Feline Myocardial Disease: Prevalence, Detection, and Diversity
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Screening for feline cardiac disease

Physical exam findings

Murmur
- The presence of a murmur indicates turbulent blood flow within the heart or great vessels
- Most studies support that patients with a murmur are more likely to have cardiac disease than those without
- Approximately 2x probability of being affected with HCM
- Most recently: 43% of control cats and 80% of cats with occult myocardial disease had murmurs
  - Mild RVOT obstruction is a common cause of benign murmur not necessarily associated with cardiac disease

Gallop
- The presence of a gallop is indicative of increased atrial pressures and/or increased stiffening of the ventricles
- The presence of a gallop greatly raises the concern for significant underlying cardiac disease, although it is an insensitive screening parameter
  - In a recent study of 227 cats – 17% of affected cats and 0% of normal individuals had a gallop
  - HR of 1.8 for risk of cardiac death

Arrhythmia
- Extrasystoles or tachyarrhythmias are supportive of underlying myocardial disease
  - HR 3.2 for risk of cardiac death
Presence of any of these abnormalities should prompt further evaluation

Prevalence of hypertrophic cardiomyopathy is 15% in our region

Auscultation findings are insensitive and non-specific
- Abnormal auscultation – Sens 33% and Spec 87.4% for Dx of HCM
- Abnormal auscultation- PPV 31% (probability of disease w/ murmur)

Thoracic radiographs
Hypertrophic cardiomyopathy is the most common myocardial disease of the cat and is defined by concentric hypertrophy in which the cardiac silhouette is not expected to increase

Generally low sensitivity to detect hypertrophic disease in the absence of left atrial enlargement
- Even in the presence of left atrial enlargement (LA:Ao >1.6) the sensitivity of thoracic radiographs to detect these changes are relatively low (up to 70%)
- Vertebral Heart Scale
  - Add dimensions of long and short axis
    - +/- 0.3 in cats
    - i.e. >8.0 is enlarged

NT-proBNP
Quantitative NT-proBNP
Superior screening tool for occult feline myocardial disease
- Study of 113 cats with occult cardiomyopathy and 114 normal cats
  - T-proBNP of >99 provided a 100% sensitivity and 71% specificity
- Study of 99 cats with occult cardiomyopathy and 102 normal cats
  - NT-proBNP of >100 provided a 92% sensitivity and 94% specificity

SNAP Test NT-proBNP
- In-clinic subjective test that is a sensitive screening tool for severe disease
  - Sensitivity for moderate/severe disease is 84%
  - Low sensitivity for mild and questionable sensitivity for moderate disease

Echocardiogram
Gold standard for the diagnosis and characterization of feline cardiac disease
- Accurate diagnosis, assessment of systolic function, left atrial size and pressure estimates, and auricular appendage velocities associated with thromboembolic risk

Feline myocardial disease is diverse
- Hypertrophic obstructive cardiomyopathy
- Restrictive cardiomyopathy
- Unclassified cardiomyopathy
- Arrhythmogenic right ventricular cardiomyopathy

**Works cited**

Pulmonary Hypertension classification by etiopathogenesis

- Primary Pulmonary Hypertension (Rare in Veterinary Medicine)
- Secondary Pulmonary Hypertension:
  - Pulmonary Disease (Reactive/Vasoconstrictive)
    - COPD
    - Pulmonary fibrosis
    - Reactive vasoconstriction
  - Flow mediated vasoconstriction
    - PDA, VSD, ASD, other
  - Left heart disease (Post-Capillary)
    - Mitral valve disease
    - Cardiomyopathy
    - Cor triatriatum sinister (Fel)
  - Thromboembolic (Pre-Capillary)
    - PTE
      - PLN, Cushing’s, Neoplasia, Sepsis/DIC, IMHA, other
    - Heartworm disease
  - Diagnostics
    - Signalment
      - Middle age/older small breed dogs
        - Exception of congenital heart disease
    - Clinical Signs
      - Cough
      - Dyspnea
      - Lethargy/weakness/exercise intolerance
      - Syncope
        - Clinical signs may be caused by the elevated pulmonary pressures and/or due to the underlying etiology
    - Physical Exam Findings
      - Variable non-specific findings
      - Reflective of both the degree of pulmonary hypertension and underlying etiology
        - Systolic murmur (R>L)
        - Split S2
        - Varying degrees of dyspnea
        - Pulmonary crackles
        - Cyanosis
        - Ascites
    - Thoracic Radiographs
      - Insensitive
      - Radiographic signs can be subtle despite severe elevations in pulmonary pressures
      - Frequently radiographic changes associated with underlying etiology complicate interpretation
        - Pulmonary disease, left sided heart disease, etc.
      - Supporting Radiographic Signs include:
        - Pulmonary arterial dilation/tortuosity
        - Right heart enlargement pattern
    - NT-proBNP
      - Significant elevations associated with pulmonary hypertension
        - Positive correlation between severity of pulmonary hypertension and NT-proBNP concentrations
        - Non-specific change – does not discern from left heart disease
    - Electrocardiogram
- Right heart enlargement pattern
  - Right axis deviation
  - Deep S waves in lead II
  - P-pulmonale

- Echocardiogram – 2D
  - Septal flattening during systole
  - Right ventricular pressures exceeding left ventricular systolic pressures
  - Right ventricular hypertrophy
    - Concentric or mixed eccentric/concentric
  - Pulmonary artery dilation
  - PA:Ao >1.0
  - Presence of heartworms or thrombi

- Echocardiogram – Doppler
  - Tricuspid regurgitation velocity
    - Modified Bernoulli equation
    - Gives accurate estimate of systolic pulmonary pressures
      - Add estimated RA pressure (5-10mmHg)
  - Pulmonic insufficiency velocity
    - Gives accurate estimated of diastolic pulmonary pressures
  - Pulmonary artery Doppler flow profiles.
    - Normal flow profiles more symmetric with gradual acceleration
    - Acceleration time (AT) decreases with increased pulmonary pressures
  - Tissue Doppler
    - Inversion of normal Tissue Doppler pattern of the tricuspid valve annulus provides supportive evidence for pulmonary hypertension

**Pulmonary hypertension treatment**

***Identify and treat the underlying etiology!***

- **Pulmonary Disease (Reactive/Vasoconstrictive)**
  - COPD
  - Pulmonary fibrosis
  - Reactive vasoconstriction secondary to edema

- Flow mediated vasoconstriction
  - PDA, VSD, ASD, other

- **Left heart disease (Post-Capillary)**
  - Mitral valve disease
  - Cardiomyopathy
  - Cor triatriatum sinister (Fel)

- **Thromboembolic (Pre-Capillary)**
  - PTE
  - PLN, Cushing’s, Neoplasia, Sepsis/DIC, IMHA, other

- **Heartworm disease**

- **Secondary to Pulmonary Disease**
  - Ideally managed by an internist with initial pulmonary work-up
    - Frequently unstable/dyspneic on initial evaluation and empiric therapy started
  - Oxygen support as indicated
  - Empiric antibiotic therapy
  - Bronchodilators
    - Theophylline
    - Terbutaline
  - Steroids
  - Inhalant therapy
    - Fluticasone
    - Albuterol

- **Secondary to Left Heart Disease**
- Manage Congestive heart Failure
  - Oxygen support as indicated
  - Preload reduction
    - Diuretics
  - Afterload reduction
    - ACE-inhibitors
    - Nitroprusside
    - Amlodipine
      - May provide adjunctive pulmonary vasodilation
  - Pimobendan
    - Balanced inodilator
    - Reduces left atrial pressures
      - (Post-capillary pulmonary hypertension)
    - Phosphodiesterase III inhibitor
    - Pulmonary vasodilation via enhancement of adrenergic relaxation
- Secondary to Thromboembolic Disease
  - Oxygen support as indicated
  - Inhibit further clot formation
    - Anti-platelet therapy
      - Low dose Aspirin (1-5mg/kg)
        - Irreversible COX inhibition
      - Clopidogrel (Plavix) (2-3 mg/kg QD)
        - ADP receptor antagonist
    - Anti-coagulant therapy
      - Unfractionated Heparin
        - Nomogram in Kirk’s Current XIV
        - AT-III required for efficacy
        - Effective in face of PLN/PLE?
      - Low Molecular Weight Heparin
        - Enoxaparin 1mg/kg q12hr
        - Dalteparin 150U/kg q8hr
          - Pharmacokinetic and pharmacodynamic data is limited for the use of these drugs in the canine patient
      - Pentasaccharides (Fondaparinux)
        - Insufficiency data for clinical use
- Pulmonary Arterial Vasodilation and Modulation
  - Phosphodiesterase V inhibitors
    - Sildenafil 1-2mg/kg q8hr
    - Tadalafil
  - Nitric Oxide Pathway
    - L-Arginine
      - NO precursor
        - 250-500mg PO TID
  - Endothelin Antagonists
    - Ex. Bosentan, ambrisentan
    - SSS
      - Concern for liver toxicities
  - Prostacyclin Analogs
    - Epoprostenol – CRI with ambulatory pump
    - Treprostinil – IV or frequent SQ injections
      - Methods of delivery prohibit long term use in veterinary medicine
  - Theophylline (5-10mg/kg BID)
    - Avoid diaphragmatic fatigue and respiratory arrest
    - Improve ventilation
Summary

- Infrequently a single underlying etiology
  - Overlap and combined therapy generally required in management
  - Identifying and treating the underlying etiology is paramount for long term treatment success
  - Severe/symptomatic pulmonary hypertension carries a guarded prognosis
The Coughing Canine: 
Cardiac vs. Respiratory
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Coughing is a common, but not universal, clinical sign of congestive heart failure in dogs. Dog breeds commonly affected by degenerative valve disease that may progress to heart failure are also commonly affected by upper and lower airway diseases that can result in coughing and can pose a diagnostic challenge. In this lecture we will review the utility of readily available diagnostics to accurately discern the underlying etiology of coughing in the canine patient in order to develop a targeted and more effective treatment plan.

Coughing is defined as a sudden, forceful expiration against a closed glottis. The coughing reflex is triggered by irritation of the pharynx, upper airways, or lower airways. Less commonly, diseases involving the pleura, pericardium, diaphragm, nasal passages/sinuses, or mediastimum can result in coughing. Other paroxysmal respiratory/pharyngeal/gastrointestinal clinical signs, including sneezing, gagging, panting, labored breathing, reverse sneezing, retching, and vomiting can mimic or otherwise be confused with coughing.

The coughing reflex is triggered by mechanical or other noxious stimuli stimulating stretch receptors or C-fibers in the pharynx, upper or lower airways, pulmonary parenchyma, pleura, and diaphragm. Afferent fibers travel within the vagus nerve and go to the medulla and the cough center. Efferent signals travel via the vagus, phrenic, and spinal motor nerves to effect the coughing response.

Coughing can be caused by inflammatory or infectious pharyngeal/airway/pulmonary diseases, airway injury, physical airway factors (e.g. tracheal collapse), neoplasia involving the pharynx/airways/lungs/mediastinum/thoracic wall, and cardiovascular disease (pulmonary edema, mainstem bronchial compression by enlarged left atrium, noncardiogenic pulmonary edema, pulmonary emboli).

Patient history
Aspects of the onset, chronicity, and nature of a dog’s cough can give indications as to whether the cough is likely related to CHF. Coughing related to CHF may come on gradually or acutely, but generally has a brief (days to several weeks) history. A longer duration of coughing is generally associated with airway disease (collapsing trachea, chronic bronchitis, etc.). Infectious airway and pulmonary disease generally presents with an acute onset.

CHF results in a productive cough, though often expectorated fluid is swallowed and not “brought up.” This may be recognized as a gag at the end of a paroxysm of coughing. Dogs with mild pulmonary edema or cardiomegaly causing bronchial compression may not show this pattern. Any airway or pulmonary disease causing an excess of airway secretions (inflammatory and infectious diseases) tends to cause a productive cough. Airway compression/narrowing/collapse and airway irritation without an excess of secretions tends to result in a nonproductive cough.

The first onset of coughing due to cardiogenic pulmonary edema is often noted associated with resting/at night/after sleeping, and then begins to occur throughout the day as the severity progresses. Coughing due to pharyngeal or tracheal disease is often accentuated by excitement, activity, and/or eating and drinking.

Pulmonary edema interferes with normal gas exchange and oxygenation, resulting in exercise intolerance at earlier stages of heart failure progressing to resting tachypnea later. An increase in resting/sleeping respiratory rate (> 30bpm consistently) and respiratory effort is typical in dogs with a cough due to pulmonary edema. Other pulmonary parenchymal diseases, especially if complicated by pulmonary hypertension, can produce exercise intolerance and resting tachypnea. Dogs with lower airway disease generally have near normal exercise tolerance and resting respiratory rates.

Unintentional weight loss is often noted in dogs with CHF, while obesity tends to exacerbate clinical signs referable to chronic airway disease.

Physical examination
General
As noted in the previous section, general observations of lethargy or (in severe cases) distress/anxiety and weight loss or poor body/muscle condition in are noted on examination of patients with coughing due to CHF.

Respiratory patterns
CHF produces tachypnea with an increase in inspiratory and expiratory effort. This pattern of dyspnea is typical of any pulmonary parenchymal disease, including pneumonia, noncardiogenic pulmonary edema, neoplasia, pulmonary thromboembolism, parasitic lung disease, or pulmonary hemorrhage. An increase in expiratory effort (“abdominal push”), often in the presence of a normal respiratory rate, is typical of lower airway disease. Inspiratory dyspnea, often accompanied by stertor or stridor and again generally with a normal respiratory rate, occurs with upper airway disease (i.e. trachea and cranial). A restrictive breathing pattern, characterized by shallow, rapid breaths +/- paradoxical abdominal motion, is typical of pleural space disease.
**Auscultation**

Dogs with CHF are usually tachycardic (> 130 bpm) with a regular heart rhythm. Presence of a respiratory sinus arrhythmia is suggestive of dominant vagal tone and is inconsistent with CHF in most cases. Supraventricular arrhythmias and atrial fibrillation occur occasionally in dogs with advanced degenerative valvular disease. Ventricular arrhythmias and atrial fibrillation often occur in dogs with dilated cardiomyopathy (DCM).

In small breed dogs, absence of a heart murmur makes CHF very unlikely, as significant atroventricular valve regurgitation will invariably cause a heart murmur. In large and giant breed dogs, in which DCM is a more common cause of heart failure, a heart murmur is usually noted in the presence of severe disease, but may be soft. An S3 gallop sound is occasionally noted in the presence of elevated left heart filling pressure in DCM.

Abnormal lung sounds in the form of accentuated/harsh bronchovesicular sounds and/or pulmonary crackles are often noted in the present of pulmonary edema. It is important to note that pulmonary edema can be present and significant without the presence of crackles on pulmonary auscultation. Coarse crackles, especially in the absence of tachypnea, are more characteristic of lower airway disease. Stertor and stridor indicate an upper airway cause for dyspnea.

**Thoracic radiographs**

The cardiac silhouette should be enlarged in almost every case of CHF. Measurement of the vertebral heart size (VHS) can be helpful in confirming cardiomegaly, especially when equivocal, and tracking progression with time. VHS is assessed by measuring the cardiac silhouette in the long axis from the carina to the cardiac apex and in the short axis at the widest part of the cardiac silhouette perpendicular to the long axis (around the ventral border of the caudal vena cava). These measurements are then converted to a number of vertebral bodies counting caudally from T4, measured individually for each axis, and summed. Mean normal VHS is 9.5-10.5 for most dogs breeds, so a score < 11 (2 standard deviations from the mean) is normal in most dogs. In some breeds higher values can be normal (especially in boxers, English bulldogs, Boston terriers, Labrador retrievers, whippets, Cocker spaniels, ± Cavalier King Charles spaniels). Left atrial enlargement, visualized as a bulge of the dorsocaudal border of the heart caudal to the carina in the lateral projection and spreading of the bronchi/widening of the cardiac silhouette on the ventrodorsal projection, is also present in most cases. Dogs with isolated respiratory disease would be expected to have a normal heart size, though some dogs may have right atrial and ventricular enlargement secondary to pulmonary hypertension or concurrent subclinical cardiomegaly from underlying cardiac disease.

Distension of the pulmonary veins is identifiable in most dogs with CHF. Cranial vessels are best visualized on lateral projections, and the caudal vessels on ventrodorsal or dorsoventral projections. Normal pulmonary artery and vein size is less than the thickness of the 4th rib where the vessels cross on the lateral projections and of the 9th rib on ventrodorsal/dorsoventral projections. Pulmonary arteries will be normal in most cases of heart failure unless significant pulmonary hypertension is also present or there is pulmonary overcirculation due to a left-to-right shunt. Isolated pulmonary arterial dilation is noted with heartworm disease or pulmonary hypertension.

Pulmonary edema in the form of unstructured interstitial ± alveolar infiltrates is evident in most cases of CHF, the exception being dogs with incipient heart failure and nocturnal tachypnea/coughing only. The distribution of cardiogenic pulmonary edema begins in the area near the pulmonary hilus and extends in the caudodorsal lung fields before becoming generalized in most cases. Care should be taken in evaluating for perihilar infiltrates, as an enlarged left atrium will occupy the perihilar area on lateral radiographs. Other distributions of interstitial and alveolar pattern (peripheral, patchy diffuse, cranioventral) are less typical of cardiogenic pulmonary edema, and infectious, neoplastic, and etiologies of noncardiogenic pulmonary edema (choking, electric shock, seizure) should be considered.

Exposure technique, patient positioning, phase of respiration, and patient body condition can affect the appearance of heart size, relative pulmonary vessel size, and pulmonary parenchymal pattern. Good quality thoracic radiographs should be exposed with a lower mAs technique than abdominal radiographs (consult your technique chart; digital machines have a thoracic technique option), be collimated to include only the thorax, have a patient positioned square to the primary beam, and be exposed at peak inspiration.

**Cardiac biomarkers**

Serum NT-proBNP may be useful in ruling out CHF. Dogs with CHF will have significant volume overload and increased atrial wall stress, which results in an increase in BNP synthesis and secretion. The presence of a normal NT-proBNP level (< 900 pmol/ml) makes CHF unlikely in a coughing dog.

While elevated (> 1400 pmol/ml) NT-proBNP levels are usually present in dogs with CHF, a positive NT-proBNP level alone should not be interpreted as diagnostic for heart failure. NT-proBNP will elevate, and sometimes markedly, in dogs with moderate to severe degenerative valve disease or DCM prior to the onset of heart failure.

NT-proBNP cannot be used to distinguish clinical signs due to pulmonary edema from clinical signs due to pulmonary hypertension, as pulmonary hypertension will also increase wall stress and NT-proBNP production.
Works cited