Catalog

Feline Cardiomyopathy: Diagnosis and Management
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Myocardial disease is an important cause of morbidity and mortality in domestic cats. Feline myocardial disease sometimes occurs in association with non-cardiac disease but when that is the case, it often is subclinical; idiopathic myocardial disease is more commonly responsible for clinical signs. Cardiomyopathy (CM) has been defined as a myocardial disorder in which the heart muscle is structurally and functionally abnormal in the absence of other cardiovascular diseases sufficient to cause the observed myocardial abnormality.
Recently proposed schema for classification of human cardiomyopathies have emphasized the cause or molecular basis of myocardial disease. Although genetic etiologic factors are likely important, feline CM remains largely idiopathic. Accordingly, the use of morphopathologic/ functional designations remains valid. Specifically, hypertrophic cardiomyopathy (HCM) is defined by diffuse or regional hypertrophy of a nondilated ventricle in the absence of hemodynamic causes of hypertrophy. Dilated cardiomyopathy (DCM) is characterized by left or biventricular dilation associated with diminished systolic myocardial function. Restrictive cardiomyopathy (RCM) is functionally defined by diminished ventricular compliance; the ventricle may have a normal or nearly normal appearance but left or biatrial dilation are consistent features. Arrhythmogenic right ventricular cardiomyopathy (ARVC) is characterized by fatty or fibrofatty replacement of right and sometimes left ventricular myocardium and often, arrhythmias. During this session, the pathophysiology, diagnosis and therapy of hypertrophic cardiomyopathy (HCM), the most common feline myocardial disease, will be emphasized.

ETIOPATHOGENSIS

It is accepted that that HCM in humans is primarily a genetic disease that is associated with numerous mutations of genes that encode sarcomeric proteins. HCM is inherited in Maine coon cats and Ragdoll cats, and in cats of these breeds is associated with distinct mutations of the myosin binding protein C (MYBPC) gene. Familial occurrence of HCM has been observed in other purebred lines and in mixbreed cats; it is therefore possible that feline HCM generally is a genetic disorder. The causes of the other forms of primary myocardial disease that are observed in the cat have not been established. It is possible that some examples of RCM represent the sequela of endomyocardial inflammation or perhaps, an alternate phenotypic expression of mutations that are associated with HCM. The prevalence of feline DCM decreased radically after the recognition of the association between this disorder and nutritional taurine deficiency, but idiopathic DCM is still sporadically observed.

EPIDEMIOLOGY

Several investigators have retrospectively evaluated the population characteristics of feline HCM. HCM is not exclusively a geriatric disease; patients of all ages can be affected and the median age at the time of detection in one report was 4.6 years. Males are more often affected than are females. A substantive proportion - between 33 and 55% - of cats with HCM are subclinical (asymptomatic) when the disease is identified. The prevalence of HCM in apparently healthy cats is close to 15%. This prevalence is seemingly high but consistent with the current understanding of the HCM in humans; it is now accepted that HCM has a broad

spectrum of phenotypic expression, often occurs in a subclinical form and is not inevitably associated with progression and poor outcome.

PATHOPHYSIOLOGY

Diastolic dysfunction is thought to be the primary pathophysiologic mechanism responsible for clinical signs in HCM. Diastolic function refers to the ability of the ventricle to fill at low pressures. The primary determinants of diastolic function are the active process of myocardial relaxation and a mechanical property of the ventricle known as compliance. Diastolic dysfunction results in increased ventricular filling pressures when ventricular volumes are normal or small. High filling pressures are reflected “upstream” potentially resulting in atrial dilation and the development of pulmonary edema or pleural effusion. In feline CM, atrial dilation almost invariably precedes the development of congestive signs. Functional abnormalities in HCM are not limited to diastole. Although the clinical implications have been debated, most patients with HCM exhibit a valve motion abnormality – systolic anterior motion of the mitral valve or, SAM – that causes obstruction of left ventricular outflow. In affected cats, hydrodynamic forces, of which drag is most important, cause systolic movement of the mitral leaflets toward the interventricular septum. This abnormal valvular orientation causes dynamic, as opposed to fixed, obstruction of the left ventricular outflow and typically, concurrent mitral valve regurgitation. In addition, some patients, presumably those with long-standing HCM, develop systolic myocardial dysfunction resulting in a cardiac phenotype that has been referred to as “end-stage HCM” or “burnt-out HCM”.

CLINICAL PRESENTATION / DIAGNOSIS

Feline CM is identified when abnormalities are detected during physical examination of apparently healthy cats, when congestive heart failure (CHF) develops, or when CM is complicated by systemic thromboembolism. Many, but not all, cats with HCM have cardiac murmurs, but it is relevant that murmurs can develop in cats in which cardiac disease is absent. Furthermore, murmurs in cats, whether related to cardiac disease or not, are often labile, meaning that the intensity can change from moment to moment. Murmurs in cats can be provoked by increases in sympathetic activation, and an increase in murmur intensity documented during serial examinations does not indicate worsening of disease.

Retrospectively evaluated case series have identified an association between the administration of corticosteroids and the development of CHF in cats. Some affected cats may have had pre-existing, but clinically silent HCM, but this has not been established. The association is relevant because the long-term prognosis of corticosteroid associated CHF might be superior than for more typical presentations.

Patients with congestive heart failure typically are presented for evaluation of respiratory distress caused by pulmonary edema or pleural effusion. Cats with CHF rarely cough. Hypothermia is frequently recorded. Tachypnea is generally evident and pulmonary auscultation may disclose adventitious lungs sounds in patients with edema or attenuated sounds in those with pleural effusions. While tachycardia caused by sympathetic activation is commonly observed in canine patients with heart failure, heart rates of cats with CHF do not differ from those of healthy cats and sometimes, feline patients with heart failure are bradycardic. Cardiac auscultation may reveal murmurs, gallop sounds and sometimes tachycarrhythmia, but these findings are not consistently present.

In the cat, radiographic patterns of specific chamber enlargement are not distinct. However, the chest film may reveal consequences of cardiac dysfunction; pleural effusion can be identified

and the finding of pulmonary opacities together with cardiomegaly provides a non-invasive diagnosis of heart failure. Echocardiography is the only non-invasive method that can definitively characterize feline CM. Congestive heart failure is a clinical and/or radiographic diagnosis; the presence of heart failure cannot be determined based on echocardiographic data alone. However, focused, point of care thoracic ultrasound can play a vital role in the diagnostic approach to feline respiratory distress; the findings of left atrial enlargement, particularly when a small pericardial effusion is present, are strongly predictive of a diagnosis of congestive heart failure.

Blood concentrations of cardiac biomarkers including endothelin, atrial natriuretic peptide (ANP), B-type natriuretic peptide (BNP) and troponin have been evaluated in veterinary patients. Circulating BNP concentration has a particular role in the diagnostic evaluation of patients suspected to have heart failure. This hormone is released by atrial and ventricular cardiomyocytes in response to increases in ventricular filling pressures; potentially, it is a blood-borne diagnostic marker of the heart failure state. The diagnostic accuracy of the quantitative NT-BNP assay, for identification of moderate or severe subclinical CM or identification of cardiac causes of respiratory distress is relatively high. However, the need to submit samples to a central laboratory is a disadvantage in the urgent/emergent setting. The point of care assay provides a binary – normal/abnormal – result and has a role when feline patients are presented for evaluation of respiratory distress, it is not possible to safely obtain diagnostic chest films and point of care echocardiographic examination is not available. Optimally, abnormal NT-BNP results are investigated echocardiographically.

THERAPY

In order to facilitate the development of therapeutic guidelines, a modification of the AHA/ ACVIM scheme for staging of heart disease can be applied to feline CM.

The role of drug therapy in patients with subclinical (asymptomatic) Stage B CM is uncertain. It is likely that most patients with HCM have slowly progressive or even non-progressive, disease. Furthermore, there is currently no published evidence that any agent can slow the progression of HCM. Based on this, periodic echocardiographic re-evaluation rather than therapy is appropriate when faced with patients that have Stage B1 disease; meaning, subclinical HCM with left atrial dimensions that are normal, or reflect only mild enlargement. In patients with stage B2 disease – those that have distinct left atrial enlargement – administration of clopidogrel is reasonable, in hopes of decreasing the probability of arterial thromboembolism [ATE]. It should be noted however, that evidence to support this approach is indirect and the incidence of ATE in patients with subclinical echocardiographically documented CM is not very high.

Heart failure is a clinical syndrome characterized by high venous pressures and/or low cardiac output that results from cardiac disease. CHF results in clinical signs related to tissue edema or body cavity effusions. Pulmonary edema is a consequence of left ventricular failure. In cats, pleural effusions may result from left ventricular or biventricular heart disease. For patients that have developed congestive signs, that is, those with Stage C CM, general, supportive measures are indicated. Supplemental oxygen can be administered through use of an oxygen administration cage or, if the patient is sufficiently tolerant, by mask, or nasal insufflation. Pleurocentesis should be performed when physical, radiographic or sonographic findings confirm that pleural effusion is responsible for respiratory distress.

In general, intravenous fluids should not be administered to patients with frank congestion. In the setting of CHF, infusion of fluid further increases venous pressures but does not improve

cardiac performance. When cardiogenic pulmonary edema is present, diuretic administration is indicated. For acute decompensation, the intravenous route is preferred but intramuscular administration is appropriate when resistance to manual restraint or other factors make intravenous administration difficult or impossible. Preload reduction is used in the setting of heart failure because it may effectively eliminate clinical signs related to congestion. In general however, preload reduction does not improve cardiac performance. Indeed, aggressive reduction in filling pressures can decrease stroke volume potentially resulting in hypotension. Other than furosemide, for which efficacy is assumed, there are no medical interventions that have demonstrated efficacy in the management of feline heart failure. Because of this, the use of additional cardioactive agents in the management of acute decompensated heart failure is difficult to justify. Exceptions to this might be the use of antiarrhythmic agents for management of pathologic arrhythmias that contribute to the development of congestive signs, or perhaps the administration of pimbendan to patients with low output heart failure.

Evidence that supports administration of ancillary, medical therapy to patients with stage C is lacking. The results of a multicenter, randomized, placebo-controlled trial that had been designed to evaluate the relative efficacy of atenolol, diltiazem and enalapril in feline patients with diastolic heart failure have been reported but not published. The primary end-point of the trial was recurrence of congestive signs and none of the agents was superior to placebo in this regard, although atenolol was inferior. The results of a prospective, double-blind placebo controlled trial have recently been reported. For patients enrolled in this exploratory trial, pimobendan administration did not lead to improved 180-day outcome.

Arterial thromboembolism is a serious and often unexpected complication of feline CM. It is unexpected because ATE is the first clinical manifestation of CM in the majority of patients in which it occurs. Emergent care of patients with ATE most importantly is supportive and includes analgesia. ATE is almost always associated with advanced CM that has resulted in left atrial enlargement and diagnostic evaluation to identify evidence of edema/effusion is important. Parenteral administration of anticoagulant therapy – unfractionated or low molecular weight heparin is reasonable in the short-term. The results of a clinical trial that compared aspirin to clopidogrel for secondary prevention of ATE provided strong evidence of the superiority of clopidogrel.

PROGNOSIS

The results of a recent retrospective analysis of risk and mortality in HCM provide clinically relevant information regarding prognosis associated with subclinical [Stage B] disease. In general, the condition is benign but has important long-term clinical consequences. Over the course of follow-up, total cardiovascular mortality was 28%. Survival after the development of congestive heart failure or the occurrence of ATE was relatively brief with a median survival of less than one year.

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Diagnosis and Management of Canine Pulmonary Hypertension
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Pulmonary hypertension (PH), defined by abnormally great pressures in the pulmonary vasculature, is a hemodynamic state that can develop in association with a variety of cardiovascular, respiratory and systemic diseases. Clinical signs of PH include cough, tachypnea and exertional syncope. These signs are not diagnostically specific and because of this, the identification of patients for which PH is an appropriate therapeutic target can be difficult. During this presentation, the pathophysiology and therapy of PH will be addressed; clinical findings which help to distinguish patients with primary left heart disease from those with pulmonary arterial hypertension will be outlined.

PATHOGENESIS

In health, pulmonary vascular resistance (PVR) – the hydraulic forces that must be overcome for a pressure difference to result in flow – is lower than is systemic vascular resistance, and as a result, pulmonary arterial pressures are lower than systemic arterial pressures. Pulmonary hypertension (PH) refers to abnormally great pressures in the pulmonary vascular system. In people, PH is defined by a mean pulmonary artery pressure that exceeds 25 mmHg. Mean pulmonary artery pressure can be only be obtained invasively, by right heart catheterization (RHC). Diagnostic RHC is rarely performed in veterinary patients, and the diagnosis is generally based on echocardiographic findings.

Pulmonary artery pressure (PAP) is related to pulmonary blood flow (Q) and PVR according to Ohm's Law: $PAP_{mean} - LAP_{mean} = Q \times PVR$, where: LAP = left atrial pressure, Q = cardiac output [flow] and PVR = pulmonary vascular resistance.

None of these quantities is routinely measured, but the concept of PVR is important because it relates to therapeutic approach. PH can result from a rise in PVR, an increase in pulmonary blood flow, an increase in LAP or combinations of these factors.

PATHOPHYSIOLOGY

When PH results from high PVR, it is described as "pre-capillary PH"; vasoconstriction and/or vascular remodeling are the principal causes of high PVR, although pulmonary thrombosis can contribute. Left atrial hypertension resulting from left heart disease is the primary cause of "post-capillary PH". The right ventricle must generate a pressure that is adequate to propel the stroke volume not just to the lungs, but to the left atrium; as a result, a pathologic increase in left atrial pressure necessitates an increase in right ventricular and pulmonary arterial systolic pressure. In some patients with left heart disease, the increase in right ventricular – and therefore pulmonary arterial – pressure, initiates a cascade of vasoconstriction and vascular remodeling, increasing PVR and resulting in PAP that is disproportionately high relative to left atrial pressure. PH imposes a pressure load on the right ventricle and potentially results in right ventricular hypertrophy, functional pulmonary and tricuspid valve regurgitation, myocardial dysfunction and right-sided congestive heart failure.

CLINICAL PRESENTATION

Clinical signs associated with PH include cough, tachypnea, exercise intolerance, syncope, and abdominal distention due to ascites. It is relevant that these clinical signs are associated with PH, but the association is not necessarily causal. For example, cough is unlikely to result directly from PH, but is more apt to reflect an underlying disease that has caused PH.

Physical findings may include adventitious lung sounds and a right apical systolic murmur resulting from tricuspid valve regurgitation. Pulmonary valve regurgitation is commonly detected echocardiographically but is rarely audible. When the clinical presentation includes respiratory distress and a right apical murmur, PH [and pulmonary thromboembolism] should be diagnostic considerations because the therapeutic approach to these disorders is different from that which is appropriate for left-sided congestive failure.

DIAGNOSTIC EVALUATION

The definitive diagnosis of PH is through direct measurement of pulmonary artery pressures, but in veterinary patients the diagnosis is most often based on echocardiographic findings.

Echocardiography

Echocardiographic evidence of tricuspid valve regurgitation (TR) is commonly observed in patients with PH. The velocity of the TR jet, obtained by continuous-wave Doppler, is related to the systolic pressure difference between the right atrium and the right ventricle by the simplified Bernoulli equation ($\Delta P = 4v^2$ where ΔP is the pressure difference and v is the velocity of the regurgitant jet measured by Doppler echocardiography).

In the absence of pulmonary stenosis (PS), right ventricular and pulmonary artery pressures are *equal* during systole. Thus, measurement of the velocity of the TR jet provide a noninvasive *estimate* of systolic pulmonary artery pressure. The diagnostic accuracy of other echocardiographic variables including various systolic time intervals, tricuspid annuluar plane systolic excursion (TAPSE) as well the fractional change in dimensions of the right pulmonary artery have been evaluated. In general, these surrogate measures can provide supportive evidence and are considered in addition to the velocity of TR or when TR is absent. It is axiomatic that they are less accurate than the criterion [“gold”] standard to which they have been compared. Partly because of the inaccuracy of Doppler echocardiographic estimation of PAP, the ACVIM consensus panel recommended a *probabilistic approach* to the diagnosis of PH. That is, the velocity of TR is considered in the context of other echocardiographic variables, and a low, intermediate or high diagnostic probability of PH is assigned based on these findings. Echocardiographic evaluation of left atrial size is generally used to determine if pulmonary hypertension is the result of high pulmonary vascular resistance [“pre-capillary”] or is the consequence of increases in left atrial pressure resulting from left heart disease [“post-capillary”]. In patients for which there is an intermediate or high probability of PH, normal or diminished left atrial dimensions provide indirect evidence that PH is pre-capillary; left atrial

enlargement provides evidence that the PH is at least partly post-capillary.

THERAPY OF PULMONARY HYPERTENSION

Treatment of causative or underlying disorders such as heartworm disease or specific pulmonary diseases is essential. Various vasodilators have been used in attempts to decrease pulmonary vascular resistance, but sildenafil is the agent that used most often in canine patients. Sildenafil is an inhibitor of phosphodiesterase type 5 and is a relatively selective dilator of pulmonary arterioles; it generally is indicated when clinical signs result from pre-capillary pulmonary hypertension

There are numerous causes of PH, but after Dirofilariasis is excluded, the distinction between pre- and post-capillary PH is the most important therapeutically relevant goal. In general, the initial therapeutic approach to patients with post-capillary PH is directed toward optimization of treatment for left-heart disease. Agents such as sildenafil that result in relatively selective dilation of pulmonary arterioles are intended to reduce PVR. The resultant increase in pulmonary blood flow can potentially raise left ventricular filling pressures and precipitate the development of cardiogenic pulmonary edema. As a result, agents such as sildenafil should be used with caution if at all, in patients with left heart disease, and generally only after resolution of pulmonary edema.

In contrast, when clinical signs are the result of high pulmonary vascular resistance and decreased systemic output, as is often the case in pre-capillary PH, the use of specific therapies such as sildenafil is appropriate.

Idiopathic Pulmonary Arterial Hypertension

For mostly valid reasons, aggressive diagnostic evaluation including advanced diagnostic imaging and lung biopsy are seldom performed in patients with respiratory distress and PH. Arguably then, pre-capillary PH, or pulmonary arterial hypertension (PAH), often is idiopathic only because diagnostic evaluation is incomplete. Regardless, there is an important syndrome of PAH in patients that do not have Dirofilariasis or a history of chronic respiratory disease, which is typically observed in older, small- breed dogs. Brachycephalic dogs might be over-represented. Clinical signs include cough, tachypnea/respiratory distress, and syncope, the latter typically observed on exertion. The precise cause is unknown but the pathogenesis relates to vasoconstriction / vascular remodeling that causes an increase in PVR, and the development of PAH. As implied, the diagnosis is presumptive and based on exclusion of known causes of PH, such as heartworm disease. The syndrome is echocardiographically characterized by a normal or small left atrium / left ventricle - caused by diminished pulmonary venous return - and evidence of PH.

The prognosis is generally poor, although some patients respond favorably to administration of sildenafil and supplementary oxygen. The distinction between PAH and acutely decompensated left-sided heart failure can be challenging, but important because diuretic treatment is potentially harmful in the setting of idiopathic PAH. In these patients, the clinical signs are not the result of high pulmonary venous pressures. Conversely, as stated, agents such as sildenafil can harm in the setting of severe left sided heart disease. If echocardiography is not available, it is important to consider the

possibility that idiopathic PAH is responsible for clinical signs such as tachypnea/ respiratory distress/exertional syncope, particularly when:

- distinct radiographic left atrial enlargement is absent
- there is a lack of response to diuretic therapy
- a cardiac murmur that is loudest over the *right* apex is identified

Additional Readings / Complete References Available On Request

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Management of Canine Mitral Valve Disease
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Degenerative valvular disease is the most common cardiac disease in dogs. The incidence is age-related and in post-mortem surveys, the prevalence of valvular degeneration exceeds 90% in dogs older than 9 years of age. However, the prevalence of clinically evident valvular disease is much lower and only a minority of affected patients exhibit clinical signs. Degenerative disease most commonly affects the atrioventricular valves and clinical manifestations usually result from mitral disease. Although a syndrome of valvular regurgitation and concurrent myocardial dysfunction is occasionally observed in large-breed dogs, the clinical syndrome of severe, clinically apparent mitral valve regurgitation (MR) is primarily observed in aged, small breed dogs. The dachshund, poodle, Pomeranian and cavalier King Charles spaniel are predisposed. Histologically, the disorder is characterized by accumulation of mucopolysaccharide within the spongiosa layer of the atrioventricular valves. This review will focus on the medical management of canine degenerative mitral valve disease (MVD).

PATHOPHYSIOLOGY OF MITRAL VALVE REGURGITATION

When the mitral valve is incompetent, part of the left ventricular stroke volume is ejected retrograde into the left atrium increasing the volume and intracavitary pressure of this chamber. The regurgitant volume augments the pulmonary venous return and then enters the ventricle during diastole. Therefore, MR imposes a volume load on the left atrium and the left ventricle; dilation and hypertrophy of the atrium and ventricle follow as a consequence. Potentially, ventricular filling pressures rise, resulting in pulmonary venous hypertension and the development of pulmonary edema. Clinical signs of tachypnea / respiratory distress and cough potentially result from the presence of pulmonary edema. In some small-breed dogs with MR, cough develops or persists in the absence of pulmonary edema. In these cases, compression of the bronchi by an enlarged atrium and concurrent primary respiratory disease are possible causes.

DIAGNOSTIC EVALUATION

Cough is commonly observed in patients with MR, but it is important to recognize that MVD is a common disease that exhibits a broad spectrum of severity. It is therefore inevitable that some patients with a murmur of MR develop a cough that is related not to heart disease but rather, is the consequence of concurrent, primary respiratory tract disease. Although primary respiratory tract disease and cardiac disease are often concurrent, one of the two typically dominates the clinical presentation. In most cases, the patient history, physical examination and thoracic radiographic examination provide the information needed to make this important clinical distinction.

In MVD, the most notable feature of the physical examination is a systolic murmur that is usually heard best over the left cardiac apex. An acquired, left apical, systolic murmur in an older, small-breed dog is almost always caused by MVD; other causes are rare. The intensity of the murmur depends on a number of factors, but severe MR usually causes a loud murmur. Perhaps more importantly, a soft murmur that results from MVD is rarely of clinically consequence. Furthermore, in small breed, elderly dogs, a cough that occurs in the absence of

a cardiac murmur is almost always due to primary respiratory tract disease, not cardiac disease.
Radiography is the single most informative diagnostic test in most cases of MVD. However, thoracic radiographs must be obtained and interpreted with careful attention to patient conformation, phase of respiration during the exposure and radiographic technique. While the boundaries of individual cardiac chambers cannot be reliably identified by plain radiography, the chest X-ray usually provides a clinically adequate estimate of left atrial size. This is fortunate because an assessment of left atrial size is necessary to ascertain the clinical consequence of cardiac disease. With very few exceptions, a diagnosis of heart disease or failure as a cause of cough is untenable in the absence of radiographic left atrial enlargement. MVD is a chronic disease and therefore, enlargement of the left atrium and ventricle precede the development of pulmonary edema. Obviously, the left atrium must be enlarged before it can be implicated as a cause of bronchial compression. In the absence of left atrial enlargement, chronic cough is related to primary respiratory tract disease.
Echocardiography can provide useful ancillary diagnostic information and is part of the optimal management of patients with MVD. This is particularly so in the assessment of subclinical cardiac disease, because there are echocardiographic criteria that identify asymptomatic patients that might benefit from therapy. Echocardiography is especially useful when radiographic evidence of left atrial enlargement is equivocal, when it is important to evaluate systolic myocardial function, and when the presence of pulmonary hypertension is suspected.

THERAPY
Surgical mitral valve repair is currently the preferred therapeutic approach when people develop severe MR as a consequence of mitral prolapse. Unfortunately, the need for special equipment and expertise as well as cost limits the widespread application of surgical treatment in canine MVD. Because of this, therapy of valvular disease in veterinary medicine has generally emphasized medical management.

In 2009, a working group of the American College of Veterinary Internal Medicine (ACVIM), Specialty of Cardiology, developed Guidelines for the Diagnosis and Treatment of Canine Chronic Valvular Heart Disease. These guidelines were revised and updated in 2019 and published as an open access document. A schema for classification of patients, modified from one proposed for the description of humans with HF, was presented.

According to the current ACVIM Guidelines, patients with MVD are classified as follows:

Stage A: patients predisposed to the development of MVD/HF

Stage B: patients with subclinical (“asymptomatic”) MVD

- B1—without cardiac enlargement
- B2—with chamber enlargement that meets criteria that identify patients for which there is evidence that medical treatment delays the onset of clinical signs.

Stage C: patients with MVD that have current *or prior* clinical signs

Stage D: patients with medically refractory HF

Therapeutic strategies for each of these stages are summarized below. Guidelines are intended to provide generally applicable principles, but all clinical data, including the results of

clinicopathologic monitoring, must be considered in the development of management plans for individual patients:

Stage A
Diagnostic screening of predisposed breeds and genetic counseling are the only management strategies relevant to this stage of the disease.

Stage B1
Medical therapy is not indicated for patients in Stage B1

Stage B2
The inodilator, pimobendan, delays the onset of clinical signs in asymptomatic canine patients *that have cardiac chamber enlargement* resulting from MVD. The evidence to support this contention is the result of a randomized, placebo-controlled and blinded clinical trial; relative to placebo, administration of pimobendan delayed the onset of pulmonary edema. The inclusion criteria for that trial identify patients that are likely to benefit from pimobendan; they are:

- grade 3/6 (or louder) murmur caused by MVD
- VHS > 10.5
- echocardiographic evidence of left atrial and left ventricular enlargement
    - LA:Ao >1.6
    - nLVDD >1.7
        - nLVDD = LVIDD/[body weight (kg)]$^{0.294}$

Pimobendan is administered at a daily dose of 0.5 mg/kg PO, divided into two not necessarily equal doses. In some patients with stage B2 MVD, there might be a role for ACE inhibition.

Stage C
Patients with stage C have, or have had, clinical signs. In practical terms, Stage C generally is defined by the development of cardiogenic pulmonary edema. In patients with stage C MVD, recommended therapy consists of:

- Diuretic therapy – generally, furosemide +/- spironolactone
- ACE inhibition – most often, enalapril or benazepril
- Pimobendan

Stage D
Patients with stage D have medically refractory heart failure which, based on the ACVIM Guidelines, is identified when a furosemide dose > 8 mg/kg is required to prevent congestive signs in patients receiving standard therapy for stage C. Therapeutic strategies used in this stage of disease include intensification of diuretic therapy – “triple diuretic therapy” or substitution of furosemide with torsemide – higher, off-label doses of pimobendan and in some cases, additional vasodilators such as amlodipine. The evidence to support these interventions generally is weak.

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Diagnostic Approach to Syncope
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Syncope is an important clinical problem; it causes pet owners considerable anxiety, it can be a manifestation of serious disorders and sometimes, it is a harbinger of sudden death. An etiologic diagnosis of syncope can be elusive and the evaluation of patients that experience syncope presents a considerable challenge.

PATHOPHYSIOLOGY

Syncope, or fainting, is a transient loss of consciousness [t-loc] that results from a decrease in cerebral perfusion. The decrease in cerebral perfusion is requisite and distinguishes syncope from other causes of t-loc that include seizures and cataplexy. Syncope results when areas of the central nervous system responsible for maintaining arousal are deprived of substrates required for oxidative metabolism.

Syncope is associated with transient reductions in systemic perfusion pressure that occur when there is an abrupt decline in cardiac performance and/or when autonomic dysfunction results in excessive vasodilation. The former can be caused by cardiac arrhythmias but structural cardiac disease could hypothetically limit cardiac output during exercise and result in syncope.

Broad etiologic categories include reflex-mediated syncope, cardiac syncope and orthostatic hypotension. The latter - hypotension that occurs shortly after standing - is an important cause of syncope in people but does not have an obvious, or easily identified, analogue in veterinary patients. While the precise reflex arc is uncertain, reflex-mediated syncope results from bradycardia and/or vasodilation associated with autonomic reflexes that result in vagal discharge and sympathetic withdrawal. These events are often precipitated by specific stimuli, vasovagal or vasodepressor syncope being examples. The term, neurocardiogenic syncope, implies a certainty with regard to pathogenesis that is currently lacking so the more general descriptors, reflex-meditated syncope or simply reflect syncope are preferred. Cardiac syncope includes events resulting from arrhythmias. Both bradyarrhythmias and tachyarrhythmias can be responsible for the decrease in cerebral perfusion that results in syncope.

DIAGNOSIS

Syncope must first be differentiated form other events associated with reduced mentation. It can be difficult to distinguish syncope from seizures. Often, syncope results in flaccid paralysis; however, some patients become tetanic or even briefly exhibit clonic movements - signs that are usually associated with seizures. A carefully elicited history is essential; the patient's behavior before and after the event are of particular importance. Events that are precipitated by exercise are often syncopal. A distinct post-ictal phase typically follows a seizure while recovery from syncope is more often rapid and complete.

The presence or absence of structural cardiac disease provides a clinically useful means of categorizing the causes of syncope. This basic determination provides diagnostic direction, and potentially, prognostic information. A careful physical examination should precede more extensive evaluation. Particular attention is paid to auscultation, examination of the mucous membranes and assessment of the venous and arterial pulse. Information gained from the

physical examination is used to refine the differential diagnosis and therefore direct the diagnostic approach. For example, the detection of a cardiac murmur should prompt appropriate imaging studies. When syncopal episodes are associated with clinically important, structural cardiac disease, therapy of the underlying disorder may be more rewarding than attempts to document the cardiac rhythm at the time of collapse. The physical examination and resting electrocardiogram are vital elements of the evaluation of patients that experience syncope. Further studies including a serum chemistry profile, complete blood count, thoracic radiographs and echocardiography are performed as directed by the historical and physical findings.

ELECTROCARDIOGRAPHY

The resting electrocardiogram

Resting electrocardiography is an appropriate part of initial evaluation of patients that experience syncope. Occasionally, the diagnosis is readily apparent. High-grade second degree AV block or complete heart block is typically a persistent finding and is detected during initial examination. In contrast, sinus node dysfunction may be sporadically evident. This condition - sick sinus syndrome - is acquired and presumably, develops slowly. During the early stages some degree of autonomic responsiveness is likely retained. High sympathetic tone during hospital visits can increase the sinus rate and mask abnormalities of sinus node function. If the cause of syncope is unclear despite evaluation of the resting electrocardiogram provocative tests including an atropine response test, vagal maneuvers and post-exercise electrocardiography can be considered.

Atropine can be administered in order to indirectly assess the function of the sinus node and the AV node. The intrinsic rate of discharge and the refractory period of these tissues are dependent, in part, on autonomic tone. Atropine blocks muscarinic receptors and therefore reveals the level of ambient sympathetic activity. Most normal dogs develop a regular sinus rhythm with a rate that exceeds 150 beats per minute after administration of atropine. Much of the moment-to-moment variation in heart rate observed in healthy dogs is the result of changes in parasympathetic discharge - thus, after atropine administration, the rhythm becomes regular when sinus node function is normal. The response of patients with AV block is variable. If elevated parasympathetic tone has resulted in prolongation of the relative refractory period of an otherwise normal AV node there is an increase in the sinus rate and normalization of AV conduction. That is, the heart rate increases and all atrial impulses are conducted to the ventricles. In cases in which an intrinsic disease process results in an increase in the absolute refractory period, the block persists. If the administration of atropine causes an increase in the rate of sinus node discharge, the A:V conduction ratio may increase causing an apparent worsening of the conduction disturbance. A normal response to atropine suggests that the intrinsic function of the specialized conduction system is normal. It does not necessarily imply that extrinsic factors such as inappropriate parasympathetic discharge are not responsible for a bradycardia that precipitates syncope.

Vagal maneuvers are most often used therapeutically or diagnostically as a means to terminate or slow supraventricular tachycardias. However, a vagal maneuver might provoke sinus pauses or periods of AV block in patients with syncope and provide clues as to the rhythm during the episodes.

Ambulatory Electrocardiography

Event Monitoring

Event monitoring refers to patient (or pet-owner) initiated electrocardiographic recordings that provide precise correlation of signs or symptoms with cardiac electrical activity. A microprocessor performs continuous acquisition of electrocardiographic data. The memory is finite and limited to about five minutes; thus the record of data is in the form of a loop. As new data is acquired the oldest data is deleted. If an event of weakness or syncope is witnessed, the pet owner presses a button, halts the process, and a record of eletrocardiographic activity during the event is retained. Implantable loop recorders intended for placement within a surgically created subcutaneous pocket are also available and can usefully document electrocardiographic events that are associated with infrequent clinical episodes.

Holter Monitoring

Continous recording of 24 or more hours of electrocardiographic data is known as Holter monitoring after the engineer that first developed the device. The electrocardiographic data is obtained and stored in analogue form on magnetic tape or, in a digital format on silicon chips. Modern recording devices are relatively small and can be attached by means of adhesive bandages to veterinary patients. Adhesive patches are placed over the chest and provide electrode contact. The collected data are, if necessary, converted to digital form and then subject to computer analysis. If a commercial Holter analysis service that is accustomed to human data is utilized it is essential that the data are “over-read” by a veterinarian familiar with recordings obtained from normal animals. Variability of heart rate that results from physiologic sinus arrhythmia can be extreme in normal dogs, long sinus pauses are often observed and heart rates of less than 40 b/m are often evident when healthy dogs are sleeping. Correlation of the patient’s activity with the electrocardiographic data is necessary for the Holter recording to provide diagnostic information. Syncopal episodes must occur relatively often if twenty-four hours of electrocardiographic data is to provide a definitive diagnosis. However, abnormalities such as inappropriate sinus pauses, inappropriate bradycardia and the occurrence of tachyarrhythmia might allow a presumptive etiologic diagnosis of syncope.

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Arrhythmogenic (Boxer Dog) Cardiomyopathy
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Although congestive heart failure is occasionally observed, heart muscle disease in North American boxer dogs is principally characterized by ventricular tachyarrhythmia (VTA). Often, arrhythmias are incidentally detected during routine examination but the disorder may become clinically apparent when episodic weakness, syncope or sudden unexpected death occurs. The cause, clinical presentation as well as the diagnostic and therapeutic approaches to this common disorder will be reviewed.

ETIOPATHOGENESIS

Cardiomyopathy has been defined as a myocardial disorder in which the heart muscle is structurally and functionally abnormal in the absence of other cardiovascular diseases sufficient to cause the observed myocardial abnormality. Boxer dog cardiomyopathy is a breed-associated myocardial disease that is typically characterized by electrical dysfunction. It is generally accepted that the syndrome of inherited VTA observed in Boxer dogs is a form of arrhythmogenic right ventricular cardiomyopathy (ARVC).

ARVC is histologically characterized by fatty or fibrofatty replacement of right and sometimes left ventricular myocardium. The phenotypic diversity of this disorder - in people, “left dominant” forms are sometimes observed - has led the suggestion that the more general designation, arrhythmogenic cardiomyopathy, might be superior to ARVC. The genetic basis of this disease in affected human beings has been extensively investigated. Numerous causative mutations have been identified although most affect genes that encode desmosomal proteins. VTA in boxer dogs is familial; the results of pedigree analysis are compatible with an autosomal dominant mode of inheritance. Meurs et al. identified a mutation of the striatin gene that is strongly associated with the ARVC phenotype in boxer dogs. The pathogenesis of ARVC is believed to involve disruption of cell-cell connections and cell death that result in impaired electrical coupling. The right ventricle is predominantly affected but left ventricular involvement is also observed. The phenotypic expression of ARVC mutations is diverse, which might be explained by genotypic heterogeneity, variable expressivity and perhaps environmental influences. Although the disease has a genetic basis, clinical signs associated with ARVC generally have an adult onset.

CLINICAL PRESENTATION

In the 1980’s Harpster classified the clinical presentation of Boxer dog cardiomyopathy:

- **Category I** - ventricular arrhythmias occur in the absence of clinical signs; these patients are identified when presented for routine veterinary care or for evaluation of non-cardiac illness
- **Category II** - syncope/collapse is observed
- **Category III** – congestive heart failure resulting from systolic myocardial dysfunction

In some Boxer dogs, there is progression from Class I to Classes II or III but this is not inevitable. Many Boxer dogs with ARVC are presented with VTA in the absence of myocardial dysfunction; that is, the echocardiogram is normal or is subtly abnormal. Dilated

cardiomyopathy is observed in boxer dogs but in many geographical regions appears to be less common than ARVC. However, published data demonstrate a strong association between the striatin mutation and echocardiographically evident myocardial dysfunction, suggesting that dilated cardiomyopathy and VTA in boxer dogs might be different phenotypic expressions of the same desmosomal defect. Recently published data suggest that arrhythmogenic cardiomyopathy also occurs in bulldogs.

History/Physical Findings

ARVC is often incidentally identified when cardiac arrhythmias are detected during routine veterinary examination. If the disease becomes clinically evident, episodes of weakness or syncope are the signs most often reported by pet-owners although tachypnea or abdominal distention due to ascites may also be observed. Premature beats and paroxysmal tachycardia are evident on physical examination. Murmurs are occasionally heard but most affected patients have minimally altered cardiac structure. Accordingly, murmurs – directly resulting from ARVC – are uncommon. Many apparently healthy boxer dogs have soft systolic basilar murmurs that reflect either a narrow aortic root – a breed-associated trait – or mild forms of aortic stenosis. However, these murmurs do not have a known relationship to ARVC.

Electrocardiographic Findings

The electrocardiographic hallmark of canine ARVC is the occurrence of VTA characterized by premature ventricular complexes (VPC) and in many cases, ventricular couplets and ventricular tachycardia. Most, but not all, ventricular ectopic complexes in affected dogs have a left-bundle block configuration; that is, the QRS is wide and upright with a negative T-wave in lead II. Some affected boxers have relatively few VPC that occur singly and infrequently while others have numerous VPC and sustained paroxysms of ventricular tachycardia (VT).

Ambulatory Electrocardiography

In the management of canine ARVC, 24 hour ambulatory electrocardiographic (Holter) monitoring can provide data that: support, or refine a provisional diagnosis, clarify the cause of syncope and possibly, guide antiarrhythmic therapy. The use of ambulatory electrocardiographic event recorders also has a role in the assessment of patients with known or suspected ARVC. These devices are digital loop recorders that can be affixed, by adhesive electrode patches, to patients for days or even weeks. A button on the device interrupts the digital loop and can be pressed if a pet-owner observes an episode of weakness or syncope. The preserved electrocardiographic data sheds light on the cause of intermittent clinical signs by providing a clear association between cardiac rhythm and patient behavior. Implantable loop recorders intended for placement within a surgically created subcutaneous pocket are also available and can usefully document electrocardiographic events that are associated with infrequent clinical episodes.

Echocardiographic Findings

In most affected individuals, the echocardiogram is normal or nearly so. Right atrial and right ventricular dilation can be observed but these findings are neither consistent nor essential for the diagnosis. Systolic myocardial dysfunction, with or without left ventricular dilation, is sometimes observed but it is not known if this is the result of histologic left ventricular involvement, the consequence of persistent tachycardia – tachycardia-induced cardiomyopathy – or reflects a distinct disease process.

DIAGNOSTIC APPROACH

The diagnostic and therapeutic approach to the patient suspected to have ARVC is determined by the clinical presentation. When a patient is urgently presented after a recent syncopal episode and a persistent, rapid ventricular tachycardia is evident, a limited diagnostic approach – perhaps electrocardiography alone – might determine the initial therapeutic strategy. More extensive diagnostic evaluation is generally appropriate for patients that are clinically stable when presented. This diagnostic evaluation may include: thoracic radiography, echocardiography, ambulatory electrocardiography, abdominal sonography and assessment of laboratory data.

When encountered in a mature boxer dog or bulldog, the occurrence of ventricular ectopy of typical QRS configuration suggests the diagnosis of ARVC. However, other cardiac disorders and indeed, non-cardiac disease, can also predispose to VTA. It is important to recognize that there is a subpopulation of boxer dogs with and without ARVC that experience syncopal episodes that are associated with transient, presumably reflex-mediated, bradycardia. When syncopal episodes are relatively infrequent, it is not always possible or practical to define the electrocardiographic rhythm during events. It is probably reasonable to assume that ventricular tachycardia is the cause of syncope when frequent, complex VTA characterized by paroxysms of VT are electrocardiographically documented. However, syncope associated with bradycardia may become more frequent if agents such as sotalol are administered. Therefore, caution is appropriate when making suppositions regarding the cause of fainting when resting electrocardiography fails to reveal dramatic arrhythmias.

THERAPY

Arrhythmias are clinically important because they can cause clinical signs - such as syncope - and because they can cause sudden unexpected cardiac death (SCD). SCD is commonly caused by rapid VT that degenerates to ventricular fibrillation (VF) - VF is a pulseless rhythm that is lethal unless promptly terminated.

There are essentially three reasons to treat arrhythmias:

1) clinical signs are associated with the arrhythmia
2) there is reason to believe that the patient is at risk of sudden death and that treatment will prevent this
3) the burden of arrhythmia places the patient at risk for the development of tachycardia induced cardiomyopathy

The subject of antiarrhythmic therapy is complex because risk factors for poor outcome are largely unknown and while recognizing that “absence of evidence is not evidence of absence”, there is no published evidence that the commonly chosen antiarrhythmic agents can prevent sudden death. It is therefore important to consider the relative risk: benefit of antiarrhythmic therapy prior to administration of antiarrhythmic agents. It is reasonable to treat arrhythmias when they are associated with clinical signs or when the arrhythmia is sufficiently severe that the development of clinical signs can be anticipated. The treatment of subclinical (“asymptomatic”) arrhythmias must be considered more carefully.

Emergent therapy of VTA associated with ARVC generally consists of parenteral therapy with agents such as lidocaine or procainamide. The pathology underlying ARVC is irreversible. Therefore, palliative therapy for canine ARVC consists of the chronic administration of oral

antiarrhythmic agents. In canine ARVC, sotalol – a potassium channel antagonist with beta-blocking properties - is the most efficacious single agent in terms of arrhythmia suppression. Whether or not this efficacy translates to a reduction in sudden death, is not known. Data from ambulatory electrocardiographic monitoring is often use to evaluate the effectiveness of therapy, though the value of this approach has not been demonstrated and it is relevant that the frequency of ventricular arrhythmia in affected boxers might vary by as much as 75% independent of therapy. Monitoring of clinical signs is a practical method by which effectiveness of therapy can be judged. In clinically stable patients, careful consideration of the relationship between arrhythmia and clinical signs should guide therapy as recently published data suggest that ventricular tachycardia is not the most important cause of collapse in boxer dogs.

It is not possible to make definitive statements with regard to prognosis. Occurrence of syncope and more frequent or complex VTA are associated with poor outcome. However, studies of the natural history of boxer ARVC provide evidence that survival of affected dogs does not differ from that of unaffected boxer dogs.

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Diagnosis and Management of Pericardial Disease
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Pericardial disease is not common but its unique pathophysiology demands a therapeutic approach that differs from the more common forms of heart disease. Furthermore, pericardial disease is occasionally curable which emphasizes the importance of its recognition.
The parietal pericardium consists of an outer fibrous layer and an inner serosal layer. The visceral pericardium, or epicardium, is a reflection of the serosal surface of the parietal pericardium. The pericardial cavity between the parietal and visceral pericardia is a potential space. In healthy individuals, it contains a small volume of serous transudate. The normal pericardium appears to fulfill only a minor role in cardiovascular function and its congenital absence does not result in clinical consequences. However, disease of the pericardium impairs cardiac filling and may lead to marked cardiovascular compromise. Congenital abnormalities of the pericardium are relatively uncommon. During this session, acquired, effusive pericardial disease of the dog will be emphasized.

PERICARDIAL EFFUSION

In dogs, pericardial effusion (PE) usually is hemorrhagic; numerous causes have been reported but most commonly, canine PE is due to intrapericardial neoplasia or idiopathic pericarditis. The prevalence of neoplastic effusion is greater than the prevalence of idiopathic PE. Right atrial hemangiosarcoma (HSA) and chemodectoma are the most common cardiac neoplasms but mesothelioma, ectopic thyroid carcinoma, rhabdomyosarcoma and metastatic neoplasia occasionally cause PE. Except in specific geographical regions, infective pericardial disease is rare.

Pathophysiology of Cardiac Tamponade

The consequences of PE primarily depend on the volume of pericardial fluid and the compliance characteristics of the pericardium; because of the latter, the rate at which fluid accumulates importantly determines the consequences of effusion. Acutely, the pericardium is minimally distensible. Therefore, when PE accumulates rapidly, relatively small volumes cause intrapericardial pressures (IPP) to rise resulting in impaired ventricular filling and hemodynamic compromise. In contrast, the pericardium can stretch to accommodate an effusion that develops slowly and large volumes of fluid may accumulate before IPP impairs cardiac filling. The syndrome of cardiac compression that results from accumulation of pericardial fluid is known as cardiac tamponade. In health, right atrial and ventricular pressures are lower than corresponding pressures of the left atrium and ventricle; because of this, the right side of the heart is initially affected by tamponade. Progressive increases in intrapericardial pressure cause equalization of left and right ventricular filling pressures, after which further increases in IPP cause ventricular filling pressures to rise in tandem. The increase in ventricular filling pressures causes venous pressures to increase, and when PE is chronic, signs of systemic congestion including ascites and pleural effusion are observed. When pericardial fluid accumulates rapidly, clinical signs of diminished peripheral perfusion typically dominate the clinical presentation.

Clinical Presentation

PE develops most commonly in large-breed dogs including retrievers and German shepherd dogs. Often, PE develops relatively slowly and patients are presented for evaluation of signs of right-sided congestive heart failure. Abdominal distention due to ascites or dyspnea related to concurrent pleural effusion may be observed. Signs of low cardiac output including weakness and syncope may also prompt veterinary evaluation and non-specific signs including inappetance, depression and lethargy are common. Patients that acutely develop severe tamponade are presented recumbent in circulatory collapse. The physical findings of cardiac tamponade are relatively distinctive. The heart sounds usually are muffled by the presence PE. Tachycardia is typically present and the arterial pulse may be weak. Pulsus paradoxus – a decrease in the strength of the arterial pulse during inspiration - is occasionally detectable; this finding is virtually pathognomonic for the presence of cardiac tamponade. When tamponade is present, the external jugular veins often have an abnormal appearance due to distention; the height of visual pulsations reflects right atrial pressure.

Diagnostic Evaluation - Radiography

When large volumes of PE are present, the cardiac silhouette is enlarged and the contours of the cardiac silhouette are lost so that cardiac shadow has a globose appearance. Often, the pulmonary vessels are small. Pulmonary edema is uncommon in the setting of tamponade.

Diagnostic Evaluation - Electrocardiography

Tachycardia is usually present when patients develop tamponade. Most often, the rhythm is sinus; pathologic tachyarrhythmia such as ventricular premature complexes and ventricular tachycardia are relatively uncommon. When the volume of PE is large, the amplitude of the QRS complexes may be markedly diminished. Occasionally, the QRS amplitude varies in a consistent alternating fashion known as electrical (or QRS) alternans.

Diagnostic Evaluation - Echocardiography

Echocardiography is invaluable in the diagnostic evaluation of patients with pericardial disease. It is the most sensitive and specific noninvasive means by which to detect PE. The size of the effusion can be semi-quantitatively evaluated, but the compliance characteristics of the parietal pericardium and the speed at which the effusion accumulates are more important determinants of clinical consequence. Because of this, a small effusion that results from intrapericardial hemorrhage can have catastrophic consequences while a large effusion that has accumulated slowly may result in minimal hemodynamic consequences. Diastolic inversion of the parietal walls of the right atrium and right ventricle provide evidence of cardiac compression with the latter having greater diagnostic specificity for identification of tamponade. Echocardiography should be considered in all cases of suspected pericardial disease because it is the diagnostic test that is most likely to provide an etiologic diagnosis. Although two-dimensional echocardiography is a highly sensitive method for detection of PE, the sensitivity of 2DE for identification of cardiac masses has not been prospectively evaluated. Based on retrospective studies, the sensitivity of 2DE for detection of cardiac neoplasia is between 17 and 82%. When multiple imaging planes are evaluated using current echocardiographic technology, it is reasonable to suppose that the true figure is close to the upper limit of this range.

Diagnostic Evaluation – Characteristics of the Effusate

Most pericardial effusions in dogs result either from neoplasia or from idiopathic pericarditis. The prognosis associated with idiopathic pericarditis is better than that associated with neoplasia and therefore, antemortem distinction between the two disease processes is important. Ideally, the two causes could be distinguished by minimally invasive techniques but

unfortunately a reliable means to do so is lacking. Cytologic evaluation of PE is rarely informative probably because almost all PE in dogs are hemorrhagic and the most common tumors exfoliate poorly. Effusions related to cardiac lymphosarcoma or infection are exceptions, but these conditions are uncommon. The non-invasive etiologic diagnosis of PE generally is made by echocardiography and clinical course.

Therapy

Pericardiocentesis is the appropriate initial therapy for patients with cardiac tamponade. The procedure is generally performed from the right hemithorax after aseptic preparation of the site. A 14 or 16 G over-the-needle catheter is introduced into the pericardial space, the needle is removed and fluid is withdrawn. A pause after removal of the first 3-5 ml allows time to determine whether or not the fluid will clot. If the aspirated fluid clots, it is possible that the catheter is within the right ventricle. Monitoring of the electrocardiogram during the procedure is suggested. The procedure can be performed blindly although some prefer to puncture the pericardium using echocardiographic guidance. Complications are relatively uncommon and the risk:benefit ratio is in favor of performing the procedure when tamponade is present.
It should be recognized that the vast majority of canine PE are hemorrhagic. Diuretic agents are unlikely to mobilize the effusion but are likely to decrease venous pressures and cardiac output. For this reason, diuretics are contraindicated in the setting of tamponade. Surgical exploration after pericardiocentesis can be considered when echocardiography demonstrates an intrapericardial mass. In cases in which a mass is detected, it is useful to consider the presumptive histologic diagnosis. Masses that originate from the right atrium or right atrial appendage are usually HSA while chemodectomas generally arise from the proximal aorta. Surgical debulking and adjuvant chemotherapy may improve survival of patients with cardiac HSA. However, it should be recognized that this neoplasm is associated with a poor prognosis and survival after diagnosis is generally less than 8 months regardless of therapy. Patients with chemodectoma fare better and median survival of 730 days after pericardiectomy has been reported. Approximately 50 % of idiopathic PE resolve after a single centesis. Surgical exploration and pericardiectomy should be considered when apparently idiopathic PE recurs after two or three centeses. Surgical exploration provides a definitive diagnosis and when idiopathic pericarditis is confirmed, pericardiectomy is potentially curative. Subtotal pericardiectomy traditionally has been performed after a median sternotomy or lateral thoracotomy. More recently, minimally invasive techniques for pericardiectomy or pericardiotomy have been developed. Thoracoscopic subtotal pericardiectomy has been reported and this technique is apt to result in lower patient morbidity relative to thoracotomy although, based on retrospectively determined post-procedural survival, there may be an advantage to thoracotomy for patients suspected to have idiopathic PE. Balloon pericardiotomy is a minimally invasive technique in which a catheter of the type used for balloon dilation of outflow tract obstructions is used to create a rent in the pericardium and therefore prevent recurrence of tamponade. This procedure may have a favorable cost:benefit ratio particularly as a palliative procedure for patients with neoplastic effusion.

Prognosis

The prognosis associated with effusive pericardial disease is largely determined by the cause of the effusion. In general, patients with cardiac HSA have a poor prognosis. The prognosis after pericardiectomy for patients with chemodectoma may be surprisingly good, presumably because the tumors grow relatively slowly and are late to metastasize. Although prolonged survival has been documented, patients with pericardial mesothelioma generally fare poorly

and have decreased survival relative to dogs with idiopathic PE. Patients with idiopathic pericardial effusion may have a good or excellent prognosis. A few clinical findings provide prognostically useful information. Patients that have ascites when first presented for veterinary evaluation live longer than those that do not. Presumably this is because patients with aggressive, hemorrhagic tumors such as HSA are more apt to develop signs associated with circulatory collapse than signs of congestion. Independent of histologic findings, the prognosis is generally poor for patients with an echocardiographically identified mass that originates from the right atrium.

CONSTRICTIVE PERICARDITIS

Constrictive pericarditis is an uncommon disease that impairs diastolic function. Ventricular filling is impeded due to pericardial constriction that is due to pericardial fibrosis. Clinical signs are generally related to systemic congestion and low cardiac output. The disease is a diagnostic challenge; cardiac catheterization studies are generally required. Surgical treatment consists of pericardial stripping.

References available from the author.

Managing Cardiac Cases…When Echo is Unavailable
CFAVM February 2023
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The therapeutic plan is optimally based on a definitive diagnosis. In cardiovascular medicine, a definitive diagnosis sometimes requires echocardiography, and even when that is not the case, the results of echocardiographic examination often provide ancillary data that usefully further therapeutic objectives. However, echocardiography is not always available and of course it is common that the practice of veterinary medicine is limited by financial constraints on the part of the client. During this session, a case-based format will be used to illustrate an approach to common cardiovascular presentations that is rational when echocardiography is not available. Implicit is an understanding that case management might not be optimal, but pragmatism sometimes demands an approach that is the best that can be achieved under the circumstances.

DIAGNOSTIC METHODS IN CARDIOVASCULAR MEDICINE - RELATIVE MERITS

An understanding of the relative merits of available diagnostic methods is necessary for a measured clinical approach that is both pragmatic and rational. This requires an appreciation of the uses and limitations of each modality.

Echocardiography

Echocardiography is the use of reflected ultrasound to define cardiac structure and function. It allows relatively precise measurement of specific cardiac chambers and is not subject to one of the primary limitations of thoracic radiography; the septae can be directly visualized and the ability of echocardiography to resolve specific chambers of the heart is excellent. Doppler echocardiography is a form of echocardiography that provides information regarding velocity, direction and character of blood flow. Doppler can be used to localize flow disturbances; in a sense, identify murmurs. However, echocardiography provides limited information regarding the consequences of cardiac dysfunction - echocardiography can provide supportive evidence, but the syndrome of congestive heart failure is generally a clinical and/or radiographic diagnosis. Although echocardiographic indices of myocardial function occasionally guide therapy, in most cases, echocardiography is used to provide etiologic information. While that information can be crucial, the etiologic nature of cardiac disease - so far as it actually determines therapy - sometimes can be assumed. For example, the probability that a geriatric small-breed dog with an acquired systolic murmur and radiographic evidence of left atrial enlargement has anything other than degenerative mitral valve disease is small. In some cases, echocardiography is required for ante-mortem diagnosis, but in others the information provided is confirmatory or ancillary. When echocardiography is unavailable, it is important to consider the information that this examination test might reasonably be expected to provide and, the epidemiological principles that determine diagnostic probabilities.

Thoracic radiography

The chest film includes only a silhouette of the heart - radiographically, the heart appears as a single fluid density - and the interatrial and interventricular septae are not visualized so that specific cardiac chambers cannot be resolved with certainty. Furthermore, the pericardial space cannot be distinguished from the epicardium; thus, a patient with a large pericardial effusion and cardiac tamponade might have a large radiographic cardiac silhouette when in fact, the heart itself is small. However, despite these limitations, chest X-rays provide a means to evaluate cardiac size in association with the appearance of the pulmonary vessels and parenchyma which, in turn, provides an indirect assessment of cardiac performance; the findings of left atrial enlargement together with pulmonary congestion/edema is evidence of impaired emptying and/or filling of the left ventricle. In dogs, left atrial enlargement often results in a distinctive distortion of the cardiac silhouette which is fortunate because left atrial enlargement is a surrogate measure of hemodynamic burden in the common, chronic cardiac diseases. The limitations of thoracic radiography are more pronounced in the evaluation of feline patients because the appearance of left atrial enlargement is generally less distinct than it is in the dog. But even then, radiographic cardiomegaly is often associated with the presence of clinically important feline myocardial disease.

Electrocardiography

The primary utility of electrocardiography is the elucidation of cardiac rhythm disturbances - arrhythmias. The electrocardiogram provides some information concerning cardiac chamber size but it is an insensitive means to detect heart enlargement.

Blood-borne Biomarkers

Biomarkers are objectively determined characteristics that potentially have a role in diagnosis, risk stratification, evaluation of disease progression, and evaluation of response to therapy. In the management of cardiac disease, B-type natriuretic peptide [BNP] is the most important of these. BNP is released from cardiomyocytes in response to increases in cardiac wall tension and supraphysiologic concentrations of BNP potentially reflect the heart failure state and this perhaps, is the primary utility of this test. Quantitative assays of BNP are commercially available for both canine and feline patients, but these tests must be performed at a central laboratory. A point of care assay for feline BNP is available. Like most diagnostic tests, the BNP assay is useful but imperfect. Increases in BNP generally reflect the presence of cardiac disease but the finding is not etiologically specific; practically any cardiac disease can raise the BNP concentration and incidentally identified increases sometimes reflect “false positives”. The point of care assay may have particular use in the evaluation of feline patients with respiratory distress.

DIAGNOSTIC APPROACH

Implicit in a pragmatic and rational diagnostic approach is an understanding of the clinical importance of physical examination findings and the way that epidemiology determines etiologic probabilities. A case-based format will be used to illustrate these diagnostic principles.

Current management of Canine Congenital Heart Disease
CFAVM February 2023
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This session is intended to provide a synopsis of the current diagnostic and therapeutic approach to congenital heart disease (CHD). The management of *canine* CHD, including the use of minimally invasive, transcatheter techniques in the most commonly occurring malformations will be emphasized.

ETIOLOGY OF CHD

The cause of canine CHD is largely unknown although a genetic basis has been proven for a few specific malformations. Barring identification of a genetic mutation that is consistently associated with an abnormal phenotype, planned breeding studies or careful evaluation of accurate pedigrees are necessary to demonstrate genetic transmission of a congenital malformation. Indeed this has been accomplished for a few defects. For example, subvalvular aortic stenosis is an inherited trait in Newfoundland dogs, genetic transmission of pulmonic stenosis has been demonstrated in the beagle hound and a spectrum of conotruncal malformations that includes Tetralogy of Fallot is inherited in keeshonden. Pronounced breed predispositions are recognized for some forms of canine CHD and in these cases, it is probable that the defect has a genetic basis.

RECOGNITION AND DIAGNOSIS OF CONGENITAL HEART DISEASE

The patient history of those with CHD rarely provides specific findings and the majority of canine patients with CHD are free of clinical signs when the disorder is first detected. Importantly, normal growth and lack of clinical signs do not imply that CHD is of no clinical importance nor necessarily imply a favorable long-term prognosis. This serves to emphasize the importance of accurate diagnosis even for cases in which clinical signs are absent.
The vast majority of cardiac malformations result in cardiac murmurs. Therefore, congenital heart disease usually is first identified when outwardly healthy puppies or kittens are presented for routine veterinary evaluation. It is noteworthy that some normal puppies and kittens have murmurs that do not result from cardiac disease. These murmurs, known as innocent murmurs, are soft, always systolic, and usually heard best over the left heart base. The intensity of an innocent murmur may vary from day to day or even from moment to moment in association with changes in heart rate. Murmurs that are innocent generally become inaudible before the patient is 8 months of age. While congenital disease can result in a soft murmur, a loud cardiac murmur, or one that is diastolic or continuous, invariably suggests the presence of cardiac disease and further diagnostic investigation is indicated. Further evaluation may include electrocardiography and thoracic radiography but these tests rarely provide diagnostically specific information. Doppler echocardiography performed by an experienced examiner provides a definitive, non-invasive diagnosis in practically all cases of CHD.

THERAPY OF CONGENITAL HEART DISEASE

Optimally, treatment of CHD is through surgical methods or interventional cardiac catheterization techniques. Medical therapy is apt to be palliative only.

Surgical Techniques / Cardiopulmonary Bypass

The availability of cardiopulmonary bypass - or rather, its lack - is one factor that limits the effective management of CHD in veterinary medicine. A few defects can be surgically managed without the need for bypass; a PDA for example, can be ligated without entering the circulation. However, defects that require access to the left ventricle and/or prolonged manipulation can only be performed with cardiopulmonary bypass. Surgical procedures that require cardiopulmonary bypass currently are performed only at a few veterinary institutions.

Interventional Catheterization Techniques

Originally, cardiac catheterization - the art and science of manipulating catheters within the cardiovascular system - was a diagnostic technique. Usually under fluoroscopic guidance, catheters can be used to selectively deliver dye, to measure blood oxygen contents and to directly measure intracardiac pressures. Beginning in the 1960's, a number of resourceful pediatric cardiologists introduced catheterization techniques that were intended to treat or "intervene". The techniques employed most often in veterinary cardiology are pulmonic balloon valvuloplasty and transcatheter occlusion of patent ductus arteriosus

PATENT ARTERIAL DUCT (Patent Ductus Arteriosus - PDA)

The arterial duct (AD) connects the ventral aspect of the descending aorta to the bifurcation of the main pulmonary artery. During fetal life, the AD diverts the majority of the right ventricular stroke volume to the aorta. In normal individuals, closure of the ductus occurs within days of birth; the process is complex but involves a prostaglandin cascade. Failure of the duct to close, which in most cases is explained by a lack, or relative lack, of ductus specific smooth muscle, results in a persistently patent AD or, "PDA". A genetic basis for failed closure of the AD has been documented in miniature / toy poodles. When the duct is the only defect, and pulmonary vascular resistance decreases following birth, blood shunts from the high pressure / high resistance systemic circulation to the low pressure / low resistance pulmonary circulation. Therefore, the shunt direction is from left-to-right; this increases pulmonary blood flow *and* pulmonary venous return imposing a volume load on the left atrium and ventricle. The development of myocardial dysfunction, mitral valve regurgitation and pulmonary edema are potential consequences of the shunt. PDA results in a continuous murmur; that is, a murmur that begins in systole and continues**,** without interruption**,** into diastole. When the shunt is substantial, the arterial pulse is hyperkinetic or, "bounding". Electrocardiography often discloses evidence of left ventricular hypertrophy. Radiographically, there is left-sided cardiomegaly that is roughly commensurate with the size of the shunt**.** Additional findings may include prominence of the main pulmonary artery, proximal aorta and left atrium. The diagnosis is confirmed echocardiographically. Specific findings of course depend on the size of the shunt but typically include left atrial and left ventricular enlargement. Doppler examination provides evidence of a continuous flow disturbance within the main pulmonary artery. The duct, and certainly the ductal orifice of the pulmonary artery**,** can be identified in almost all cases.

Therapy

A minority of patients have a small, well-tolerated duct but usually, intervention is indicated when a PDA is identified in a dog that is younger than 24 months old. Surgical ligation can be performed without cardiopulmonary bypass and though minimally invasive transcatheter

occlusion has become routine, surgical ligation remains an appropriate therapeutic approach that is associated with low mortality.
Transcatheter PDA occlusion using different devices and subtly different techniques have been reported. Thrombotic Gianturco coils were widely used until two veterinary cardiologists, Ngyuenba and Tobias, in collaboration with a manufacturer of cardiovascular devices, developed a metallic plug, the Amplatz Canine Ductal Occluder (ACDO), that was specifically designed to occlude the canine ductus. Use of this device has almost completely supplanted the use of the Gianturco coil in veterinary practice. The ACDO can be used to successfully occlude PDA over the broad range of ductal size and morphologies.
Numerous variations on the basic technique of transcatheter ductal occlusion have been reported. Most often the devices are deployed within the ductus after retrograde catheterization of the aorta. Briefly, after induction of general anesthesia, access to the femoral artery is most often obtained after a small inguinal incision but vascular access can be percutaneously obtained using the modified Seldinger technique. Using fluoroscopic guidance, an angiographic catheter and/or vascular sheath is advanced to the ascending aorta and an angiogram recorded after injection of contrast material in the proximal descending aorta. Then, the device is advanced through a catheter or vascular delivery sheath and deployed within the ductus. Major complications of transcatheter intervention for PDA include intra-operative death, incomplete occlusion, post-procedural hemolysis, and coil migration. Mortality associated with transcatheter intervention for PDA generally is quite low, near 2%, although higher mortality has been reported in small studies that specifically recruited high risk patients. Body size is an important determinant of the suitability of the technique. Because of the size of the delivery devices, transcatheter occlusion of PDA in patients that weight less than 3 kg is problematic.

SUBVAVULAR AORTIC STENOSIS (SAS)
In dogs, left ventricular outflow tract obstruction most commonly results from the presence of a subvavular fibrous or fibrocartilaginous ring that develops in the first weeks of life. The pressure gradient across the obstruction, which can be measured - by cardiac catheterization - or estimated - by Doppler echocardiography - is used as a clinical measure of stenosis severity. Pressure gradients that are less than 40 mmHG are mild and those greater than 100 mmHg are severe; intermediate gradients are described as moderate. Aortic stenosis is most common in large breed dogs including Golden retrievers, Rottweilers, as well as Boxer, Newfoundland and German Shepherd dogs. Clinical signs in puppies are uncommon; syncope and sudden death are observed in young adults with severe obstructions.
Cardiopulmonary bypass is required for surgical repair. Although a new technique in which a “cutting balloon” is used might have promise, published data suggest that neither surgical correction nor balloon dilation improve survival relative to medical therapy consisting atenolol. Evidence that administration of atenolol is superior to placebo or no treatment is lacking.

PULMONIC STENOSIS (PS)
Right ventricular outflow tract obstruction usually results from valvular dysplasia. PS occurs commonly in terriers, English Bulldogs, miniature schnauzers and Samoyeds.
Therapy
PS can be treated surgically but is more often addressed by transcatheter balloon dilation. The pressure gradient that represents an indication for intervention is not known with precision although it is known that balloon dilation confers a survival benefit for those with gradients that exceed 80 mmHg. Balloon dilation of PS is performed under general anesthesia. After aseptic

preparation of the groin or cervical region, access to the jugular or femoral vein is typically obtained percutaneously. After hemodynamic and angiographic studies, an end-hole diagnostic catheter is guided fluoroscopically into the pulmonary artery. The end-hole catheter is then exchanged for a balloon dilation catheter over a long wire-guide. Balloon catheters are available from manufacturers in numerous sizes; appropriate dimensions are determined by the size of the patient and echocardiographic (or angiographic) measurement of the pulmonic valve annulus. Several inflations with saline-diluted contrast material are performed after the balloon has been positioned across the stenosis. The inflation is observed fluoroscopically; the obstructive valve results in the appearance of a “waist”; ideally, the waist disappears suddenly during the first inflation and is not observed during subsequent attempts. After balloon inflation, catheters are withdrawn and patient is recovered. Most patients are discharged the day after the procedure which permits echocardiographic re-evaluation after complete recovery from anesthesia. Success seems to depend to a great extent on the nature of the stenosis; patients with isolated valvular stenosis in which there is fusion of otherwise normal valve cusps tend to benefit the most from the procedure.

VENTRICULAR SEPTAL DEFECT

A defect of the interventricular septum results in a communication between the left and right ventricles. Presumably because of its complex embryonic derivation, most ventricular septal defects (VSD) involve the membranous part of the septum; generally these defects are subaortic with a right ventricular orifice that is immediately subjacent to the septal tricuspid leaflet. The clinical importance of the defect depends on: the size of the defect and, the presence or absence of other concurrent defects. When the VSD is the only cardiovascular lesion, blood shunts from left-to-right; this increases pulmonary blood flow and pulmonary venous return resulting in a volume load on the left atrium and left ventricle. A VSD results in a systolic murmur; this is because the pressure difference between the ventricles drops to nearly zero during diastole and shunting during this phase of the cardiac cycle is negligible.

Therapy

Definitive repair requires cardiopulmonary bypass but pulmonary artery banding is occasionally used as a palliative surgical intervention. Transcatheter occlusion of VSD is routinely practiced in pediatric cardiology and transcatheter closure in dogs has been reported. Most VSD in dogs are small, well tolerated and do not require treatment.

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Cardiac Radiology - Brief Review
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This session is intended to provide a review of diagnostic principles that underlie interpretation of chest films obtained from patients known or suspected to have cardiac disease. A pragmatic approach, emphasizing therapeutic implications, will be presented. Objective methods of cardiac mensuration will be introduced.

UTILITY/LIMITATIONS/INDICATIONS

Thoracic radiography allows evaluation of cardiac size in relation to the pulmonary vessels and parenchyma. Because of this, the chest film provides an indirect assessment of cardiac performance; the findings of left atrial enlargement, pulmonary venous distention and pulmonary edema are indirect evidence of impaired emptying and/or filling of the left ventricle. Of course, relative to other imaging modalities, there are limitations. The chest film provides only a silhouette of the heart; the interatrial and interventricular septae are not visualized and so the specific cardiac chambers cannot be resolved with certainty. The limitations of thoracic radiography are pronounced in the evaluation of feline patients and most of the statements below relate specifically to he canine X-ray film.

A discussion of the radiographic technique is not within the scope of this review. However it is axiomatic that the chest film must be interpreted in the context of patient position and the phase of respiration during which the exposure is made. The implication of the latter with respect to the appearance of the lung is obvious but it is also relevant to subjective evaluation of cardiac size. When the cardiac silhouette is evaluated subjectively, to some extent the cardiothoracic ratio is determinative. If the thoracic cavity is small - as it is in a relative sense, during expiration - then the cardiac silhouette appears to be large.

Indications

Broadly, in the context of cardiac disease, there is an indication for thoracic radiography when there is a history of cough, tachypnea or respiratory distress that can be related - based on the history and physical examination - to cardiac disease. Focused sonographic examination can sometimes appropriately take the place of radiography but is not always available and has imperfect predictive value for identification of therapeutically relevant abnormalities such as pulmonary edema.

Although syncope, embolism and cyanosis occasionally are occasionally explained by cardiac disease, clinical signs of cardiac dysfunction most often result from venous congestion. Cardiogenic pulmonary edema causes tachypnea/respiratory distress and sometimes in canine patients, cough. Pleural effusion, particularly in feline patients, is an important manifestation of cardiac dysfunction. The pathophysiologic basis of edema and effusions in the setting of cardiac disease is diagnostically relevant. Rises in ventricular filling pressures - meaning, increases in end-diastolic pressures - are reflected back on the atrium, the venous circulation, and the capillaries and it these elevated pressures that are responsible for the development of edema/effusion. In some diseases, atrioventricular valve regurgitation contributes to the rise in atrial pressures. Left atrial pressure and and

left atrial size are different quantities, but in chronic disease, they are related. Because the common cardiac diseases are chronic and progressive, left atrial enlargement is essentially a requisite for the diagnosis of cardiogenic edema. Rare exceptions to this are rupture of a mitral chord and endocarditis which can occasionally cause rapid increases in atrial pressure that result in congestive signs prior to the development of chamber enlargement.

ASSESSMENT OF THE CARDIAC SILHOUETTE

Global cardiac size can be evaluated subjectively, but cardiac mensuration might have a particular utility for inexpert observers. The vertebral heart score [vhs] first described by Buchanan, has been studied in both canine and feline patients. Although there are limitations to this technique - not least of these is a breed effect - it is a useful, objective means of evaluating the size of the cardiac silhouette. It is also relevant that a VHS in excess of 10.5 was an inclusion criterion for the clinical trial that established the effect of pimobendan on canine patients with asymptomatic mitral valve disease, and therefore the VHS has direct therapeutic implications.
There are minor variations, but the vertebral heart score is calculated from the two perpendicular dimensions of the cardiac silhouette viewed in the lateral projection. These two dimensions are defined in terms of the lengths of vertebral bodies, beginning at the fourth thoracic vertebra. In general, a VHS that exceeds 10.5, is evidence of cardiomegaly but there is an important breed effect. Cavalier King Charles spaniels, to name only one example, commonly have a VHS that exceeds this figure in the absence of echocardiographically evident cardiac disease. A similar method has been applied to feline patients. In this species, a VHS > 8.3 generally reflects cardiomegaly.

Left Atrial/Left Ventricular Enlargement

Left atrial size is a surrogate marker of hemodynamic burden and reliable methods of identification of atrial enlargement are clinically useful. The canine vertebral left atrial size [VLAS] - measured from the ventral border of the bifurcation of the trachea to the union of the dorsal border of the caudal caval vein and the cardiac silhouette - can be measured in terms of vertebral bodies analogous to the VHS method. A similar method has been applied to the cat.
The left atrium is left of, and caudal to, the right atrium. Radiographically, it occupies the caudodorsal area of the cardiac silhouette in the lateral projection. In the absence of left atrial enlargement, the caudal portion of the trachea curves ventrally over the caudal aspect of the cardiac silhouette. In canine patients, when the left atrium is enlarged, the caudal border of the cardiac silhouette straightens, and the trachea is forced dorsally to varying degrees. With marked left atrial enlargement, the left mainstem bronchus is narrowed, and the trachea adopts a path that is parallel to the thoracic vertebrae. Occasionally, severe left atrial enlargement has the appearance of a mass that separates the mainstem bronchi. In the orthogonal projection, the left atrium is located near the center of the cardiac silhouette. When enlarged, the left atrium splits the mainstem bronchi potentially resulting in a “double opacity” and an appearance that is sometimes known as the “crab sign” or the “bowlegged cowboy”. Additionally, in the ventrodorsal view, enlargement of the left atrium may cause a bulge which represents the atrial appendage at the 3 o’clock position. In general, radiographic evaluation of left atrial size is more reliable in dogs that it is in cats.

It general, the silhouette of the left ventricle cannot be reliably distinguished from that of the left atrium. There are exceptions, but many disorders that result in left atrial enlargement also result in left ventricular enlargement, while left ventricular enlargement in the absence of left atrial enlargement is uncommon. In general, when viewing the lateral projection, a canine cardiac silhouette that has enlarged in the long-axis usually reflects left-sided cardiomegaly.

Right Atrial/Right Ventricular Enlargement

Radiographic assessment of right atrial/right ventricular size is difficult and in general, there are few diagnostically reliable markers of chamber enlargement. And concurrent enlargement of the left atrium/left ventricle makes the task more difficult still. In canine patients, a heart that is “tall” in the lateral view reflects enlargement of the left ventricle and/or left atrium. A cardiac silhouette that has enlarged in the craniocaudal dimension - when evidence of left atrial enlargement is lacking - generally right-sided cardiomegaly or a large pericardial effusion.

RADIOGRAPHIC FINDINGS OF CONGESTION AND EDEMA

Pulmonary venous distention suggests pulmonary congestion and may precede the development of pulmonary edema. However, it is known that this finding is not a diagnostically sensitive marker of feline pulmonary edema and the same might be true of canine edema. Presumably, in most cases, the development of interstitial pulmonary edema precedes the appearance of alveolar edema. Blurring of vascular detail in the presence of left atrial enlargement and, sometimes, concurrent pulmonary venous distention characterizes the radiographic appearance of interstitial pulmonary edema. When tissue fluid weeps into the pulmonary alveoli, it provides contrast with air-filled structures such as the bronchi, resulting in air bronchograms. Alveolar pulmonary opacities together with radiographic evidence of left atrial enlargement are diagnostic of left-sided congestive heart failure. The presence of alveolar pulmonary edema indicates severe CHF that is almost invariably associated with noticeable respiratory distress. In dogs, a central, or perihilar, distribution sometimes characterizes cardiogenic pulmonary edema but cardiogenic edema is commonly asymmetrical and focal; there is a predilection for the right caudal lung lobe. Pulmonary edema in cats generally is patchy and diffusely distributed. in cats, but generally not in dogs, pleural effusion in the absence of ascites is a relatively common manifestation of congestive heart failure, and this is the case even when the causative disease appears to affect the left ventricle primarily.

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