Cardiac Diagnostics

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Thoracic radiographs

Thoracic radiographs are essential for any patient with clinical signs of tachypnoea or dypsnoea that is potentially referable to pulmonary pathology. Radiographs should be performed cautiously in any patient with respiratory distress to minimize additional stress. In the dog with congestive heart failure, mild pulmonary oedema is characterized by perihilar alveolar infiltrates. With more significant oedema, infiltrates are evident in the caudodorsal fields, occasionally with greater involvement of the right caudal lobe. In the cat, there is no alveolar pattern specific for cardiogenic oedema and localized or diffuse infiltrates can be identified.

Dilation of the pulmonary veins relative to their paired artery, while not commonly appreciated, is highly specific for cardiogenic pulmonary oedema.

As a general rule, left-sided congestive heart failure will only follow the development of significant generalized cardiomegaly and severe left atrial enlargement. Calculation of a vertebral heart score (VHS) can be helpful in determining whether cardiomegaly is present radiographically. To obtain the VHS from a lateral radiograph, the long axis of the heart is measured from its origin at the hilus to the apex. Using a measurement device, this length is positioned at the start of the 4th thoracic vertebra and the number of vertebral bodies spanned by the length is determined. Next, a line is drawn perpendicular to the length measurement across the widest part of the cardiac silhouette in order to measure the width of the heart. The number of vertebral bodies spanned by this length is determined and the length and width measurements are summed to determine the VHS. The normal value for VHS in most dog breeds is <10.5 and in the cat is <8.1. There are several limitations to assessment of cardiomegaly using VHS; certain breeds of dogs and overweight dogs have higher VHS in the absence of heart disease.

Of equal, if not greater importance than the identification of generalized cardiomegaly in a patient with suspected cardiogenic pulmonary oedema is the assessment of the left atrium. Except in rare instances

of chordae rupture or endocarditis, left atrial enlargement is a necessary precursor to the development of congestive heart failure, and hence, strong supportive evidence for a cardiac aetiology of pulmonary oedema or for cats, pleural effusion. Irrespective of the type of heart disease present, the compensatory response to left heart dysfunction is left atrial dilation, or more correctly, left atrial eccentric hypertrophy. Be it diastolic dysfunction with hypertrophic cardiomyopathy, volume overload with myxomatous mitral regurgitation or patent ductus arteriosus, or primarily systolic dysfunction with dilated cardiomyopathy, the left atrium "grows" in response and pressure within is normalized. Chronically, it is only once left atrial growth capacity is reached, that progressive dysfunction can no longer be compensated, left atrial pressure rises and this hypdrostatic pressure is transmitted retrograde to the pulmonary veins and pulmonary capillaries which begin to transude fluid to create pulmonary oedema (or pleural effusion in some cats).

On the lateral radiograph of dogs, the left atrium is located immediately ventral to the hilus. When the left atrium is normal, the caudal cardiac silhouette should extend caudoventrally from the point of the hilus to the cardiac apex. When the left atrium is enlarged, the cardiac silhouette immediately caudal to the hilus, initially extends horizontally or even dorsally. On the dorsoventral film, the body of the canine left atrium lies immediately caudal to the hilus between the two caudal mainstem bronchi. When the left atrium is enlarged, the body of the left atrium may displace the left mainstem bronchus cranially, giving a bow-legged cowboy appearance to the mainstem bronchi. The enlarged left auricle can be appreciated as a bulge in the cardiac silhouette at the 2-3 o'clock position. Recently, a method for objective radiographic assessment of the left atrium has been described. Vertebral left atrial score (VLAS) describes left atrial dimension when measured from the caudoventral aspect of the carina to the caudal left atrial border where it intersects with the caudal vena cava on a lateral thoracic radiograph. This dimension is then expressed as the number of vertebral bodies that traverse this measured length as counted from the cranial edge of the 4th thoracic vertebra. A VLAS of ≥ 2.3 is a useful predictor of LA enlargement and stage B2 disease severity in dogs with myxomatous mitral valve disease.

In cats, generalized cardiomegaly may be difficult to appreciate because hypertrophic cardiomyopathy (HCM) produces concentric change that may

not substantially alter the cardiac silhouette on thoracic films.

Furthermore, in cats lateral thoracic radiographs are insensitive for the detection of left atrial enlargement because the cranial location of the left atrium means it is superimposed on the cardiac silhouette rather then causing distortion of the caudal cardiac border as in dogs. Rarely, the large left atrium will be appreciated as a radio-opaque density overlying the cardiac silhouette in a right lateral radiograph but generally the dorsoventral radiograph is fair more useful for identifying left atrial enlargement in cats. In cats with left atrial enlargement sufficient to precipitate congestive failure, the cardiac silhouette takes on a valentine appearance on the dorsoventral projection as the left atrium enlarges in the 2-3 o'clock region. In cats with trivial pleural effusion, assessment of the cardiac silhouette is typically obscured in the dorsoventral projection. For these patients, acquisition of a ventrodorsal film is indicated. This allows the pleural fluid to pool either side of the vertebral canal exposing the cardiac silhouette. This technique can allow assessment for left atrial dilation with the small residual volumes present immediately post-thoracocentesis.

Thoracic radiographs, particularly when performed serially, are valuable at predicting impending risk of congestive heart failure in a patient with preclinical heart disease, irrespective of the specific cardiac disease present. For example, a dog which is diagnosed via echocardiography with mild myxomatous mitral valve degeneration may be radiographed at time of diagnosis and every 6-12 months thereafter until progressive left atrial enlargement is noted. Identification of a significant left atrial change will herald an imminent risk of congestive failure and the client can be educated to monitor for early signs necessitating therapeutic instigation.

Thoracic radiographs can also provide valuable information that may be supportive of a specific cardiac disease. For example, although poorly sensitive, identification of a bulge in the ascending aorta on the lateral film of a dog with a left basilar systolic murmur, is highly supportive of a diagnosis of subaortic stenosis.

For a clinically normal cat with a heart murmur, thoracic radiographs are likely to be particularly low yield. The high frequency of innocent murmurs in this species coupled with the insensitivity of radiographs at detecting mild-moderate HCM, the most common heart disease in cats, means that echocardiography is the superior diagnostic tool.

Echocardiography
Echocardiography is the gold standard tool for definitive diagnosis of heart disease and it is indicated for any patient with an abnormal cardiac auscultation or clinical signs or physical exam findings that are potentially referable to a cardiac aetiology (syncope, pulmonary infiltrates, pleural effusion, ascites). Echocardiography, by an experienced echocardiographer, is also necessary to accurately screen high-risk breeds for cardiomyopathy and congenital diseases. While identification of cardiac disease sufficiently advanced to produce clinical signs may be straightforward, achieving an accurate definitive diagnosis of specific cardiac disease and identifying the subtleties of mild disease, requires advanced training and extensive echocardiographic experience, requiring referral to a specialist cardiologist.

Electrocardiography (ECG)
Electrocardiography is indicated when cardiac auscultation identifies an irregular rhythm, tachycardia or bradycardia; when a patient is diagnosed or suspected to have a cardiac disease known to be associated with arrhythmias such as dilated cardiomyopathy; and in any patient with suspected syncope. ECG identification of a tachycardia as sinus, supraventricular or ventricular is necessary in order to ascertain risks posed the arrhythmia and to guide appropriate medical suppression. Similarly, an ECG is necessary to determine the nature of a bradycardia, the risks associated with it, and appropriate therapeutic approach. Unless it is recorded at the time of the syncopal event, the resting ECG is incapable of definitively identifying a causative arrhythmia. However, a resting ECG may provide supportive evidence for arrhythmia-associated syncope in some dogs and cats. For example, the identification of pre-excitation (short PR interval and delta waves) on sinus complexes is indicative of a congenital accessory pathway and is known to facilitate the development of supraventricular tachycardia-associated syncope in affected dogs and cats. Similarly, the identification of ventricular premature complexes on the ECG of a Doberman is supportive of a diagnosis of dilated cardiomyopathy and the risk of ventricular tachycardia-associated syncope/ sudden death.

CBC/ Biochemistry
Complete blood count, biochemistry and urinalysis are not generally indicated in clinically well animals with abnormal cardiac auscultation. For patients presenting initially with congestive failure, baseline CBC/

biochemistry are typically performed in order to rule out significant concurrent disease and to identify any pre-existing azotaemia which may preclude the use of angiotensin converting enzyme inhibitors and which may become clinically significant in the face of diuretic therapy.

Biomarkers

In the recent past, the role of serum biomarkers in the identification of subclinical heart disease and their ability to aid diagnostic distinction between cardiogenic and non-cardiogenic causes of respiratory signs has been investigated. These biochemical markers of cardiac disease can be broadly categorized into two groups: 1. Leakage markers indicating myocardial damage eg. Cardiac troponin I (CTnI) and cardiac troponin T (CTnT) and 2. Neuroendocrine markers indicative of cardiac functional impairment eg. Atrial (ANP) and brain natruretic peptides (BNP).

The longer half-life of BNP relative to ANP has made BNP a superior serum marker for clinical purposes. Several studies have reported higher NT-proBNP in dogs and cats with heart disease compared to healthy controls and most recently it's ability to distinguish non-cardiac and cardiac causes of respiratory signs has been assessed. Typically, these studies report a sensitivity and specificity of NT-proBNP in the range of 80-85% thereby precluding the test's ability to definitively identify aetiology of respiratory signs in an individual, but providing a potentially useful adjunct to guide further diagnostic tests in general practice.

Of the cardiac troponins (CTn), CTnI has been the focus of most investigations because it is more readily released into the circulation with cardiomyocyte injury, hence is a more sensitive serum marker than CTnT. While many studies have documented elevated CTnI with various forms of heart disease in dogs and cats, it has no worth in distinguishing cardiac versus non-cardiac causes of respiratory signs.

Advanced Diagnostic Tests

Ambulatory 24hr Holter ECG, event monitoring and diagnostic cardiac catherterization are indicated for some cardiac patients. These diagnostic tools require advanced training to achieve proficiency and when indicated for a patient, referral to a specialist cardiologist is indicated.

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