>
</tr>
<tr>
<td>STAGE B1</td>
<td>Asymptomatic disease, minimal remodeling</td>
</tr>
<tr>
<td>STAGE B2</td>
<td>Asymptomatic disease, significant remodeling</td>
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#### Heart failure

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<th>Stage</th>
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<td>STAGE C</td>
<td>Past or current signs/symptoms of CHF</td>
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<tr>
<td>STAGE D</td>
<td>End stage CHF, signs refractory to Rx</td>
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C. Staging system as a guide for therapy:

1. most therapy begins at Stage C, when clinical signs are present
2. exception: some therapies for DCM begin in Stage B1 or B2 if systolic dysfunction is detected
3. once in Stage C, continue medical therapy is required (cannot go back to B2)
4. system does NOT take arrhythmias into account: may be present and need to be treated at Stages B1-D
I. Goals of heart failure therapy
   A. Acute stabilization of critical patients
   B. Maintain quality of life chronically
      1. Maintain breathing comfort
      2. Maintain ability to exercise
      3. Avoid complications
   C. Extend the length of good quality life

II. Factors to consider when deciding on specific therapies
   A. Do the life-threatening clinical signs indicate congestive or low output (hypotensive) problems?
      1. Congestive Signs: require furosemide, mechanical drainage, oxygen
         a. Pulmonary edema
         b. Ascites
         c. Pleural effusion (less common in dogs)
      2. Low output signs: require fluid and inotropic support
         a. Hypotension
         b. Cold extremities
         c. Pallor, weak pulses, slow CRT
   B. Are the clinical signs typical of right-sided, left-sided or biventricular heart failure?
      1. Right-sided HF: jugular distension, hepatomegaly, ascites and/or pleural effusion - often require mechanical drainage acutely
      2. Left-sided HF: cough, coughing up foam, dyspnea, pulmonary crackles and cyanosis - requires oxygen and immediate diuretic therapy
   C. Presence of arrhythmia
      1. May require direct therapy
      2. May be the immediate cause of CHF
      3. Resolution may be required in order to resolve CHF
III. Which medications should I keep on hand?

A. **Acute:** oxygen, injectable furosemide, pimobendan

B. **Chronic:** oral furosemide, pimobendan, angiotensin-converting enzyme inhibitors (ACEI), spironolactone (other vasodilators for late stage patients: amlodipine, hydralazine)

C. **Anti-arrhythmic medications:** IV lidocaine, oral digoxin, diltiazem, sotalol and mexiletine

D. **Anti-anxiety:** injectable butorphanol or buprenorphine, midazolam

IV. Acute therapy of heart failure

A. Recognize general signs of acute heart failure

1. History: auscultably abnormal heart, dyspnea, abdominal swelling, cyanosis, exercise intolerance due to dyspnea or cough, pet may not have slept for several days

2. PE: weakness, depression, anxiety/panic and tachycardia (arrhythmias may be detected)

B. Recognize signs typical of left sided failure

1. Gallop rhythm, coughing up foam, dyspnea, harsh lung sounds or pulmonary crackles, left-sided heart murmurs

2. Problems to attack

   a. Dyspnea due to pulmonary edema:

      i. Oxygen: oxygen cage, mask, nasal cannula

         • Blood gas not needed

         • When in doubt, oxygenate

      ii. IV furosemide (2-4 mg/kg IV, repeat in ½ hour if no overt improvement)

         • Still the “go to” diuretic

         • Given IV, produces clinical effects within ~15-30 minutes

         • Provide urination opportunities

   b. Decreased cardiac performance: pimobendan (vasodilation/inotropic effects)

      i. Pimobendan: combined inotrope and vasodilator (0.3 mg/kg PO)

         • Indicated when left-sided CHF is present
• Oral medication, but effective within 1-2 hours after administration
  
i. Dobutamine: positive inotrope, given as continuous rate infusion
  
c. Stress related to discomfort and anxiety: sedation with (usually) opioids

C. Recognize signs typical of right sided failure
  1. Jugular distension, dull heart and breath sounds, hepatomegaly, dyspnea/open mouthed breathing if pleural effusion, positional discomfort if ascites and a right-sided heart murmur
  2. Problems to address
     a. Must acutely rule out pericardial effusion (by radiograph or echo)
     b. Relieve dyspnea/discomfort/anxiety related to severe fluid accumulations via centesis
        i. Pericardiocentesis: if needed, remove as much as possible, analyze
        ii. Thoracocentesis: remove as much fluid as possible, analyze
   (furosemide is not a substitute for centesis, but can be a helpful follow-up)
        iii. Abdominocentesis: remove approximately 2/3 of amount in abdomen

D. Additional Testing Recommended
  1. 6 second (or more) lead II (or equivalent) ECG strip
  2. Thoracic radiographs: if feasible:
     a. Should not be pushed in dyspneic animals unless diagnosis is in doubt and waiting may kill the patient
     b. Even single and crooked views can help!

V. Therapy of Chronic Congestive Heart Failure
   A. Medications to treat chronic left-sided heart failure due to CVD or DCM
      1. Diuretics: decrease the inflated circulating blood volume
         a. Furosemide: still the mainstay diuretic
         b. (Spironolactone: more of a neurohormonal modulator than diuretic)
      2. Neurohormonal blockade
         a. Angiotensin-converting enzyme inhibitors: block conversion of angiotensin I to angiotensin II, which blocks the ATII-induced vasoconstriction (relative vasodilation) and reduces ATII-induced production of aldosterone, decreasing sodium retention.

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b. Spironolactone: competitive blockade of aldosterone receptors, reduces effect of any aldosterone made through non-ATII mechanisms
c. Digoxin: modulates the reactivity of the sympathetic nervous system in heart failure and decreases “cross-excitation” of renin-angiotensin-aldosterone and vasopressin systems

3. Positive inotropes: increasing the contractile force of the heart
   a. Pimobendan: a newer positive inotrope that increases actin-myosin interaction by sensitizing the contractile proteins to the existing amount of intracellular calcium, rather than increasing intracellular calcium
   b. Digoxin: a weak positive inotrope that increases cross-bridging of actin and myosin fibrils in myocytes by increasing intracellular calcium

4. Vasodilators: decreasing afterload (arterial) and preload (venous)
   a. Angiotensin-converting enzyme inhibitors: decrease the amount of vasoconstriction rather than causing primary vasodilation. Effective but less powerful than direct-acting vasodilators.
   b. Amlodipine: direct arterial dilator, effective and long-acting. Less useful in acute heart failure due to prolonged onset of effects.
   c. Hydralazine: potent and direct-acting, leads to rapid and significant decreases in arterial pressure
   d. Pimobendan: often thought of as more of a positive inotrope, pimobendan has significant vasodilating properties due to the inhibition of phosphodiesterase III (PDE III and V) activity

B. Approach: choose medications based on underlying disease and clinical signs (=stage of heart failure)
   1. Underlying disease
      a. Adult onset left-sided diseases (chronic valvular disease or DCM) are treated the same EXCEPT for pre-clinical phases
      b. Adult onset right-sided diseases are treated based on individual disease (e.g. heartworm disease vs. pericardial effusion vs. chronic tricuspid insufficiency)
   2. Stage of Disease (ACC/AHA classification) for left heart failure
      a. Stage A: at risk for heart disease but no evidence of disease: no treatment
      b. Stage B1: heart disease is present but heart is not enlarged: no treatment
      c. Stage B2: heart disease is present with cardiac enlargement
         i. No treatment vs.
         ii. ACEI for chronic valvular disease vs.
         iii. ACEI + beta blocker (carvedilol) for DCM
d. **Stage C**: heart disease and heart failure are present: first offenders:
   TRIPLE THERAPY (furosemide, ACEI, pimobendan), + spironolactone

e. **Stage D**: repeat CHF, refractory CHF: adding inotropes short term,
   additional vasodilators, consider therapy for pulmonary hypertension if present

C. Recurrence of CHF after previously stable period: consider
1. Disruption of normal lifestyle (stress, boarding, change in exercise or weather)
2. Change in medication compliance or misunderstanding of medication
3. Occurrence of new arrhythmia
4. Development of complication: pulmonary hypertension, azotemia, diarrhea
5. If all previous ruled out…consider advancement of disease process and add appropriate medications
Confident Diagnosis and Treatment of Feline Heart Disease:  
Rebecca L. Stepien DVM, MS, DACVIM (Cardiology)

I. Heart disease vs. heart failure: same situation as in dogs  
   A. The purpose of the cardiovascular system is to maintain adequate blood pressure (BP)  
      to supply organ perfusion.  
      1. Heart disease: physical or functional abnormality of component(s) of the CV  
         system  
      2. Heart failure: CO is inadequate to support BP despite normal hydration  
         a. Low cardiac output = signs of low BP (hypotension)  
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      3. Compensatory mechanisms: renin-angiotensin-aldosterone, sympathetic  
         nervous system, antidiuretic hormone  
   B. Why bother?  
      1. HEART DISEASE----(usually)------→ MONITOR  
      2. HEART FAILURE------(almost always)---→ TREAT

II. Detection of asymptomatic feline heart disease: not straightforward  
   A. ~1/4 to 1/3 of overtly healthy cats have heart murmurs  
   B. approximately 50% of cats with murmurs have diagnosable cardiac disease  
   C. So how can we tell who is who? – look for additional abnormal findings:  
      1. gallop rhythms are specific for myocardial abnormalities (permanent or  
         temporary)  
         a. Gallop rhythm = “stiff ventricle”  
         b. Detection of gallop rhythm increases the chances that a heart murmur is  
            indicative of actual disease  
      2. Arrhythmias: if present, increase the chance of true cardiac disease  
   D. Why should we worry about normal cats with heart murmurs?  
      1. Client interest  
      2. Preanesthesia or pre-fluid therapy  
      3. Identify systemic diseases by identifying its cardiac repercussions  
      4. We have the chance to rule OUT heart disease  
   E. NT-proBNP as a tool to identify heart disease in cats  
      1. Can identify heart disease in asymptomatic cats
2. Can differentiate heart failure from other causes of dyspnea
3. NOT helpful to tell WHICH cardiac disease is present

F. When is an echocardiogram needed?
   1. Client interest
   2. Breeding or pedigree questions in breeds at risk
   3. Fluid therapy or anesthesia is anticipated
   4. We know some sort of heart disease is present, but don’t know if it’s bad enough to cause clinical signs
   5. We have the chance to rule OUT heart disease
   6. We have the chance to detect intracardiac thrombi

G. Left ventricular outflow tract obstructions as a cause of heart murmurs
   1. Velocity of LV outflow tract “pulls” MV leaflet into outflow tract in systole, leading to obstruction to outflow and secondary mitral insufficiency
   2. May be related to congenital abnormalities of the mitral valve leaflet
   3. May be seen in cats with and without LV hypertrophy
   4. Clinical importance remains unclear, but may be a contraindication to use of pimobendan (a pro-contractility medication)
   5. Beta blockade (typically atenolol) has been used to decrease HR and contractile function in order to decrease dynamic obstruction – clinical advantage of this strategy is unclear. May be of some theoretical advantage in hyperthyroid cats or cats with significant arrhythmias

H. Summary: Diagnostic evaluation of asymptomatic cats
   1. Physical examination
   2. Thoracic radiographs: heart size gives information on staging
   3. ECG if arrhythmia is detected
   4. Echocardiogram
   5. NT-proBNP may precede/replace echo in some cats
   6. Appropriate testing for systemic disease in at risk cats (e.g. T4, renal)

III. Therapy of pre-clinical (asymptomatic) cardiomyopathy
   A. Diltiazem no longer recommended unless atrial fibrillation is present
   B. Minimal data that any specific therapy changes clinical signs or survival
      1. Clopidogrel may be considered if left atrial enlargement is severe, left atrial “smoke” is present OR if an intracardiac thrombus is found (echo findings)
2. Beta blockers may be useful in hyperthyroid cats, LV outflow obstruction cats or cats with tachyarrhythmias
3. ACEI may be useful in cats on the verge of CHF (severe LA enlargement)

IV. Diagnosis of congestive heart failure in cats
   A. History is similar regardless of type of cardiomyopathy
      1. Low output may only be noticed as hiding
      2. Congestion: tachypneic, dyspneic (ascites and coughs are rare)
   B. Physical examination is similar regardless of type of cardiomyopathy
      1. General “not well” look, weight loss, dehydration if inappetent
      2. Evidence of thromboembolism
      3. Distended jugular veins in CHF with pleural effusion
      4. Gallop rhythm
      5. Differentiate effusion from edema with stethoscope
   C. Radiographs are similar regardless of type of cardiomyopathy
      1. Cardiomegaly (but may be obscured by pleural effusion)
      2. Non-specific edema patterns (distribution unpredictable)
      3. Pleural effusion is common

V. Treatment of congestive heart failure in cats is similar regardless of etiology
   A. Manage acute hypoxia: oxygen, thoracocentesis if needed
   B. Manage fluid accumulation: thoracocentesis, IV furosemide, ACEI, spironolactone
   C. Manage combined systolic/diastolic dysfunction: dobutamine or pimobendan
   D. Manage arrhythmias/thrombi: diltiazem, beta blockers, clopidogrel or low molecular weight heparin

VI. Pimobendan use in cats: Not approved by FDA
   A. Reason to believe: pimobendan is prosystolic and prodiastolic
   B. No studies of efficacy published to date
   C. Several studies implying safety when used in CHF cats
   D. May be contraindicated in LV outflow tract obstruction cats
   E. Anecdotal information implying better survival
VII. Summary: Therapy of CHF in cats
   A. Remove fluid accumulations when severe
   B. Chronic oral medication: “Triple therapy plus one”
      1. Furosemide: 1-2 mg/kg PO q 12 hrs
      2. ACEI: 0.5 mg/kg PO q 12 hrs
      3. Pimobendan: 1.25 mg per 5 kg cat PO q 12 hrs
      4. Spironolactone: 1-2 mg/kg PO q 12 hrs

VIII. Initial diagnostics for first time CHF
   A. Physical: Severity of signs, look for precipitating cause/concurrent disease
   B. Quantitate “how much” failure: radiographs
   C. Echocardiogram: eventual diagnosis of disease, changes since previous echo
   D. Labwork
   E. ECG if indicated

IX. Monitoring
   A. One week recheck: recheck rads, labwork to define new stable medication level
   B. Once stable on meds: approximately every 3 months
      1. Recheck previous concerns (specific for each cat)
      2. Check renal function/potassium in cats on chronic diuretics
   C. Client information
      1. Drug information for client (handouts are helpful)
      2. Need to notify veterinarian if cat stops eating
      3. Monitor respiratory rate: normal 18-24, call if rate > 30 or increases by ~ 10 per minute
One of the first questions a client will ask their veterinarian after their pet receives a diagnosis of heart disease is “How long will she live?”. As clinicians and human beings, we tend to remember and prognosticate based on the most extreme cases or the last disastrous case, we are swayed by our feelings for the pet and the clients and are concerned about what decisions a client might make if we misstate the situation. Hard scientific data on survival of cats and dogs with naturally-occurring heart disease is available, but often represents a small population of purebred animals with specific diseases peculiar to the breed, animals treated in state-of-the-art facilities or with unlimited funds or animals treated with a specific medication (with no information on survival of the same disease treated differently). Despite this daunting list of problems related to prognostication, veterinarians perform this service every day, with profound consequences. A closer examination of common findings affecting prognosis in cardiovascular disease allows identification of key moments in the pet’s course of disease and allows the clinician to individualize a prognosis to the patient.

I. The Clinician’s Role in Communicating Prognosis

As veterinarians, we are trained to consider medical factors in patients with disease. As the professional on a given case, we are responsible for relaying as much information as we can about the patient’s medical situation.

   A. Consider the disease

      1. Identify the disease or condition to the fullest extent possible

      2. Know therapies

         a. Is a “cure” possible? (often the case with a surgical disease)

         b. Is medical therapy likely to help?

         c. Does the patient need to see a specialist?

      3. Know the prognosis in general terms (e.g. weeks, months, years)

         a. Frequently asked question: how will therapy change the prognosis?

         b. Will the patient’s other diseases change the prognosis?
4. Critical clinician pitfalls
   a. Giving a prognosis based on most recent case (may be overly optimistic
      OR overly pessimistic)
   b. Making assumptions about the client’s understanding
      i. Assuming that the client understands the terminology
      ii. Assuming that the client understands the concept
      iii. Assuming that the client is not distracted
         • Children in the room
         • Memories of family member/previous animal with similar
           condition
   c. Taking responsibility for client’s emotions (“this will be too much for
      him to deal with”)
   d. Assuming knowledge of the client’s financial status or interest in further
      testing or therapy

5. Maximizing client understanding
   a. Serious conversations involving important diagnostic and prognostic
      questions occur most optimally in quiet surroundings and without the
      pet present in most situations
   b. Information should be given in bite size “chunks” with frequent verbal
      checks of understanding
   c. Prioritize most important information
   d. Avoid prejudging client’s level of financial commitment or what would
      be “best” for the client’s perceived situation – give all options

B. When full information is not available at the time of discussion: delay is better than
   false information!

II. Interpreting published survival information
   A. Remain up-to-date on published data regarding survival times in common diseases
   B. Median survival time is the most frequent reported indicator of survival
      1. Definition: Median survival time: interval at which 50% of the patients are
         dead
a. 50% are still alive at this date
b. Usually no additional indicator of how long the remaining 50% lived
c. *Is not the “average” survival time*

2. Effect of euthanasia on survival studies
   a. Euthanasia is usually treated as death from disease, but this is often not the case
      i. Diseases with expensive therapy will have artificially shortened median survival times due to euthanasia related to cost of therapy
   ii. In diseases in which survival of an initial episode is critical (i.e. acute aortic thromboembolism in a cat), many animals die or are euthanized immediately but those who do survive live significantly longer. In these animals, median survival time in typical studies is an inaccurate reflection of a given animal’s prognosis.
   iii. If euthanasia is based on poor prognosis, the prognosis worsens

### III. The Client’s Role in Affecting Prognosis

Many pets with cardiovascular disease require therapy for their clinical signs (e.g. cough, difficulty breathing). Medications for these problems can vastly improve the quality of the patient’s life, but come at a cost to both client and patient. Most symptomatic cardiac diseases are ultimately fatal, but survival is artificially shortened when the client perceives that the recommended medications are making the patient more ill, are not improving the situation, cost too much, are too difficult to administer or are causing intolerable side effects in the patient or the household.

A. Typical problem points with client perceptions regarding heart failure therapy
   1. Therapy is typically lifelong
   2. Narrow therapeutic range for many drugs (increased risk of toxicity)
   3. Different time schedules for drugs
      a. As needed (furosemide)
      b. On schedule (antiarrhythmic drugs)
4. Cost: the better the patient does (longer survival), the more therapy will cost
5. Desire for immediate normality

B. Preventing these sorts of problems from affecting the patient’s survival:
   1. informing the client in advance (and preferably in writing via a prepared
      handout)
      a. drug’s desired effect
      b. possible side effects
      c. ways to cope with the side effects
   2. assuring the client that changes in drugs and doses are common in the first two
      weeks of therapy and the need for changes is not a “bad sign”
   3. informing the owners which drugs need to be given on a regular schedule
      (usually drugs in which a stable blood concentration is necessary for optimal
      effects, e.g. anti-arrhythmic drugs) and which drugs can be safely administered
      an hour early or late (drugs with a very long half-life e.g. digoxin, or drugs not
      intended to attain steady state e.g. furosemide)
   4. prioritizing medications if cost or difficulty with administration is an issue

IV. Prognosis in Cases of Congenital Heart Disease

Little information has published regarding the natural history of most types of congenital heart

disease, especially if the disease is untreated. Survival information regarding patients with
treated patent ductus arteriosus (PDA) or dogs with subaortic stenosis (SAS) has been
published. Most other congenital heart diseases such as mitral dysplasia (MVD), tricuspid

dysplasia (TVD), ventricular septal defect (VSD) atrial septal defect (ASD) or pulmonic
stenosis (PS) follow some general rules that can be adapted for the individual animal. Because
of the paucity of specific survival information in many congenital heart diseases, the clinician
is required to concentrate more on analyzing “good signs” and “bad signs” regarding a
patient’s condition and projected survival rather than concentrating on remembering specific
time frames (the following points apply to diseases that are not correctable with surgery or
other invention).
A. Findings associated with a better prognosis
   1. Well-grown, energetic and inquisitive patient, the same size as littermates
   2. No clinical signs of disease (exercise intolerance, cyanosis, difficulty breathing, ascites)
   3. No concurrent disease (e.g. hydrocephalus, porto-systemic shunt)
   4. Loud murmur is good if a VSD is present
   5. No arrhythmias
   6. Radiographic and echocardiographic findings show minimal cardiac enlargement and contractile function is normal

B. Findings associated with a worse prognosis
   1. Stunted, lethargic or excessively quiet patient, smaller than littermates
   2. Clinical signs of heart failure, cyanosis or arrhythmias are present
   3. Concurrent disease is known or suspected
   4. Loud murmurs are bad in stenotic lesions (PS, SAS), regurgitant lesions (MVD, TVD) or if an ASD is present
   5. Radiographic and echocardiographic findings show major anatomic abnormalities accompanied by significant chamber enlargement

C. Overview of survival: General statements for congenital heart disease
   1. Animals with severe clinical signs of non-surgical disease at a young age are more likely to have a very limited life span (weeks to months from the time of diagnosis).
   2. Animals with severe anatomic changes and cardiac enlargement but no clinical signs might be expected to live several years (exception: some dogs with severe tricuspid valve dysplasia may live to be middle aged despite horrifying echocardiographic findings).
   3. Animals with less severe anatomic changes and minimal cardiac enlargement may live well into middle age, and animals with small defects and relatively normal cardiac size radiographically and echocardiographically may live a normal life span.
   4. In all cases, the clients should be warned that complications such as arrhythmias or vegetative endocarditis might shorten the animal’s life span unexpectedly.
5. Cats are survivors - some cats with large ventricular septal defects at a young age with no signs of heart failure may “grow into” their defect.

D. Specific disease conditions

1. Patent ductus arteriosus

PDA remains the “best” congenital heart disease to have – surgical ligation or Amplatz device occlusion of the ductus usually results in a relatively normal life span for the pet. Exceptions include animals with other concurrent congenital abnormalities or severe myocardial failure (systolic dysfunction) and congestive heart failure (CHF) accompanying the PDA. These animals may recover fully after ductal ligation, or may require lifelong therapy and still have a shortened life span. Most untreated PDAs will result in CHF within 1-3 years; some untreated animals may develop pulmonary hypertension at an early age that reverses flow in the PDA and renders it untreatable surgically (i.e. reversed PDA). Rarely, animals will live a normal life span with a very small untreated ductus.

2. Subaortic or aortic stenosis

Subaortic stenosis is a common congenital heart defect and varies widely in severity. In 1994, a comprehensive survival study grouped dogs with SAS according to severity of obstruction. Dogs were divided into groups with “mild” (transvalvular gradient < 35 mmHg), “moderate” (gradient 36-80 mmHg) or “severe” (> 80 mmHg) stenosis.

a. Dogs with mild obstructions usually remained asymptomatic and had normal life spans.

b. Dogs with moderate obstructions were more likely to develop clinical signs of SAS, but still usually lived into middle age. The complications of left heart failure or infective endocarditis occurred more frequently in these groups than in the more severely affected dogs, perhaps because mild or moderately affected dogs lived longer.

c. Severely affected dogs had a median survival significantly shorter (18.9 mos) than mildly (51.1 mos) or moderately (30.5 mos) affected dogs.
Severely affected dogs were 16 times more likely to die a sudden death than less severely affected dogs.

3. **Pulmonic stenosis**
   a. Severity usually defined by outflow gradient (Doppler echo)
      i. Mild: 10 to 49 mmHg
      ii. Moderate: 50 to 80 mmHg
      iii. Severe: more than 80 mmHg
   b. Survival without intervention
      i. 50% of severely affected dogs, 10% of moderately affected dogs and 4% of mildly affected dogs will die of cardiac causes
      ii. Gradients > 60 mmHg predict cardiac death with Se 86%, Sp 71%
      iii. Clinical signs of lethargy increase chance of cardiac death
   c. With balloon valvuloplasty
      i. Improvement in gradient still present at one year in most dogs
      ii. Relief of clinical signs is typical, so may prolong life

V. **Prognosis in Case of Acquired Heart Disease**

As is the case in congenital heart disease, several broad generalizations can be made that affect survival in pets with acquired heart disease in which the underlying cause is unknown or unfixable.

A. Situations associated with prolonged survival:
   1. Minimal cardiac enlargement
   2. No CHF or arrhythmias
   3. No concurrent diseases that affect drug therapy (e.g. renal failure)
   4. Brisk response to initial therapy with minimal side effects
   5. Hearty appetite
   6. Dedicated, observant and creative caregiver

B. Situations associated with decreased survival
   1. Presence of CHF
      a. Indicates that disease has been present chronically
b. Indicates that the heart’s ability to compensate has been exceeded
c. Introduces the effects of medication to survival

2. Significant cardiac enlargement
3. Decreased systolic function
4. Severe anatomic derangements
5. Disease is associated with catastrophic complications (e.g. aortic thromboembolism)
6. Concurrent diseases limit drug therapy or lead to side effects
7. Drugs are poorly tolerated or administered inconsistently

C. Specific diseases

1. Feline hypertrophic cardiomyopathy (HCM)
   Asymptomatic cats with idiopathic HCM can span a wide range of phenotypes and therefore, the asymptomatic form of the disease has an unpredictable course.
   a. Cats with echocardiographically evident HCM but no clinical signs have a median survival time of 5 years or more
   b. Cats with heart failure have a shorter median survival, ranging from 92-563 days. Survival with CHF appears to have improved with use of pimobendan in some animals.

2. HCM with thromboembolic complications
   Development of aortic thromboembolism (ATE) severely decreases survival times for cats with HCM for several reasons.
   a. Cats that develop ATE as a complication of HCM frequently have severe cardiac pathology and CHF, contributing to decreased survivability.
   b. The clinical signs of ATE are often dramatic and painful, with a history of limited success with therapy and a well-publicized poor prognosis for recovery. This constellation of findings and facts often leads to rapid euthanasia in affected animals.
   c. Published survival times for cats with HCM and ATE range from 61 to 184 days
d. When all feline cardiac diseases are included, the mean survival for cats with ATE who survived the initial thrombotic event (37% of cats) was 11.5 months, with 28% of cats dying during the initial event and 35% euthanized.

3. **Feline atrial fibrillation (AF)**

Atrial enlargement provides a substrate for the occurrence of AF in all species and is frequently associated with significant heart disease and heart failure.

   a. In a recent retrospective study of 50 cats with AF
   b. Most common underlying disease: restrictive cardiomyopathy
   c. 56% of cats with AF were presented with signs of heart failure
   d. In 23% of cats studied, AF was an incidental finding.
   e. Survival was longer than expected, with an overall median survival of 165 days.
   f. Presence of clinical signs at the time of diagnosis did not have a significant effect on survival.

4. **Canine mitral valve insufficiency (MVI)**

The prognosis for survival in dogs with MVI due to degenerative valve disease is generally better than survival in dogs with dilated cardiomyopathy.

   a. Typically, the onset of the heart murmur of MVI is first noted in middle age (approximately 6-9 years old).
   b. Data drawn from studies of angiotensin converting enzyme inhibitors:
      i. Median time from discovery of heart murmur to heart failure: ~1300 days (~3.5 years)
      ii. Median survival with pimbendan therapy (CHF → death): 267 days (8-9 months)
   c. Degree of cardiomegaly is related to projected time to heart failure; sudden increases in left atrial size predict CHF within several months

5. **Canine dilated cardiomyopathy (DCM)**

The presence of CHF at the time of diagnosis has a significant negative effect on survival in dogs with idiopathic DCM.
a. Overall survival rate of approximately 20% at one year
b. With CHF and pimobendan therapy, the median survival is approximately 4.75 months
c. Approximately 25% of dogs with CHF will survive to one year

While these statistics are daunting, it is important to remember that many dogs are euthanized soon after diagnosis due to poor prognosis rather than because of severe clinical signs. This group is likely to skew the survival statistics downward. Doberman pinscher DCM is particularly malignant with median survival times post-diagnosis of approximately 6 weeks; about 20% of affected dogs die suddenly during this period. Newer medications (e.g. pimobendan) are increasing survival times in DCM dogs.
The dictionary describes a “misconception” as a “mistaken idea”. In clinical medicine, most cases are complicated enough that many pieces of information must be integrated and coordinated, and when large numbers of diagnoses, medications and concurrent problems are involved, it is almost impossible to proceed without some questions or misunderstandings.

In cardiovascular medicine, some misconceptions are well-entrenched, perhaps because the misunderstanding of the topic is widespread, the situation in cats/dogs differs from conventional wisdom for similar human patients, or because the facts are counter-intuitive. The following misconceptions are common, widespread, and can negatively affect patient care and the clinical success of therapy.

Please note that the following ideas are FALSE:

1. Angiotensin-converting enzyme inhibitors cause renal failure, but benazepril is less “toxic”.
   - Due to the mechanism of action of these medications, most changes in renal values and electrolytes associated with their use are due to poor renal perfusion and can be avoided by only using ACEI in hydrated patients.
   - Acute CHF can be treated without ACEI in the short term, and these long-term medications can be started safely when the patient is hydrated and eating.
   - Enalapril and benazepril do not differ in their mechanism of action, only in their metabolic disposition. Enalapril is eliminated primarily via the kidneys, and benazepril via the liver. Therefore, benazepril is recommended in patients with known renal disease to avoid decreased elimination.

2. Dental disease is a frequent cause of canine valve disease.
   - Dental disease is a risk factor for many co-morbidities, but valvular heart disease is not one of them. There has been a single well-documented case of endocarditis following a dental procedure, and no good data in dogs regarding any relationship between dental disease and degenerative valve disease, the most common type of heart disease in dogs.
   - Bacteremia during dental procedures has been well-documented and poses a risk to many vital organs, especially the kidneys. This fact provides a rational argument for peri-procedural antibiotic administration.
   - Supportive fluid administration and limiting the length of the procedure can protect the patient from renal complications post-anesthesia.
3. Patients with heart disease cannot be vaccinated or anesthetized safely.

- Patients with active congestive heart failure have limited ability to respond to physical or medical challenges and most routine elective procedures (e.g. vaccinations, dips and extensive grooming, dental procedures) should be postponed until the patient is clinically stable.
- Patients without CHF or those who are being treated chronically and are stable may receive all routine care as usual.
- Stable cardiovascular patients can be anesthetized safely, using cardiac-friendly medications and good monitoring. Xylazine, medetomidine and dexmedetomidine are not appropriate for patients with stable or unstable cardiac conditions, and ketamine should be avoided if possible.

4. Chronic cardiac medications should not be given on the morning of anesthesia.

- The day of anesthesia, with its associated cardiorespiratory challenges, is not a good day to suddenly stop chronic cardiac medications.
- Cardiac medications (except angiotensin-converting enzyme inhibitors) should be given as usual but without food on the morning of anesthesia. The patient should have access to water until induction.
- Fluid therapy during anesthesia is an important contributor to renal-safe anesthesia in cardiac patients and the usual standards for fluid administration to anesthetized patients should be followed.
- Close attention to blood pressure during anesthesia allows early detection of hypotension.

5. Elderly patients are like everyone else, only older.

- Geriatric patients are often surprisingly resilient to heart disease and therapy for heart disease, so much so that the clinician often forgets how elderly patients differ from younger patients.
- Older patients are more likely to have concurrent disease that limits drug metabolism or appetite.
- Older patients are often thin and may be debilitated, and have difficulty tolerating “usual” doses of medication.
- Older patients may receive multiple non-cardiac medications, which may provide opportunities for adverse drug interactions to occur. NSAIDs have multiple side effects that may be exacerbated by use of cardiac medications and should be used with great care, if at all, in these fragile patients.
- The advantage of working with geriatric patients is that they are usually highly valued as part of the family and often have very dedicated owners.
6. ECG machines and monitoring devices are more accurate than people.

- Reading ECGs is challenging for human beings. It’s hard to expect that machines can do better at counting heart rate during arrhythmias, and at diagnosing arrhythmias, and monitors will sometimes arrive at interesting diagnoses such as “signal detection failure”.
- Heart rate identification is the easiest part of assessing rhythm abnormalities, and should be performed by a human being to avoid machine error.

7. Patients with abdominal fluid need an exploratory for liver disease.

- Abdominal distention due to fluid accumulation without the presence of a heart murmur is often misdiagnosed as non-cardiac in origin.
- Pericardial effusion is a very common cause of accumulation of abdominal fluid, and should be ruled out prior to further diagnostics for liver disease.
- Pericardial effusion can cause sudden death if unrecognized; a survey lateral thoracic radiograph is indicated for any patient with abdominal fluid accumulation.

8. Patients usually only have one disease at a time, therefore, clinical abnormalities in a patient with heart disease are due to that disease.

- It’s tempting to assume that once a patient has serious disease of any major organ, most new clinical signs will be referable to that disease.
- In cardiac patients, the extended survival that is becoming the norm for CHF patients allows time for other diseases to develop. Some of these problems may have a clinical presentation that looks like cardiac disease.

9. Assessing for systemic hypertension can be difficult; luckily it’s rare and causes few problems – BP measurement is more trouble than it’s worth.

- Systemic hypertension can be a clinically silent disease and requires a high index of suspicion for diagnosis.
- Unrecognized systemic hypertension can result in permanent blindness, progressive renal deterioration and unexpected congestive heart failure after fluid administration.
- The presence of retinal detachment, retinal hemorrhage or intraocular hemorrhage (hyphema) should be considered a strong indication for measurement of blood pressure in cats and dogs.
- Renal disease, especially proteinuric renal disease, is a significant risk factor for development of systemic hypertension.
- Systemic hypertension in cats tends to be stable once successfully treated, but in dogs, hypertension tends to be progressive over time and may require additional medication after an initial period of stable response to antihypertensive medications.
10. Congestive heart failure patients have a poor prognosis, and since they’re often old, it’s better to “not put them through” therapy for the disease.

- Newer treatments for CHF that are applicable in both dogs and cats have significantly prolonged survival for veterinary patients with congestive heart failure, and most of these medications have very tolerable side effect profiles.
- Because these medications improve quality of life, patients can enjoy longer periods of clinical stability and therefore, prolonged survival.
- Many problems that arise with first therapy of CHF can be fixed with medication adjustment or substitution.
- Prolonged high quality of life survival in these patients enhances their lives with their owners and provides ample opportunity for veterinarians to both educate and learn from their clients.