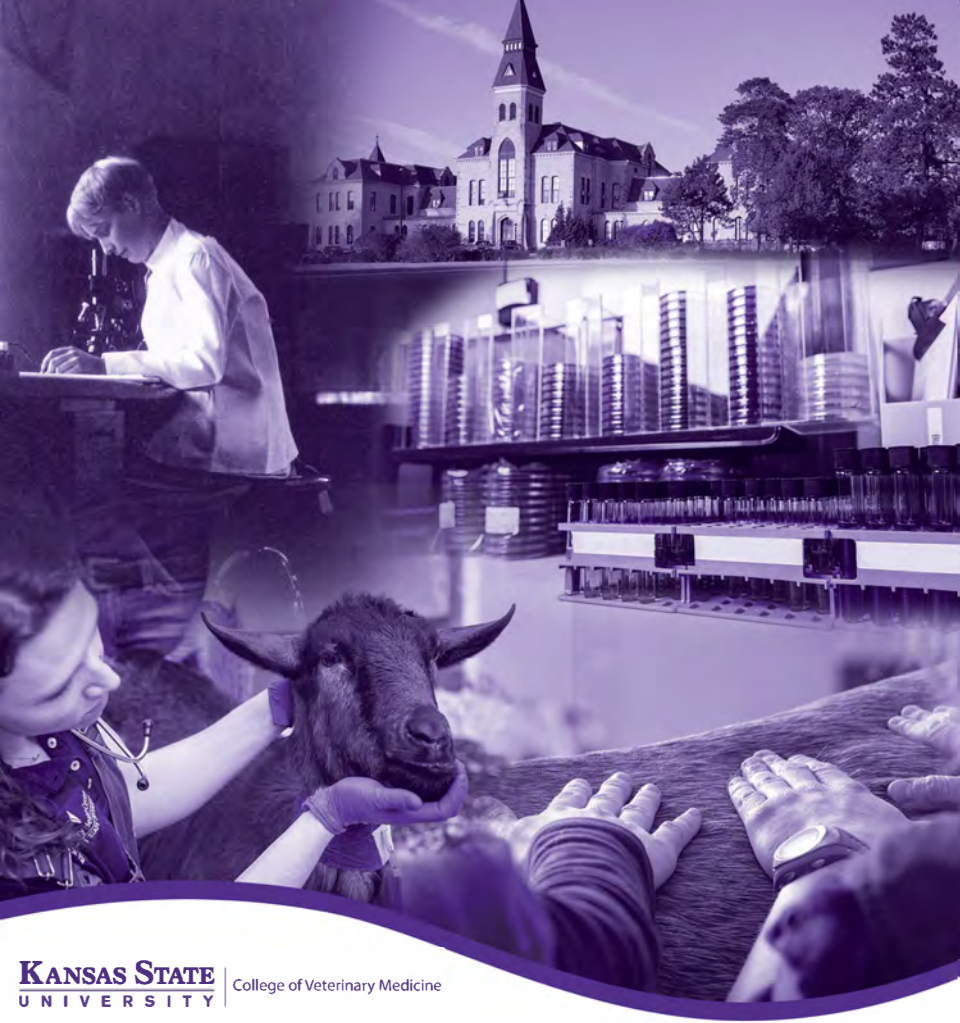

KANSAS STATE UNIVERSITY | College of Veterinary Medicine

82nd Annual Conference for Veterinarians

Sunday, May 31 – Monday, June 1, 2020

VIRTUAL CONFERENCE

Small Animal Proceedings

Feline Uveitis: Ocular Manifestations of Systemic Disease in the Midwestern United States

Dr. Jessica Meekins, Kansas State University

Feline Uveitis in the Midwestern US

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Outline

- Brief review of uveal tract anatomy
- Clinical signs of uveitis
- Differential diagnoses
  - Regional considerations**
- Diagnostic workup
- Treatment and prognosis

Anterior Uveal Tract

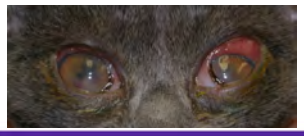
- Blood Aqueous Barrier
  - Endothelial cells of iris vessels
  - Tight junctions between nonpigmented ciliary body epithelium
- Functions to prevent passage of almost all protein, cells, other materials into anterior chamber

Posterior Uveal Tract

- Blood Retinal Barrier
  - Endothelial cells of retinal vessels
  - Tight junctions between RPE
- Blood Aqueous Barrier + Blood Retinal Barrier = 'Blood Ocular Barrier'

Uveitis = Inflammation of Uveal Tract

- Anterior uveitis → iridocyclytis
- Posterior uveitis → chorioditis
- Panuveitis → anterior and posterior uveitis



Hallmark Signs of Anterior Uveitis

- Normal

- 1. Aqueous flare

Hallmark Signs of Anterior Uveitis

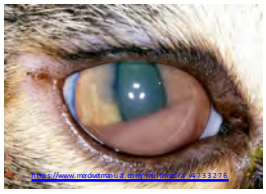
- 2. Miosis

Hallmark Signs of Anterior Uveitis

- 3. Hypotony = *lower* than normal IOP

Other Signs of Anterior Uveitis

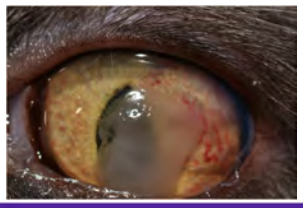
- Hypopyon



- Hyphema

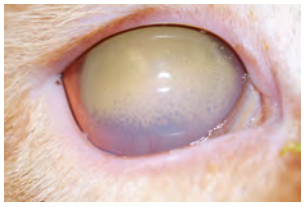
Other Signs of Anterior Uveitis

- Fibrin in anterior chamber



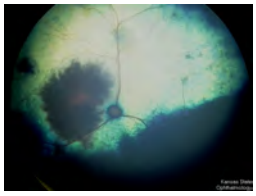
Other Signs of Anterior Uveitis

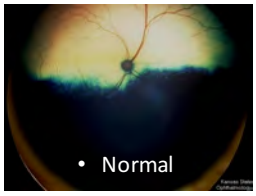
- Keratic precipitates: WBC accumulates adhering to corneal endothelium



Signs of ACTIVE Posterior Uveitis

- Subretinal cellular infiltrate/granuloma = micro-detachments

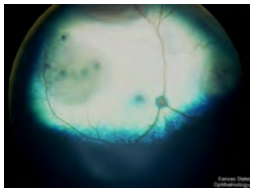


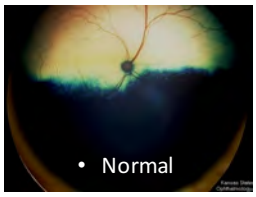


- Normal

Signs of ACTIVE Posterior Uveitis

- Chorioretinitis

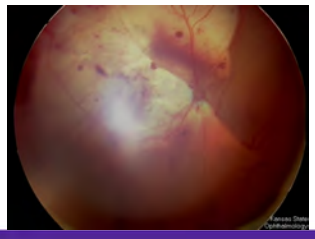




- Normal

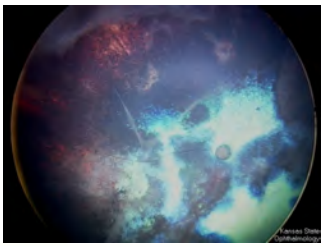
Signs of ACTIVE Posterior Uveitis

- Retinal hemorrhages
- Retinal detachments (geographic)



Signs of INACTIVE Posterior Uveitis

- = Chorioretinal scars
- Tapetal hyperreflectivity
- Retinal degeneration



Causes of Uveitis in Cats

- Bacterial
  - *Bartonella henselae?*
- Fungal
  - *Histoplasma capsulatum*
  - *Blastomyces dermatitidis*
  - *Cryptococcus neoformans*
  - *Coccidioides immitis*

**Immune-mediated
Chronic lymphocytic/plasmacytic

- Protozoal
  - *Toxoplasma gondii*
  - *Cytauxzooan felis*
- Parasitic
  - Ocular larval migrans (*Toxocara, Balisascaris*)
  - Ophthalmomyiasis interna (Cuterebra)
- Viral
  - Feline infectious peritonitis
  - Feline leukemia virus/LSA
  - Feline immunodeficiency virus

Uveitis Workup

- Thorough medical history
  - Weight loss, vomiting, diarrhea, coughing, sneezing, skin lesions
  - Vaccination status
  - FeLV, FIV status
  - Animal environment (indoor/outdoor?)
  - Travel history
- Complete physical examination

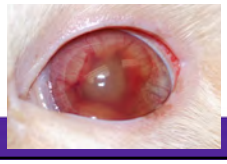
For every uveitis case:

- CBC, chemistry panel
- Urinalysis
- FIV/FeLV snap test
- Infectious disease screening tests**

For select cases, based on PE:

- Thoracic radiographs
- Abdominal radiographs, ultrasound
- Aspirates of any lumps/bumps/cutaneous lesions
- Lymph node aspirates

For blind, painful eyes:
Enucleation *with histopathology*
Diagnostic and therapeutic!



Histoplasmosis

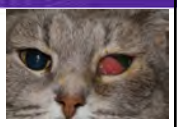
- Dimorphic fungus found in bat and bird feces
- Route of infection: inhalation
- General clinical signs (non-specific):
  - Weight loss
  - Anorexia
  - Fever
  - Anemia

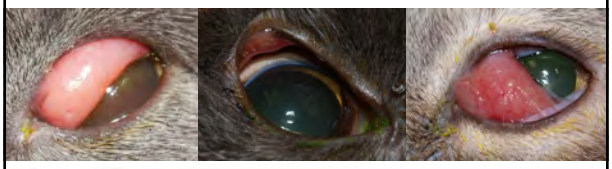
Histoplasmosis

- Ocular clinical signs:
  - Anterior uveitis
  - Chorioretinitis
  - Panuveitis

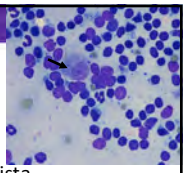
Histoplasmosis

- Ocular clinical signs:
  - Adnexal lesions**





Histoplasmosis



- Diagnosis
  - Cytology or histopathology
  - Antigen testing (serum or urine) – MiraVista
- Treatment
  - Oral fluconazole 10 mg/kg PO q 12 h
  - Minimum 4-6 months
  - Confirm negative antigen test prior to d/c
  - Retest once more ~2 months after stopping antifungal therapy

Cytauxzoonosis

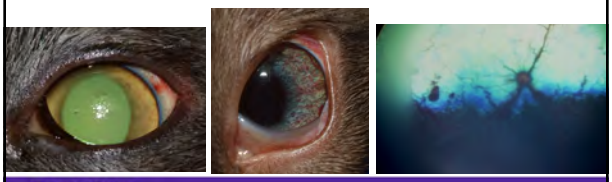
- Hemoprotozoan parasite transmitted by ticks (American dog tick, Lone Star tick)
- Bobcat is reservoir host ('Bobcat fever')
- 2 tissue forms:
  - Piroplasm (blood; erythrocytes)
  - *Schizont (tissue; macrophages)
- Vascular obstruction and organ failure
  - High mortality rate (close to 100%)

Cytauxzoonosis

- General clinical signs:
  - Severe *acute* febrile illness (1-2 weeks after tick bite)
    - Lethargy
    - Anorexia
    - Significant progression in hours to days
      - Vocalizaton, weakness, respiratory distress, icterus, abnormal mentation

Cytauxzoonosis

- Ocular clinical signs:
  - Hemorrhagic panuveitis

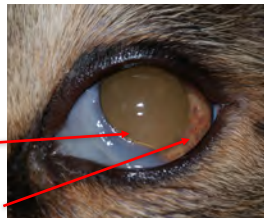


Cytauxzoonosis

- Diagnosis
  - Identification on peripheral blood smear or tissue aspirates, histopathology (necropsy)
- Treatment
  - Antiprotozoal therapy and supportive care
    - Atovaquone 15 mg/kg PO q 8 h *and* azithromycin 10 mg/kg PO q 24 h x 10 days (up to 50-60% survival)
  - Survival rates may improve with new developments

Uveitis Symptomatic Treatment

- Goals of therapy
  - Decrease inflammation
  - Relieve pain
  - Prevent sequelae



'Idiopathic' lymphocytic-plasmacytic uveitis

Uveitis Symptomatic Treatment

- Corticosteroids
  - 1% prednisolone acetate
  - 0.1% dexamethasone or NeoPolyDex
  - NOT hydrocortisone preparations
- Atropine 1%
  - Decreases ciliary m. spasm
  - Dilates pupil to prevent posterior synechiae
  - Stabilizes BAB

Systemic Anti-Inflammatory Therapy

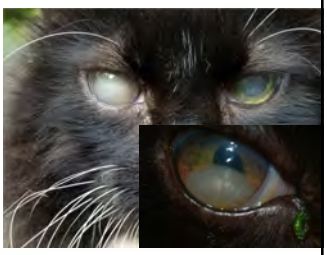
- Steroids
- NSAIDs*
- Treats anterior and posterior uveitis

Other Uveitis Treatments

- Systemic antibiotics
  - Empirical treatment for common bacterial infectious agents
  - Pending test results *or* if workup declined by owner
  - Clindamycin 12.5 mg/kg PO BID x 28d (Toxoplasmosis)
  - Azithromycin 10 mg/kg PO q24 h x 21 d (Bartonellosis)

Uveitis prognosis and sequelae

- *Prognosis dependent on underlying etiology
- Anterior synechia
- Posterior synechia
- Cataract
- Lens luxation
- **Secondary glaucoma**
- Phthisis bulbi
- **Blindness**



Summary

- In cats, uveitis is often an ocular manifestation of systemic disease that requires a thorough diagnostic investigation
  - Idiopathic uveitis is a diagnosis of exclusion
- In addition to being painful and potentially blinding, uveitis can be life-threatening (depending on the underlying cause)
- The most common clinical signs of uveitis should signal the clinician to perform a complete PE and diagnostic testing based on exam abnormalities and individual cat exposure risk(s)
  - *Histoplasma capsulatum* and *Cytauxzoon felis* are two infectious etiologies of feline uveitis that are regionally specific to the Midwestern US

A 'How-To' Guide: A Case-Based Approach to Utilization of Feeding Tubes in Small Animal Patients

Dr. Maria Jugan, Kansas State University

A 'How-To' Guide: A Case-based Approach to Utilization of Feeding Tubes in Small Animal Patients

Case 1: 6-year-old, MC Maltese

History & Physical Examination:
Presented for a 24-history of acute vomiting, diarrhea, and inappetence. It is possible that he got into the trash the night before. He seems otherwise healthy but has a history of mild, similar episodes occurring every few months.

Physical examination:
T: 102.5 F P: 190 bpm R: 42 brpm

Body condition score: 7/9 with normal muscle score
Hydration: 7% dehydrated
EENT: Tacky, pink gums, hypersalivation. Moderate dental tartar
Heart/Lungs: No abnormalities noted.
Abdomen: Tense and painful on cranial palpation. No organ enlargement noted.
Peripheral lymph nodes: Normal
Rectal: Soft, brown fecal material

Diagnostics:
CBC:
Hct 50%, Total protein 8.0
Total leukocytes 20.5 (4.1-15.4), neutrophils 16.4 (3-10.5), band neutrophils 0.3 (0-0)
PLT 350,000

Serum Chemistry:
BUN 35 mg/dL (5-20), Creatinine 1.9 mg/dL (0.6-1.6)
tCa 8.8 mg/dL (9.3-11.6)
ALT 75 IU/L (10-55), AST 60 IU/L (12-40), ALP 130 IU/L (15-120)
t. bilirubin 1.0 mg/dL (0.1-0.4)

Urinalysis:
USG: 1.050
Trace protein
+1 bilirubin

Abdominal ultrasound: Enlarged hypoechoic pancreas with peri-pancreatic hyperechogenicity and small volume anechoic effusion in the region of the pancreas. Mild common bile duct dilation.

Diagnosis: Suspected acute pancreatitis

Clinical progression: Persistent inappetence 48 hours following rehydration and control of vomiting

What are the nutritional concerns in this patient?
Historically, early enteral feeding was considered contraindicated due to the possible risk of increased pancreatic enzyme secretion and iatrogenic worsening of pancreatitis. However, this has not been supported by more recent studies.

Early enteral nutrition has shown benefits in humans, including decreased infectious complications and decreased length of hospitalization. In dogs and cats, early enteral feeding decreases vomiting episodes compared to parenteral nutrition and improves the GI mucosal barrier.

Fat tolerance is a consideration in dogs, as high-fat diets may be a risk factor for pancreatitis in some dogs, especially those with hypertriglyceridemia. A high-fat diet is not a risk factor for cats with pancreatitis, so fat restriction does not need to be considered in the feeding plan. Conversely, many cats are relatively carbohydrate intolerant, and a high carbohydrate feeding plan could worsen GI signs.

Any feeding-associated risks to consider?
This patient is at a relatively low risk for complications from feeding tube placement or from the feeding process itself. General anesthesia (and associated hypotension) could risk worsening pancreatitis but is unnecessary based on the feeding plan of choice (see below).

It is important to note that some liquid human diets have relatively low amino acid concentrations. These diets may not provide sufficient protein sources in cats. It is therefore recommended to use formulated veterinary diets, ensure the human diet chosen contains adequate amino acid concentrations, or supplement essential amino acids if the chosen diet is deficient.

When feeding patients with pancreatitis, it is important to avoid nutrition-induced hyperglycemia. This risk is relatively lower when providing enteral nutrition compared to parenteral nutrition.

Enteral feeding tube choice? Nasogastric feeding tube

Feeding plan:
Placement of an NG tube allows feeding of a liquid diet. A slow, continuous rate infusion (CRI), at 25-30% of resting energy requirement (RER), can be initiated on the first day. As long as feeding is tolerated, caloric intake can be increased by 25-30% per day until full RER is achieved. Feeding can be facilitated with anti-emetics and pro-kinetics, as needed.

Voluntary food intake is encouraged and can be offered every 4-6 hours. It is recommended to remove any uneaten canned food after 15-20 minutes to avoid the development of food aversion. These general guidelines apply to any inappetent hospitalized patient.

Once full RER is achieved, one can consider switching to a “bolus-feeding” approach. This divides the caloric allotment into several infusions of 3-4 hours’ duration, with a 2-hour period during which the CRI is paused. The patient is then offered food for voluntary intake after the 2-hour break in feeding. This allows time during which the stomach is empty, providing additional stimulus for voluntary intake.

Suggested nutrient profiles include low-moderate fat diets in dogs (2.5 - 5 g/100 kcal fat) and moderate-high protein diets in cats (9 - 12 g/100 kcal protein).

Case 2: 10-year-old, FS Domestic Shorthair

History & Physical Examination: Presented for a several week history of hyporexia, progressing to a one-week history of anorexia. Owners appreciated substantial weight loss over several weeks.

Physical examination:
T: 99.5 F P: 180 brpm R: 30 brpm

Body condition score: 7/9
Hydration: 8% dehydrated
EENT: Icteric sclera, gums, pinnae. Gums slightly tacky. Moderate dental tartar.
Heart/Lungs: No murmurs, arrhythmias, or pulse deficits. Fair pulses. Lung auscultation normal in all fields.
Abdomen: Hepatomegaly
Peripheral lymph nodes: Normal
Musculoskeletal: Weak; Moderate, diffuse muscle atrophy.
Neuro: Depressed to dull, responsive.

Diagnostics:
Blood glucose: 50 mg/dL

Blood pressure: 100 mmHg systolic (Doppler)

CBC
Hct 19%, Plasma Protein 6.4
Total leukocytes 10.7 (4.1-19.1), neutrophils 9.8 (3.0-8.1), lymphocytes 0.3 (1.0-4.7)
PLT 282,000

Serum Chemistry
BUN 7 mg/dL (15-31), Creatinine 0.3 mg/dL (0.6-1.6)
Phosphorus 1.7 mg/dL (3.2-8.1), tCalcium 8.2 mg/dL (9.3-11.6)
Na 141 mEq/L (143-153), K 3.6 mEq/L (4.2-5.4), Cl 106 mEq/L (109-120)
ALT 216 IU/L (10-55), ALP 280 IU/L (15-120), GGT 4 IU/L (2-8)
t. bilirubin 4.4 mg/dL (0.1-0.4)
Cholesterol 120 mg/dL (80-315)
Total protein 5.1 g/dL (5.1-7.1), albumin 1.7 g/dL (2.9-4.2), globulin 3.4 g/dL (2.2-2.9)
Glucose 80 mg/dL (77-126)

Point-of-Care (venous): iCa 4.8 mg/dL (4.55-5.77)

Abdominal ultrasound: Diffuse hepatomegaly and hyperechogenicity.

Liver aspirate/cytology: Clusters of hepatocytes with abundant, discrete vacuolation, resulting in cellular distention. Moderate amount of green pigment within hepatocytes and lining cell borders, consistent with moderate to severe hepatic lipidosis and cholestasis.

Diagnosis: Hepatic lipidosis (HL) with concern for hepatic insufficiency

What are the nutritional concerns in this patient?
It is important to provide adequate nutrition to minimize additional hepatic lipid accumulation and ultimately, allow for hepatic regeneration. Most models show that providing 25% RER is sufficient to minimize additional lipid accumulation; though, higher energy is required for regeneration.

Many cats with HL also have essential amino acid (e.g. taurine and arginine) and vitamin (e.g. cobalamin) deficiencies that require correction with nutrition. In severe cases of HL, these may need supplemented in addition to the chosen diet.

Most cats with HL require prolonged nutritional support, on the average of 3-6 weeks, and providing supplemental nutrition (i.e. feeding tube placement) is one of the few positive prognostic indicators for survival in cats with HL.

Any feeding-associated risks to consider?
As presented, this cat would be a high risk anesthetic candidate. It would benefit from improved hemodynamic stability prior to anesthesia and correction/stabilization of electrolytes prior to initiating enteral nutrition. The presence of vitamin K deficiency in many cats with HL can increase the risk of bleeding with surgical procedures, including esophagostomy tube (e-tube) placement.

It should be considered that prolonged anorexia results in GI stasis and decreased gastric capacity, which may decrease tolerance to food. Prolonged anorexia also increases the risk of refeeding syndrome, which predominantly manifests as hypokalemia and hypophosphatemia. If the dull mentation does not improve with supportive care, there is also clinical concern for hepatic encephalopathy, which can be exacerbated by feeding and may warrant different diet choices.

Enteral feeding tube choice?
Ideally, this cat would be initially stabilized with intravenous fluids and electrolyte/dextrose supplementation, as needed. An NG tube can then be placed, usually within the first 24-48 hours. NG tube placement allows initial provision of nutrients, along with continued hemodynamic, neurologic, and biochemical stabilization. Once stabilized, placement of an e-tube under general anesthesia would be optimal and allows for continued nutritional support following discharge.

Plan:
A suggested nutrient profile for cats with HL would include about 30-40% energy from high-quality protein, 50% energy from fat, and 20% energy from carbohydrates, whether a liquid NG-tube diet or canned/dry diet formulated for e-tube administration is used. Many recovery or “diabetic” feline diets fit this profile. Mild protein restriction may be needed in cats with clinical hepatic encephalopathy; however, the general rule is to supply as much protein as is tolerated, without exacerbating neurologic signs.

Initial feeding amounts are usually lower in cats with hepatic HL, with provision of 15-25% RER on the first day. Caloric intake can be increased 10-25% daily, depending on refeeding complications and GI tolerance. If complications are noted, meal size is decreased 25-50% for 12 hours. The risk of complications from refeeding or hepatic encephalopathy are greatest within the first 12-72 hours of initiating feeding. Depending on patient severity, electrolytes, including phosphorus, are re-evaluated 4-8 hours after initial feeding and PCV/TS 12-24 hours after initial feeding. Frequency is adjusted based on blood work stability. Care should be taken to minimize total volume of blood drawn to decrease the risk of iatrogenic anemia.

The CRI feeding method, as described above, is utilized when the NG tube is in place. Once transitioned to an e-tube, feedings are performed 3-4 times daily, with at least 3 hours between feedings. Food

tolerance can be aided using anti-emetics and pro-kinetics. It can take 3-7 days to achieve 100% RER in cats with HL.

Supplements to consider: Vitamin K_1 is administered *subcutaneously* 0, 12, and 24 hours prior to placement of an e-tube. Cobalamin and a glutathione donor (e.g. N-acetylcysteine or S-adenosylmethionine) are recommended in addition to diet therapy in cats with HL.

The following supplements are not needed in most cats when provided a balanced veterinary diet but can be considered in specific cases:

- Thiamine: Supplement if clinical (e.g. cervical ventroflexion, central blindness)
- L-carnitine: Consider in severe cases
- Taurine: Consider in severe cases
- Arginine: Consider if encephalopathic

Case 3: 4-year-old, FS Yorkshire terrier

History & Physical Examination:
Presented for a 1-year history of diarrhea, characterized by large-volume defecations 2-3 times per day, with a cow-pie like consistency to the stool. The owners have not seen any blood or mucus, and she does not vomit. There is documented gradual weight loss over the last year. She has developed progressive hyporexia over the last month and developed abdominal distention within the last week.

She receives routine flea-tick-heartworm prevention and has already received two courses of fenbendazole for prophylactic deworming.

Physical examination:
T: 100.9 F P: 110 bpm R: 28 brpm

Body condition score: 3/9 with moderate, diffuse muscle atrophy
Hydration: Adequate
EENT: Mucous membranes pink and moist, capillary refill time <2 seconds; Mild dental tartar.
Heart/Lungs: No murmurs, arrhythmias, pulse deficits. Heart and lung sounds slightly decreased
Abdomen: Moderately distended with fluid wave.
Peripheral lymph nodes: Normal.
Rectal: Liquid, brown fecal material

Diagnostics:
CBC:
Hct 45%, Plasma protein 3.1
Total leukocytes 18.0 (4.1-15.4), neutrophils 15.4 (3-10.5), lymphocytes 0.8 (1-4.7)
PLT 570,000

Serum chemistry
tCalcium 5.2 mg/dL (9.3-11.6)
Na 138 mEg/L (143-153), K 3.9 mEq/L (4.2-5.4), Cl 110 mEq/L (109-120)
Cholesterol 44 mg/dL (80-315)
Total protein 2.6 g/dL (5.1-7.1), albumin 1.5 g/dL (2.9-4.2), globulin 1.1 g/dL (2.2-2.9)

NOVA/Point-of-Care (venous): iCa 3.2 mg/dL (4.55-5.77)

Urinalysis: USG 1.035; Protein: Negative

Fecal flotation: Negative

Baseline cortisol: 6 ug/dL

Calcitriol: 56 pmol/L (RR 164-523)

GI biopsies: Lacteal dilation and moderate lymphoplasmacytic inflammation in the ileum and duodenum; presence of crypt abscesses

Diagnosis: Protein-losing enteropathy (lymphangiectasia)

What are the nutritional concerns in this patient?
For patients with lymphangiectasia, considerations for diet-of-choice relate to both the cause and effects of the disease. Excess GI fat loss, results in secondary GI inflammation. Therefore, most nutrition plans implement a fat-restricted diet. In patients with primary lymphangiectasia, this may be sufficient. However, in patients with lymphangiectasia secondary to severe inflammatory bowel disease (IBD), there is consideration for dietary antigenic intolerance. These patients may benefit from diets that are both fat restricted, as well as either novel protein or hydrolyzed.

Patients with PLE often have electrolyte and vitamin deficiencies due to malabsorption, including calcium, magnesium, cobalamin, and vitamin D. These patients are at risk for protein malnutrition due to their hypermetabolic state in combination with GI protein loss.

Many patients also require prolonged nutritional support. Goals of supplemental enteral nutrition include treating the primary disease process, replenishing proteins and restoring oncotic pressure, and maintaining and restoring lean muscle mass.

Any feeding-associated risks to consider?
There is a moderate risk of general anesthesia in patients with severe PLE, as in the case described above. Ventilation can be challenging due to the presence of pleural and peritoneal effusion, and therapeutic thoracocentesis/abdominocentesis may aid ventilatory capacity.

Theoretically, hypoalbuminemia and general protein malnutrition could impede e-tube stoma site healing, but clinically this is considered a low risk.

Enteral feeding tube choice? Esophagostomy feeding tube

Feeding plan:
A suggested nutrient profile for patients with lymphangiectasia would include a low-fat diet (≤2 g/100 Kcal in dogs), as an up to 80% response rate is noted in dogs with primary lymphangiectasia. These can be either commercial veterinary diets or home-cooked diets.

Temporarily, home-cooked diets can be used, with protein and carbohydrate sources including potato and whitefish, low-fat turkey, low fat cottage cheese, and cooked egg whites. These are typically not administered through a feeding tube and are not balanced long-term. If an owner wishes to feed a home-cooked diet for this disease condition, consultation with a veterinary nutritionist is recommended.

In addition to commercial low-fat diets, a fat-modified hydrolyzed or novel protein diet for patients with secondary PLE (e.g. severe IBD) may improve response.

ADDITIONAL RESOURCES

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Mitral Valve Disease in Dogs

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Mitral Valve Disease in Dogs

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Introduction

Myxomatous Mitral Valve Disease (MMVD), also know as Degenerative Mitral Valve Disease (DMVD) or just Chronic Valvular Disease, is the most frequent heart disease in the dog, and the most common cause for congestive heart failure (CHF). Mitral valve disease can also occur as a congenital defect in dogs, and mitral regurgitation can happen secondary to other diseases as well, such as dilated cardiomyopathy or bacterial endocarditis. For MMVD, the stage of disease has implications for drug and diet therapy, anesthesia, fluid administration, and for prognosis.

Overall Incidence

The incidence of MMVD is reported as being between 11% (clinical determination) and 42% (necropsy determination) depending on the age of the dog and method of examination. The mitral valve is most commonly affected, and many dogs have disease in both the mitral and tricuspid valves. The aortic and pulmonic valves may be affected by degenerative changes, but clinical disease in small animals is unlikely. The disease is primarily a non-inflammatory, myxomatous degeneration of the atrioventricular valves that is commonly referred to as endocardiosis. Congenital mitral valve dysplasia, conversely, is infrequent but can cause CHF in some large breed dogs very early in life, including Weimaraner, German Shepherd dogs, Pit Bull dogs, and other medium to large breed dogs. Mitral valve endocarditis is also uncommon and is typically accompanied by fever or other signs of systemic illness

Signalment

MMVD is most common in small to medium sized breeds of dogs, and the incidence of CHF is increased in male dogs relative to females (1.5 to 1.0). MMVD is typically slowly progressive with lesions sometimes beginning early in life (e.g., by 2 to 3 years microscopically), but overt clinical disease is unlikely before middle age. Cardiac decompensation and CHF typically occurs in later life (e.g., 5 to 14 years of age or older). Up to 90% of Cavalier King Charles spaniels can be affected, and they get disease earlier in life than other dog breeds. MMVD is common in miniature poodles, Dachshunds, Shi Tzu dogs, and many other small breed dogs. The disease is less common in large breed dogs but can be seen in German shepherd dogs, Doberman pinschers, Bull Terriers, and other breeds.

Etiology

The etiology for MMVD is unknown. A genetic tendency to develop the disease has been proved in the Cavalier King Charles Spaniel. Serotonin levels may be elevated in some affected dogs or at certain stages of the disease. The exact interplay of genetics, diet, exercise, or other risk factors is currently uncertain.

Clinical Syndromes

A wide range of clinical presentations are possible for dogs MMVD. Many dogs will be presented for routine examination and a cardiac murmur, mid-systolic click, or arrhythmia will be noted. Baseline testing can be offered to the owner and is often helpful for comparison at subsequent examinations or for determination of whether early therapy is indicated. In dogs with a III/VI or louder murmur, therapy to delay the onset of CHF might be available (pimobendan) and baseline thoracic radiographs are recommended. If the VHS is > 10.5 then echocardiography is recommended. Alternatively, signs resulting from congestive heart failure may be the cause for presentation with intermittent cough, nocturnal dyspnea or altered sleep habits, abdominal distention and exercise intolerance being presenting complaints. Syncope or collapse may be the trigger for a veterinary exam, and collapse is common in dogs at the first onset of CHF, most likely due to a vagally-mediated event. Variations on these presentations can be a result of cardiac arrhythmias, ruptured chordae tendinae, or left atrial splitting leading to cardiac tamponade.

Physical examination

Auscultation of a cardiac murmur is a classic finding in MMVD, and an extra systolic sound known as a mid-systolic "click" has been associated with early disease, due to mitral or tricuspid valve prolapse. A third heart sound, typically an S3 gallop, can develop at the time of CHF. In dogs with MMVD there is a tendency for the intensity of the murmur to be roughly correlated with the severity of cardiac dysfunction, and most dogs with CHF have at least a IV/VI systolic murmur over the left cardiac apex.

Dogs with CHF may have tachypnea, dyspnea, orthopnea, or anxiety with a reluctance to lie down. A cough may be noted or elicited by tracheal palpation. Cough may end in swallowing or the production of a white foam; in advanced cases with severe pulmonary edema, a pink or blood-tinged froth is produced. Mucous membranes are pink in early stages of CHF but may progress to a muddy to somewhat cyanotic color with severe pulmonary edema due to left-sided CHF. Pulmonary

auscultation may also reveal increased respiratory sounds which progress to "crackles" with the onset of alveolar edema. Hepatomegaly and ascites may be evident in dogs with RCHF from advanced disease and/or significant tricuspid regurgitation, and in these cases the jugular veins are typically distended. The femoral pulses are often normal in dogs with MMVD, unless severe decompensation has developed. Cardiac arrhythmias, when noted, are more likely to be supraventricular premature depolarizations, although other arrhythmias are possible. When ventricular arrhythmias are noted the disease is often advanced and these dogs often do not do as well; some of them have an endocardial split with a thrombus in the left atrium.

Thoracic Radiography

Cardiomegaly, especially left atrial enlargement, is identifiable in most dogs with MMVD. On the lateral projection, the left atrium is visualized at the caudal-dorsal aspect of the cardiac silhouette and can be seen to enlarge with disease progression. On the DV view the LA bulge is noted at the 2:00 to 3:00 location. Left atrial and left ventricular enlargement result in elevation of the trachea and carina. The left mainstem bronchus may become elevated (compressed) in cases of marked left atrial enlargement and cause cough related to airway compression. Pulmonary venous dilation occurs as dogs get closer to CHF, but dogs with acute rupture of a chordae tendinae and severe CHF might not have as much atrial enlargement and may lack pulmonary vein distention. Early pulmonary edema is seen as a diffuse increase in interstitial density in the hilar or caudal lung fields, progressing to fluffy densities and air bronchograms (alveolar pattern) with the onset of alveolar edema. Ascites, hepatomegaly +/- splenomegaly may be present in dogs with concurrent tricuspid valve disease.

Studies that have followed dogs longitudinally have identified that dogs with a VHS of > 11.5v are at increased risk for development of CHF in the next 6 to 12 months.

Cardiac Biomarkers – BNP and troponin testing

Recent studies have documented that BNP testing can help guide treatment decisions. Both c-BNP and NT-proBNP tests are available, but there is more research to guide interpretation of NT-proBNP. Dogs with MMVD that have an NT-proBNP < 1500 pmol/L have a lower chance of developing CHF in the next 6 to 12 months (maybe 10-20% chance for CHF), while dogs with an NT-proBNP concentration > 1500 to 1600 pmol/L might have a 70% or greater chance to develop CHF in the next 6 to 12 months. The presence of an NT-proBNP > 1500, plus a VHS > 11.5, plus a markedly enlarged left atrium on echocardiography helps to more specifically identify dogs at high risk for CHF, and these 3 tests can be seen as complimentary (for the dedicated owner who is not limited financially).

Dogs with MMVD and CHF usually have an NT-proBNP > 2,000 to 3,000 pmol/L, and most dogs with active CHF have a value > 3000 pmol/L. Identification of a normal range NT-proBNP should result in reconsideration of a diagnosis of CHF – it might be CHF, but it is more likely that the dog has clinical signs due to something other than CHF.

Dogs with MMVD can have elevated cardiac troponin I concentration, and there is a chance that more significantly elevated values might be associated with a somewhat worse outcome.

Electrocardiography

ECG findings in dogs with MMVD can include evidence of left ventricular hypertrophy and left atrial enlargement (P mitrale). ST segment slurring is seen in some dogs with LV hypertrophy, and ST depression may result from hypoxemia. Sinus rhythm or sinus tachycardia are typical, although many dogs have atrial premature depolarizations. Ventricular arrhythmias are uncommon in dogs with MMVD and when seen might trigger concerns for a worse prognosis, a concurrent disease, or recent myocardial insult (maybe get a cardiac troponin I concentration). Atrial fibrillation develops in some dogs, especially those with marked left atrial enlargement.

Echocardiography

Diffuse valvular thickening may be appreciated, particularly of the "anterior" mitral leaflet, in dogs with MMVD, and ruptured chordae tendineae may be seen as the valve tip prolapses into the LA during systole. As the quality of images from echo machines has gotten better and better, more and more dogs with MMVD are identified to have a least a small chordal rupture, even in the still compensated stages of the disease. Left atrial enlargement is often severe in dogs with MMVD and CHF. Fractional shortening is normal to exuberant in dogs with earlier stage MMVD, but the contractile function on echo can drop off in the latter stages of the disease. Mitral regurgitation can be clearly identified on color-flow Doppler. Tricuspid valve prolapse and regurgitation are also commonly seen. Pulmonary hypertension, evidenced by an increased tricuspid regurgitation velocity, often develops as CHF advances, and this can be a target for therapy (sildenafil). The E wave from mitral inflow is often increased (> 1.25 and often close to 1.5 m/sec), as is the E:E' (which is often above 11-14; some clinicians prefer the E:IVRT). Dogs with CHF should have at least moderate left atrial enlargement, unless CHF is due to an acute ruptured chordae tendinae. Endocardial splitting can develop and may result in pericardial effusion and cardiac tamponade, or the split may go through the interatrial septum and result in a left-to-right ASD and often signs of R-CHF. Some dogs with endocardial splitting can have a thrombus seen in the left atrium, and if pericardial effusion has developed then a thrombus may be noted in the pericardial space. Pleural effusion may develop in dogs with biventricular heart failure.

Treatment of dogs with MMVD

Treatment of MMVD can be divided into several stages of CHF or clinical scenarios. Below is an attempt to summarize these stages and clinical scenarios.

Stage A – The dog without a murmur that is at risk for MMVD

Except for dogs used for breeding, there is no need to identify these dogs. It is prudent to have these dogs eat a balanced diet and to maintain a normal body condition score. Certain screening protocols and breeding recommendations have been proposed for dogs actively used in a breeding population (e.g., Cavalier King Charles Spaniel).

Stage B – The asymptomatic dog with a murmur due to MMVD

Dogs with a I/VI or a II/VI murmur usually require no additional testing.

Dogs with a III/VI or louder murmur might be candidate for pimobendan administration. Since pimobendan, in the EPIC trial, resulted in a significant delay until the onset of CHF (perhaps by an average of 15 months) and prolonged overall survival time, it seems appropriate to start dogs on pimobendan if they fulfill the 4 key criteria from the EPIC trial. These 4 criteria are 1) at least a III/VI murmur; 2) VHS > 10.5v; 3) LA/Ao > 1.6 on a short axis echocardiographic view; and 4) a normalized left ventricular end diastolic dimension (LVIDd) of > 1.7 via echocardiography. Thus, to determine whether pimobendan is clearly indicated in the asymptomatic dog with MMVD, one needs a physical exam, and thoracic radiographs, and an echocardiogram -- these are the criteria we typically use to determine eligibility for starting pimobendan in our clinic. As some owners cannot afford echocardiography, some researchers have suggested that a VHS of > 11.5v might identify the population of dogs that would have fulfilled these criteria. Others have suggested that since an NT-proBNP > 1500 pmol/L identifies dogs at increased risk for developing CHF within a year, the presence of VHS > 11.5v or an NT-proBNP > 1500 pmol/L might also be triggers for pimobendan administration.

The role for ACE inhibitors (e.g., enalapril, benazepril, lisinopril) or angiotensin receptor blockers in management of asymptomatic dogs with MMVD is more hotly debated, and perhaps the role is less clear-cut. In our clinics, we try to obtain blood pressure measures on dogs with MMVD and if a dog is persistently hypertension (above 160 mmHg) then we start an ACEi. We also might look for concurrent proteinuria, as this is another indication for giving an ACEi, and if proteinuria is documented in a dog with MMVD then we start the ACEi. Finally, for dogs already on pimobendan who have marked LA enlargement, where CHF seems imminent, we also will start an ACEi. If we elect to start an ACEi then we obtain baseline kidney values and electrolytes, we repeat these values in 10-14 days, and the recheck them q 6 months until the onset of CHF.

Some would recommend dietary sodium restriction, and at least having the owner paying attention to treats fed to the dog for sodium content. Maintenance of a normal BCS and MCS is recommended at this stage, as well as feeding a complete and balanced diet. Rechecks are performed every 6 to 12 months for examination +/- thoracic radiographs +/- echocardiography +/- NT-proBNP +/- blood pressure measurement.

Stage C and D – The dog with MMVD and CHF

The ACVIM consensus statement recommends that a certain number of diagnostic tests be done to confirm a diagnosis of CHF. The tests we typically perform in the client who is not financially limited include thoracic radiographs, echocardiography, an ECG (especially if arrhythmia), a CBC and serum biochemistry profile, a blood pressure, and an NT-proBNP. Performing all tests allows for baseline data to be collected before starting cardiac medications that might alter kidney function or electrolytes and minimizes the chance for misdiagnosis.

Drug therapy at the onset of CHF usually includes furosemide (or torsemide), an ACE inhibitor such as enalapril, benazepril or lisinopril, and pimobendan. Many clinicians will also add in spironolactone, and the current ACVIM consensus statement includes a recommendation to use all 4 of these drugs once CHF is diagnosed. Once dogs become refractory to this combination of medications then additional manipulations include optimizing doses of current medications, going to higher (off label) doses of pimobendan, adding in sildenafil if pulmonary hypertension is moderate to severe, adding in more diuretics (e.g., torsemide, injectable furosemide, hydrochlorothiazide), adding in amlodipine or hydralazine, or other medications.

Dietary sodium moderation is usually recommended at this stage, with attention paid to all dietary sources of sodium – severe sodium restriction might not be needed. We usually also recommend either a diet that is high in omega-3 fatty acids or fish oil supplementation.

The following list of drugs that follows includes some additional information relative to use and dosing. For additional information on diets or heart disease visit: https://heartsmart.vet.tufts.edu/

Furosemide

Diuretics are indicated for animals with CHF, and furosemide is the most commonly used diuretic in dogs and cats. In most cases, diuretics should be combined with either pimobendan, or an ACE inhibitor, or both. Many cardiologists would not recommend single agent use of furosemide for treatment of CHF. The “correct” dose of diuretic required by any individual

dog with CHF is difficult to define, although many veterinary cardiologists propose and initial dose of 2 mg/kg once or twice a day. The ideal dose is the amount of diuretic required to clear significant edema accumulations and cause the animal to be minimally symptomatic. Breathing rate and effort are is close to normal (less than 32 to 35 breaths per minute), ascites is controlled, normal hydration is maintained, plasma electrolytes remain normal, and renal values remain in the normal range or are minimally elevated. Unfortunately, this ideal dose is often close to a dose that might result in electrolyte disturbance, dehydration or azotemia. It is recommended to measure renal function prior to starting therapy and then repeat the BUN, creatinine and electrolytes 5 to 10 days after starting drugs to treat congestive heart failure.

We currently try to find the "Lowest dose of furosemide that controls clinical signs" in animals with CHF. We almost always stay on some dose of furosemide or another diuretic. We have seen too many animals where the diuretics were stopped and the animals returned for another emergency visit for active CHF, and many owners cannot afford this hospitalization and elect to euthanize their pet, so we almost never have a long term goal of "try to stop the diuretics" in an animal who has been clearly documented to have CHF.

Finding the ideal dose of diuretics often involves a degree of experimentation to find the right dose, and the dose on any given day might fluctuate if the dog eats a high sodium meal or engages in a lot of activity. Giving an owner upper and lower limits for acceptable furosemide dose, and carefully explaining to them that they should "give more for difficulty breathing or rapid respirations, and give less if the animal seems weak, lethargic, anorexic, or depressed" has worked successfully for the author. In most instances, canine patients are given 2.2 mg/kg q 12 hours, or less, for initial chronic therapy. We ask owners to measure respiratory rate and to pay attention to the amount of abdominal effort during breathing. If the breathing rate rises above 32 to 35 breaths per minute, especially if accompanied by additional abdominal effort to breathing, the owner is instructed that they have a clear recommendation to "give an extra dose of diuretics" for these findings.

When a furosemide dose is rising, and congestion is still present then we usually add in additional cardiac medications. We suspect that diuretic resistance has developed, and more furosemide might not be as helpful as adding in other diuretics or other cardiac medications such as spironolactone, amlodipine, sildenafil, or higher doses of pimobendan. Some clinicians might stop the furosemide and add in torsemide in an equivalent dosing approach, but our strategy is the maintain the same dose of furosemide and start to add in torsemide as another diuretic. Thus, many of our dogs with advanced CHF are on > 4.4 mg/kg/day of furosemide, plus spironolactone, plus torsemide, plus an ACE inhibitor and pimobendan and maybe other medications. In some dogs with advanced CHF, use of injectable furosemide once a day (e.g., add in a mid-day subcutaneous injectable dose to the BID oral dose) can restore a diuresis.

Spironolactone

Multiple well-designed clinical trials performed on people with CHF have documented improved outcome for individuals treated with aldosterone receptor blocking drugs, and at least 1 veterinary clinical trial has also hinted at an improved outcome. As a result, spironolactone was added to the list of drugs recommended for management of Stage C heart failure in the most recent ACVIM Consensus Guidelines for mitral valve disease in dogs. The actions of aldosterone receptor antagonist drugs like spironolactone extend beyond the effects as a mild diuretic, and the actions of blocking aldosterone receptors in the myocardium and throughout the body may be part of the reason for the improved outcomes in clinical trials. Some veterinary cardiologists start spironolactone very early, in a pre-clinical stage, in the hopes that the aldosterone blocking actions might prevent fibrosis in the myocardium or that this class of drugs might have other positive benefits. Other veterinary cardiologists view the evidence of spironolactone as a bit weaker and argue that drug-drug interactions increase, and client compliance decreases, as you use more than 3 medications – these individuals might start spironolactone just a bit later in the disease process.

Torsemide

Torsemide is a loop diuretic with some aldosterone receptor antagonist action. The drug was recently approved in areas of Europe for use in management of CHF in dogs. One potential advantage of torsemide is the long duration of action compared to furosemide, so it might be possible to dose the drug less frequently, and this could be an advantage for some owners. In the US, some clinicians are using torsemide as a first line drug for management of CHF, but more clinicians are still starting with furosemide and then adding in (or switching to) torsemide when the animal is refractory to management with furosemide plus ACEi and pimobendan. Currently, we tend to use torsemide in dogs with advanced CHF. Some veterinary cardiologists substitute torsemide for furosemide as CHF progresses, trying to give an equivalent dose of torsemide and taking away the furosemide. We often will continue the furosemide but start to add in incremental doses of torsemide, so we have dogs with advanced CHF on furosemide, torsemide and spironolactone all at the same time. Torsemide is usually dosed at approximately 1/10th to 1/20th of the dose of furosemide that you might give in a similar situation (e.g., if you would have given 50 mg of furosemide, give 2.5 to 5 mg of torsemide). The proposed dose range is 0.1 to 0.5 mg/kg q 12-24 hours in dogs; typically, we start q 24 hours at 0.1 to 0.2 mg/kg when we add it into ongoing furosemide treatment. Possible side effects are similar to other diuretics and include dehydration, azotemia and electrolyte depletion. Serial measures of renal values and electrolytes is recommended.

Pimobendan

Pimobendan is a calcium sensitizing drug that is useful as a positive inotrope in addition to having properties as a phosphodiesterase inhibitor with vasodilating effects. In most veterinary studies, pimobendan treated dogs have fared as well or better than dogs treated with ACE inhibitors. Pimobendan also seems to be associated with a low side effect profile. Gastrointestinal side effects can be seen, perhaps arrhythmia in some dogs, and excitability in cats can be dose limiting, but the side effects are infrequently associated with a major negative impact on the animal's well-being. The drug has been studied in dogs with active CHF, as well as in dogs with pre-clinical heart disease. The usual dose for pimobendan is 0.25 to 0.3 mg/kg q 12 hours. Pimobendan is usually avoided in animals with outflow tract obstruction.

Pimobendan is typically used in one of 3 clinical settings in dogs: 1) Dogs with documented CHF that is not due to pericardial disease or obstruction to cardiac outflow 2) Dogs with occult DCM who have documented cardiomegaly and reduced LV contractile function and 3) Dogs with asymptomatic MMVD (Stage B2) who fulfill all 4 of the EPIC trial inclusion criteria.

Pimobendan usually does not require any specific monitoring to search for side effects of the drug. Sometimes, the dogs who had pimobendan (or pimobendan plus ACE inhibitors) administered before stage C will be more difficult to treat once CHF with pulmonary edema is present. However, the studies suggest that the overall survival time (pre-clinical time plus time with clinical CHF) will be longer if pimobendan is used early, and that the duration of time with lower quality of life during active CHF will be shorter. Pimobendan is usually given in the acute management of severe pulmonary edema, and often we administer it 3 times a day until CHF is controlled; some areas of the world have injectable pimobendan for use in the emergency setting. In advanced CHF that is refractory to standard therapy, pimobendan dose can be escalated, and we sometimes go as high as 0.6 mg/kg q 8 hours.

ACE inhibitors

Angiotensin-converting enzyme (ACE) inhibitors are commonly used in the management of CHF. Their use is predicated on the knowledge that interfering with the activation of the renin angiotensin system leads to diminished plasma levels of angiotensin II and reduced stimulation of aldosterone. As a result, fluid retention and vasoconstriction are blunted. The beneficial effects of ACE inhibition likely result from both the vasodilation and the drug's effects to reduce cardiac remodeling. Angiotensin-converting enzyme inhibitors have proved to be useful in a variety of settings. In well-designed canine heart failure trials, ACE inhibitors resulted in improved clinical signs and often a prolonged the time until an animal dropped out of the study (equivalent to improved survival). ACE inhibitors are typically used in dogs with congestive heart failure. The role of ACE inhibitors in the treatment of animals with asymptomatic MMVD is debated, although there might be slightly greater enthusiasm for their use in dogs with occult DCM. In dogs with mitral regurgitation and concurrent systemic hypertension, as well as in dogs with mitral regurgitation and concurrent significant proteinuria, administration of an ACE inhibitor is currently recommended by the author. The author also adds ACE inhibitors to the treatment of dogs with MMVD when cardiomegaly progresses despite administration of pimobendan. Twice a day therapy is recommended.

Sildenafil

Sildenafil inhibits phosphodiesterase type-5 (PDE5). PDE5 is found in high concentration in the smooth muscle of the pulmonary vasculature, so the intended cardiovascular result is pulmonary vasodilation and reduced pulmonary hypertension. The main indication for sildenafil is pulmonary hypertension, which is most reliably demonstrated based on tricuspid regurgitation velocity (and use of the modified Bernoulli equation $4 \times V^2$). The drug may be most useful when clinical signs result from pulmonary hypertension (collapse, shortness of breath when lung fields are clear, refractory ascites with biventricular failure) or when the estimated PA systolic pressure is at least moderate to severely elevated (above 50-60 mmHg). A starting dose of 1 mg/kg q 8 hours is titrated up based on clinical response, up to a dose of 3 mg/kg q 8 hours. Possible side effects include systemic hypotension and weakness – avoid with concurrent nitrate use (e.g., stop sildenafil if you are going to administer nitroglycerin or sodium nitroprusside). Tadalafil has also been used in these clinical settings.

Sodium Nitroprusside

Dogs with severe pulmonary edema that is unresponsive to 1 to 3 doses of furosemide at 4 mg/kg can be very difficult to manage successfully. The author has had some success with dobutamine; however, the drug that most reliably controls life-threatening pulmonary edema for me is sodium nitroprusside. A continuous rate infusion is required in order to give sodium nitroprusside, but this drug can be very effective in this setting. Measurement of blood pressure is important however the author has become more permissive about how low a blood pressure can be tolerated for 4 to 12 hours in order to control severe CHF. Many dogs and cats with severe pulmonary edema and a systolic blood pressure of 70 mmHg can still tolerate an infusion of sodium nitroprusside for several hours without apparent long-term renal damage. Close observation of the animal, skilled technicians, and frequent re-evaluation of the animal's condition are needed to find an effective dose. Doses ranging between 2 and 10 mcg/kg/min are often successful in controlling edema in dogs. We usually start at 2 mcg/kg/min in dogs and if the

dog is not clinically improved in 1 hour then increase to 4 mcg/kg/min. Clinical improvement is usually noted as relief of dyspnea, slower respiratory rate, very pink to injected membrane color, and improved attitude. The drug is usually administered for 12 to 48 hours until severe edema is resolved, and other cardiac medications can be added into the drug regimen. It is important to continue to give generous doses of diuretics during nitroprusside administration, even when clinical signs of improvement have been noted, or signs of CHF will often return when the drug is titrated down or discontinued.

Dietary Modifications and Diet-Associated Cardiomyopathy

A variety of diets are reduced in sodium; some have more specific modifications which are desirable for heart disease. In general, moderate dietary sodium intake is recommended for heart disease or CHF. Protein restriction should be avoided as this can contribute to cardiac cachexia and muscle loss, and dogs with muscle loss have been shown to have worse outcomes with CHF. Many renal diets have inadequate protein, even though they might be sodium restricted, so we usually avoid kidney diets unless there is concurrent significant azotemia. Diet acceptance by the dog can be challenging if a sudden dietary switch is made when CHF is active or new drugs which affect appetite are being introduced, so we usually get the owner to start to slowly change the diet after the cardiac drugs have been started and are being well tolerated, with good appetite, for at least 4 to 7 days. For access to diet handouts and other helpful information for owners of pets with heart disease, see: https://heartsmart.vet.tufts.edu/

Diet associated cardiomyopathy has been increasingly recognized in dogs consuming certain diets. Many of these diets are noted to be "grain free" or to have peas or lentils as one of the top 5 to 10 ingredients. Some of these diets also have unusual protein sources. We have seen some of these dogs have improvement in their cardiac function after switching to other diets. In general, the improvement has been more remarkable in some dogs than others, and our observation has been that the dogs with the worst heart failure might not have as much improvement as dogs with earlier disease or milder CHF. We have also seen some dogs with myxomatous mitral valve disease and concurrent myocardial failure have some clinical improvement and improvement in myocardial function after switching to a different diet. Most of the dogs we see with diet-associated cardiomyopathy are not taurine deficient, so we do not think that supplementing with taurine without changing the diet is a good strategy. Hopefully more information will be available in the future about the relationship between these diets and DCM in dogs. Additional information on diet-associated cardiomyopathy can be found at the Petfoodology web site https://vetnutrition.tufts.edu/2019/07/dcmupdate/

When Should CHF Recheck Examinations Be Done, and What Tests Should I Do?

The author routinely recommends re-evaluation of the patient with a chemistry profile to check renal function and electrolytes 7 to 10 days after initiation or alteration of cardiac medications. Physical examination, packed cell volume, total proteins, blood pressure, follow-up thoracic radiographs, follow-up electrocardiography, and historical reports from the owner are all useful in trying to assess response to therapies. In many instances, the doses or types of medications need to be adjusted at the time of this initial recheck and a subsequent visit 7 to 10 days later should be scheduled.

The next recheck visit should be scheduled for 2 to 3 months and at that time a physical examination with chemistry profile should be performed. Finally, 6 months after initial diagnosis the author recommends a follow up examination with echocardiogram to search for changes in the appearance of the heart or other alterations which might dictate a need for change in therapy, such as development of pulmonary hypertension, new arrhythmias, or complications of treatment.

Feline Cardiomyopathy

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Feline Cardiomyopathy

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Introduction

Feline cardiomyopathy can be difficult to identify and difficult to manage. Forms of cardiomyopathy include hypertrophic, dilated, restrictive and arrhythmogenic right ventricular cardiomyopathy. Cats with heart disease can present with asymptomatic disease, congestive heart failure (CHF), or with signs of arterial thromboembolism (ATE). Key diagnostic tests include the cardiac physical exam, ECG, echocardiography, laboratory testing, and thoracic radiographs. NT-proBNP and cardiac troponin I can play an important role in the diagnosis and management of cats with cardiac disease. There are few prospective clinical trials to support a specific set of recommendations in cats, so what follows is the author's current approach to feline cardiomyopathy, which is a mixture of experience, mistakes, research, and the current literature. This approach tries to cover the common clinical situations and discuss possible management approaches for each scenario. Much of what is suggested below involves off-label (unapproved) uses of drugs in cats.

ASYMPTOMATIC FELINE CARDIOMYOPATHY

The cat with LV hypertrophy, no outflow tract obstruction, and no atrial enlargement

Most people identify LV hypertrophy in a cat as a diastolic wall thickness > 0.6 cm for the interventricular septum (IVS) or left ventricular free wall (LVFW, or LVPW). There is probably some wiggle room here and some older cats and some smaller cats likely have LVH when the walls are > 0.55 cm, and one strategy using a weight-based method would identify hypertrophy when the walls are much thinner in many smaller cats. Cats with LV hypertrophy should be checked for systemic hypertension, and if they are > 6-7 years of age then thyroid testing to exclude hyperthyroidism is also recommended. Older cats with kidney disease and those with an enlarged aorta should have several measurements of blood pressure to exclude systemic hypertension.

Many cats with asymptomatic LV hypertrophy do not require therapy. If a cat is particularly tachycardia, or in cats with significant arrhythmias, one might treat with a beta blocker. In most cats, I would recheck the echocardiogram and cardiac exam in approximately 12 months. Some people may use beta-blockers in this population of cats, to prevent tachycardia-induced CHF, or to just slow the heart rate and enhance time for diastolic filling, but I have shied away from routine use this strategy for most cats (some people also use diltiazem). One of the concerns is the challenge of giving cats medications twice a day on an indefinite basis, and part of my concern is the uncertain benefit of this strategy (I suspect the number needed to treat to prevent 1 episode of CHF is quite high). A recent study identified that many of these cats will remain asymptomatic and die from non-cardiac causes. One possible exception to the "no treatment" approach is cats with very severe LV hypertrophy (LVFWd > 0.8 to 0.9 cm) and an NT-proBNP > 500 pmol/L – in this situation I might start an antithrombotic (despite lack of LA enlargement), usually clopidogrel, due to perceived increased risk for ATE formation.

The asymptomatic cat with LV hypertrophy and Left Ventricular Outflow Tract Obstruction

Beta blockers are the most commonly used drug in cats with hypertrophic cardiomyopathy who have left ventricular outflow tract obstruction (LVOTO). Cats with LVOTO often have significant LV and especially septal hypertrophy. Sometimes the top of the IVS is very hypertrophied and contributes significantly to LVOTO. LVOTO can be complicated by, or be mostly due to, systolic anterior motion (SAM) of the mitral valve. Due to any number of factors (elongated mitral valve, abnormal papillary muscle position due to LV hypertrophy, or congenital malformation, etc.), during systole the anterior mitral valve leaflet moves (or is carried) into the LV outflow tract and contributes to obstruction. The mitral valve leaflet may even come into contact with the IVS in systole in the LVOT below the aortic valve, creating a "slap lesion" on the interventricular septum. As the valve moves into the LVOT, the mitral orifice is no longer covered by the valve and there is typically secondary mitral regurgitation. The mitral valve regurgitation, plus the stiff LV in diastole, contributes to the development of left atrial enlargement in many cats.

Use of beta-blockers, in selected cats, can result in a reduction in the LVOT gradient, less SAM of the mitral valve, a reduction in the degree of mitral regurgitation, and some reduction in the degree of LV hypertrophy. While these echocardiographic findings can improve, and often the accompanying murmur is softer, it is less clear whether beta-blockade will delay the time until collapse, CHF, or arterial thromboembolism. One study looking at atenolol use in cats with HCM over a 5-year period failed to find a clear clinical improvement of the cats treated with beta-blockers. Due to this study, and my own clinical experience in which several cats placed on atenolol progressed a bit faster to CHF than was expected, I preferentially use carvedilol instead of atenolol in cats with LVOTO. The usual trigger for us to use a beta-blocker is the presence of moderate to severe LVOTO, often with SAM of the mitral valve, where the left ventricular outflow tract velocity is at least 3.5 to 4 m/sec. Atenolol is often given at doses of 6.25 mg PO q 12 hours,

and sometimes titrated to higher doses. Carvedilol is often started at a low dose with subsequent slow titration upwards to a higher dose. In many cats I start with ½ of a 3.125 mg tablet PO q 12 hours and after 2 weeks go up to 1 tablet PO q 12 hours (in very small cats or cats with prior/current CHF I start at ¼ tab PO q 12 hours). For stable cats with persistent LVOTO at the 4 to 6 month recheck I will titrate up to 1.5 tablets and then eventually 6.25 mg PO q 12 hours.

The asymptomatic cat with cardiomyopathy and moderate to marked LA enlargement

Asymptomatic cats with impressive left atrial (LA) enlargement are at risk for both CHF and ATE. In most cases, especially in cats with an NT-proBNP > 500 pmol/L, we would start an antithrombotic. The other options include initiation of an ACE inhibitor or pimobendan. We would usually start 1 or the other of these drugs in an attempt to delay the onset of CHF. In cats with more impressive concentric LVH and in those with LVOTO, we would usually start an ACE inhibitor. In cats with more LV dilation (LVIDd > 1.6-1.8 cm) and/or those with reduced LV contractile function (fractional shortening < 35 %) and no outflow tract obstruction, then we might be more likely to start pimobendan. Whether these drugs affect the trajectory of the disease and time until development of CHF is uncertain. In cats who are thought to be close to CHF, we might send them home with a few tablets of furosemide with instructions to start furosemide if dyspnea develops or if the resting respiratory rate rises > 35 breaths per minute.

CARDIOMYOPATHY AND CONGESTIVE HEART FAILURE

The approach for cats with CHF is similar regardless of whether a cat has HCM, dilated cardiomyopathy, or restrictive cardiomyopathy (except for cats with HCM and concurrent LVOTO plus CHF). CHF should be documented by a combination of clinical methods. CHF is most convincingly present in cats with clinical signs of dyspnea, and radiographic or ultrasonographic evidence (B-lines) of pulmonary edema and/or pleural effusion, and cardiomegaly with echocardiographic evidence of moderate to marked left atrial enlargement, and a quantitative NT-proBNP test of > 260 pmol/L. Most cats will also be strongly positive on a bedside (SNAP) NT-proBNP test. For the quantitative NT-proBNP test, the higher the value the more likely it is that a cat has CHF. Many of these cats will also have a mildly elevated cardiac troponin I concentration. Many cats with CHF will have a cardiac gallop on auscultation, often an S4 gallop, which results from atrial contraction into a stiff ventricle. Once the diagnosis of CHF is established then the following therapies might be used, typically in some combination. Most cats with CHF have enough left atrial enlargement that they are at risk for arterial thromboembolism, so antithrombotics (e.g., clopidogrel, a low molecular weight heparin, rivaroxaban or apixaban) should be used on a chronic basis.

Furosemide

Furosemide is indicated once CHF has been confirmed. The dose range is quite variable, from 1 mg/kg q 48 hours for chronic low-grade CHF up to 4 mg/kg every 1-2 hours for life threatening pulmonary edema. The lowest dose required to clear significant edema accumulations and cause the animal to be minimally symptomatic is the best dose. The lowest possible dose reduces the chance for electrolyte disturbance (hypochloremia, hypokalemia, hyponatremia, hypomagnesemia, and metabolic alkalosis), dehydration and the development of pre-renal azotemia. If concerning azotemia develops (BUN > 35-45 mg/dl, creatinine > 2.2-2.5 mg/dl) then in most cats a dose reduction will be required for the cat to maintain a normal appetite. In some cats, mild azotemia is “the price to pay” in order to achieve decongestion and lowering the dose of furosemide leads to recurrent signs of CHF. Serial measures of total solids can also help in furosemide dose adjustment. In acute pulmonary edema these values are checked daily, while in chronic management of CHF re-evaluation of renal values and electrolytes should be done at 7-10 days after adjustment of doses of diuretics or ACEi, and afterwards every 3 to 6 months.

In order to determine the lowest possible dose of furosemide which with control signs of CHF, a degree of experimentation and dose adjustment is usually necessary. We give the owner an upper and lower limits for acceptable furosemide dose, and carefully explaining to them that they should "give more for difficulty breathing or rapid respirations, and give less if the animal seems weak, lethargic, anorexic, or depressed". We use of a target respiratory rate of < 32 to 35 breaths per minute, when the cat is at home, at rest, and not purring, although some studies suggest a lower respiratory rate is usually present in cats with well managed CHF. Owners are instructed to give extra furosemide if the respiratory rate is > 35 breaths per minute or if respiratory effort (abdominal effort to breathing) is noted. Additional information on monitoring respiratory rate and effort can be found on the HeartSmart website. https://heartsmart.vet.tufts.edu/

In most cats we initially try to use 6.25 mg/cat once or twice a day for chronic therapy. Some cats require higher doses of furosemide, especially those with pleural effusion which required thoracentesis (in cases that required thoracentesis we usually start twice a day furosemide). Most veterinary cardiologists would not recommend single agent use of furosemide for treatment of CHF, so furosemide is used in combination with one or more of the drugs outlined below. Diuretic use in asymptomatic heart disease, before the onset of CHF, is usually not recommended. In asymptomatic cats with marked left atrial enlargement and thought to be at risk of CHF, we might send the owner home with furosemide tablets to give if dyspnea, tachypnea or shortness of breath is noted. Once cats have had well-documented CHF, I am usually reluctant to stop furosemide altogether, due to concerns about recurrent CHF. Even in

cats with a degree of azotemia and smaller left atrial size who maybe do not need furosemide, my preference is to still give furosemide at least every third day.

Angiotensin-Converting Enzyme Inhibitors

Angiotensin-converting enzyme (ACE) inhibitors are usually indicated once CHF has been documented. We do sometimes prescribe an ACE inhibitor in an asymptomatic cat with moderate to marked left atrial enlargement who is judged to be at higher risk for CHF. Possible side effects of ACE inhibitors include azotemia, hypotension, weakness, anorexia and hyperkalemia. Of these, azotemia and anorexia are the more common dose limiting side effects, but a low dose can often be tolerated in most cats with CHF. Target doses are as follows: enalapril 0.5 mg/kg q 12-24 hours, lisinopril 0.5 mg/kg q 24 hours, and benazepril 0.25 to 0.5 mg/kg q 12 to 24 hours. In most cats we use enalapril and start at 2.5 mg/cat/day (except in small cats < 3.5 to 4 kg) and increase to twice a day dosing in refractory CHF. I have had more luck using lisinopril in cats where I am worried about the kidneys, and usually start at 1.25 (small cat or cat with azotemia) to 2.5 mg/kg/day. Renal values and electrolytes should be measured before initiation of an ACE inhibitor, and in cats with CHF these values should be rechecked in 7 to 10 days and dose adjustments to the diuretic (or ACE inhibitor) made at that time. It is recommended to measure renal function serially when using ACE inhibitors, at least once a year if used in asymptomatic cats and q 3 to 6 months in cats concurrently getting furosemide.

Pimobendan

Pimobendan is a calcium sensitizing drug and phosphodiesterase inhibitor that is useful as a positive inotrope with vasodilator properties. This combined action of positive inotropy and vasodilation has been referred to as inodilation. Pimobendan has been well studied in dogs with documented utility both with CHF and before the onset of CHF, however it is less well studied in cats. Pimobendan seems to be associated with a low side effect profile in dogs, and except for cats that have LVOTO this has been our experience with cats as well. The main concern with cats is that those with LVOTO, or even mid-ventricular obstruction, might have worsening of the obstruction if given a positive inotropic drug. In general, pimobendan should be avoided, or given with caution only after other therapies have failed, in situations of cardiac outflow tract obstruction. In cats this usually means that an echocardiogram should be performed prior to initiation of pimobendan to exclude cats with LVOTO and/or SAM of the mitral valve. We have used pimobendan in cats with LVOTO, when other routine therapies have failed to control CHF. The usual dose for pimobendan is 0.25 to 0.3 mg/kg q 12 hours, and in most cats that translates to 1.25 mg/cat PO q 12 hours. We routinely prescribe pimobendan to cats with all forms of cardiomyopathy with CHF if they do not have LVOTO. On rare occasion, we see a cat that does not feel well on this drug, or is weak, and another possible side effect is hyperexcitability.

Beta-blockers in CHF

Beta blockade are usually not used in cats with CHF in our practice, unless there is marked to severe LVOTO, or concerning arrhythmias, and if we use a beta-blocker in this setting we are more likely to use carvedilol, starting at a low dose and titrating the dose up slowly, q 2 weeks.

ADVANCED CHF REFRACTORY TO THE ABOVE THERAPIES

When a furosemide dose of 6.25 mg/cat twice a day is exceeded during chronic therapy, the author usually thinks that diuretic resistance has been reached and may undertake one of several options to combat this complication. One choice is to add in spironolactone starting at 6.25 mg PO q 48 hours, and if this is well tolerated after 1 week then increase to 1 mg/kg q 24 hours, and eventually up to 2 mg/kg q 12 hours. Some veterinary cardiologists would routinely use spironolactone in cats with CHF, but in our practice, we see a fair number of cats that do not eat well on the drug, and so it is currently not a first line medication for us. I am less likely to use the combination tablet of hydrochlorothiazide with spironolactone unless a cat has refractory pleural effusion that requires frequent thoracentesis. We also sometimes use torsemide at 0.5 to 1.5 mg/cat orally once a day (in addition to the prior dose of furosemide), although this may be too much diuretic for some cats and serial evaluate of renal values and electrolytes is strongly recommended. In our practice, we might use torsemide before use of spironolactone or hydrochlorothiazide. Increasing the ACE inhibitor to q 12 hour is another option for refractory CHF. Increasing pimobendan to 3 times a day, or twice the original dose q 12 hours, is another option. Sildenafil can be added in at 1-2 mg/kg q 8 hours, and I am most likely to add in sildenafil to cats with pleural effusion and those with a pulmonary artery larger than the aorta on echocardiography. Dietary sodium intake should be reviewed and moderated if appropriate and possible. Finally, the furosemide dose can be escalated further, or sometimes injectable furosemide (subcutaneously) will be effective when oral administration seems less effective.

PREVENTION OF ARTERIAL THROMBOEMBOLISM

Arterial thromboembolism (ATE) is a common complication of feline heart disease, occurring in 5-40% of cats with cardiomyopathy and significant left atrial enlargement. The thrombus may develop in either the left ventricle or the left atrium, however, a left atrial origin from the left auricular appendage is most common. Dilation of the left atrium, reduced LA contractile function resulting in stasis of blood within in the enlarged LA, and perhaps endothelial damage

leading to activation of the clotting cascade and all likely contributes to thrombus formation. Since the treatment of arterial thromboembolism is very difficult and often unsuccessful, prevention of this devastating event becomes very important. We will often start antithrombotic medications in any cat with moderate atrial enlargement, cats with spontaneous contrast n echo, in almost all cats with CHF, and perhaps in cats with an NT-proBNP concentration > 500 pmol/L (as most cats we see with ATE have a BNP greater than this value).

Clopidogrel and Aspirin

A recent study demonstrated that clopidogrel was superior to aspirin in preventing recurrent ATE. This finding, combined with concerns regarding the action of aspirin on the GI mucosa and on the kidneys in cats with reduced renal perfusion, has caused us to largely abandon use of aspirin in cats. Other cardiologists routinely use a combination of clopidogrel and aspirin, termed dual antiplatelet therapy as they work on different targets in the platelet, and this works well in their experience. Aspirin can also make furosemide less effective. The antiplatelet drug clopidogrel (Plavix) is now our routinely used drug for prevention of ATE in cats. The drug comes in a 75 mg tablet and we give ¼ tablet PO q 24 hours to most cats, except for cats < 3.5-4 kg, in which case we might compound the drug to give a smaller dose. Clopidogrel seems to be well tolerated in many cats, although foaming at the mouth or vomiting has been seen in 15-40% of cats. Compounding the drug may reduce this side effect for some cats (try marshmallow flavor), although the taste may still trigger this reaction in other cats. In most cats we place the ¼ tablet inside of an empty gelatin capsule and have the owner administer the gelatin capsule (followed by some liquid or food if possible). There is no routine monitoring of clotting times or any other blood tests for this drug. Some clinicians use a dual antiplatelet strategy, and give both clopidogrel and aspirin. A very rare side effect of clopidogrel is a hepatopathy, with increased bilirubin and liver enzymes, which is usually reversible with discontinuation of the drug (and perhaps this occurs more often in small cats).

Low Molecular Weight Heparins - Dalteparin and Enoxaparin

Low molecular weight heparins (Dalteparin and Enoxaparin) are sometimes used to prevent thrombus formation in cats at high risk of ATE, especially in cats who have already experienced ATE. We have used dalteparin (Fragmin; 160 to 250 U/kg) subcutaneously twice a day. Enoxaparin is given at 1 mg/kg subcutaneously twice a day. One major action of the drug is to inactivate clotting factor Xa. Both dalteparin and enoxaparin can be expensive to use on a long-term basis (several hundred US dollars per month for twice a day treatment in most cats). The drug must be given by subcutaneous injections. Yet, many owners prefer injections to oral medications in cats, and the drug is well tolerated by most cats. We sometimes combine clopidogrel and a low molecular weight heparin, especially in cats with prior ATE, in those with a thrombus visualized in the LA, and in cats with heavy “smoke” or spontaneous contrast in the LA.

Coumadin (Warfarin)

Coumadin works by blocking the vitamin K dependent clotting factors II, VII, IX and X. By titrating the dose of warfarin, a degree of anticoagulation can be achieved to reduce thrombus formation. We do not use coumadin in our practice for cats due to the higher risk of bleeding and difficulties with dose titration.

Rivaroxaban and Apixaban

These drugs are anticoagulants that will likely replace coumadin, and eventually may be the “go to” drugs for prevention of ATE in cats. We have used rivaroxaban in a handful of cats at 2.5 mg/cat/day. Unexpected bleeding is the main concerning side effect, and if these drugs are used then cats should be protected from trauma, and care should be taken for phlebotomy, cystocentesis, surgery and other routine procedures. A proposed dose of apixaban in cats is 0.625 mg PO q12h in cats < 5 kg, and 1.25 mg PO q12h in cats > 5 kg, but we have less experience with this drug in our practice. For owners that can afford these drugs and are willing to try something with less of a track record for safety and efficacy, we would use them in cats with a prior ATE and in cats judged to be at very high risk for ATE (thrombus seen on echo or very impressive spontaneous contrast).

TREATMENT OF NEW ONSET ATE

Once ATE develops, treatment is largely empiric, and there is little scientific information to direct the best treatment approach. Yet, all cats that are euthanized for ATE die, and some that are given supportive care will survive. Most cats with a front leg ATE will regain function of the limb (over 90%). Perhaps 40-50% of cats with a single back leg ATE will regain function. The survival is worse for cats with bilateral back limb ATE, and those with bilateral rear limb ATE a rectal temperature < 98.5 degrees F likely have the lowest chance for survival. If treatment is to be initiated then it should be started ASAP, ideally within 10 minutes of arrival at the hospital, even before discussions with the owner have concluded. I would recommend giving at least ½ to almost a full tablet of clopidogrel (a loading dose) and giving 250-300 units/kg of unfractionated heparin subcutaneously (or intravenously) as soon as the diagnosis is evident. While use of a central blood glucose and/or lactate compared to blood glucose and lactate in the affected limb has been recommended, we usually try to avoid this approach. It is true that the blood glucose is usually lower in the affected

limb, and the lactate is usually higher, it is also true that the affected limb has reduced blood flow and thus limited blood flow to counteract the breach in the skin. I worry that infection in the affected limb is more likely if there is no blood flow to the limb. In most cats, the diagnosis can be established via a combination of historical and physical exam findings. Most cats will have sudden onset weakness or paralysis of the affected limb(s), lack of arterial pulses, a limb that is cool to the touch, pale or bluish nail beds, and the muscles of the limb may become firm over time.

Control of pain, nursing care, some antithrombotic, management of CHF if present, and general supportive measures are indicated. Many cats have open mouth breathing due to discomfort, without CHF, and if furosemide is given to cats without CHF then it can volume contract them and further reduces the chance that reperfusion will occur (so ideally get a thoracic radiograph before giving furosemide!). Heparin is the drug most commonly used to prevent further enlargement of the thrombus. Heparin can be administered subcutaneously or intravenously. An initial intravenous bolus of heparin (250-300 units/kg) can be followed by a continuous rate infusion of heparin (12-25 units/kg) or subcutaneous administration (200-300 units/kg q 6-8 hours). Heparin is usually continued until the time of hospital discharge or until other antithrombotic therapy has been initiated for at least 24-48 hours. Low molecular weight heparin (LMWH) has been used in this setting instead of unfractionated heparin, and if the setting of ATE we give high doses with increased frequency (dalteparin at 200-300 U/kg q 6 hours or enoxaparin 1-1.5 mg/kg q 6 hours). We usually will not give aspirin in the acute setting of ATE, although there is evidence in people to suggest that aspirin administration is settings of acute thromboembolic disease (e.g., coronary or cerebral events) might be useful.

Thrombolytic drugs can actually dissolve a thrombus once it has formed. There is no clear consensus as to whether these drugs should be used if a thrombus is identified in a cardiac chamber, as their use may result in liberation of the thrombus and subsequent arterial thromboembolism. Tissue plasminogen activator, urokinase and streptokinase have been used in cats, but without clear cut superior outcomes compared to supportive care and antithrombotic drugs. Bleeding complications are possible and complications resulting from reperfusion injury (hyperkalemia and metabolic acidosis) are almost always life threatening. If thrombolytic drugs are to be used they should be initiated immediately, ideally within two to four hours after the onset of clinical signs, as delayed use of thrombolytics in people has been associated with lack of efficacy and increased complications. Surgery (embolectomy) can be attempted in cats, but anecdotal clinical experience in veterinary medicine indicates that surgery for thromboembolism probably doesn't improve the clinical outcome.

Getting the Most of your Cardiology Friend

Dr. Liz Rozanski - Tufts University and Dr. John Rush - Tufts University

Getting the Most Out of your Cardiology Friend

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Respiratory distress, heart murmurs and irregular pulses can cause anxiety for any practitioner no matter how much experience they have! The ACVIM directory has < 300 cardiologists listed in the US, and obviously fewer in the Midwest. KSU has an active cardiology program. As with any specialist, it is nice to work more closely with one or two people if possible, rather than a larger number, as it is easier to know your practice style and have better referrals on from all three perspectives, including the practitioners knowing what to expect and how to prepare the family, the family knowing about possible outcomes, and the cardiologist knowing what goals are of the evaluation. Many internists and some criticalists also do echocardiograms, and depending on the complexity of your patient's disorder, this may be more than adequate. Additionally, for hospitals that have ultrasound capability, many clinicians are able to quickly gain skills in real-time patient assessment that can help make a decision if more definitive (eg. Referral for echo) testing is warranted.

https://www.youtube.com/watch?v=I4U8AoxYmok&feature=youtube

Skills you would expect your cardiologist colleague to have include obviously the echocardiogram, but also the overall assessment of cardiovascular health, and recommendations for treatments and perception of prognosis. Some cardiologists are also able to perform interventions such as pacemaker implantation, and PDA occlusion.

So, what kinds of questions should you ask of a cardiologist?

About the individual pet

1) Does this pet have heart disease?
2) What should I do about it?

About cardiology

3) *Some increased understanding of the disease*
4) *What is "new" in cardiology and what is developing as an issue (pimobendan versus filiarbits)*

What should a cardiologist hope for from a referred case? If it is not clear, why the consult/referral indicated and what the general practitioner is looking for. A summary of past health may be relevant, including if murmur has been heard before or if there is increased coughing or other concerns, diet history, heartworm preventive, and often recent laboratory work.

We are taught in veterinary school and recognize in practice the physical examination has strong predicative ability for the presence or absence of heart disease. However, in some cases, it is not enough to support or refute heart disease, as well as fully recognize the severity of the underlying condition. Most of this discussion will be case- based, but the following in a summary of when you might want to talk to a cardiologist.

New puppy or kitten examination

Flow murmurs are common in puppies and kittens; key findings that would make you want to consult a cardiologist include a loud murmur (>2/6), weak or bounding pulses or blue mucous membranes. PDA murmurs can sometimes be hard to hear, but almost always have a bounding pulse quality. If you feel a bounding pulse, remember to ausculate very far cranially (in the axilla) if you don't hear a murmur clearly over the heart. Some specific breeds of dogs (Goldens, Newfies) are predisposed to subaortic stenosis and a murmur in those puppies should be taken seriously. Brachycephalic puppies may be predisposed to pulmonary stenosis. "Puppy mIll" puppies may also be more commonly affected with congenital cardiac disease.

New Murmur – Small breed dog

The presence of a new murmur can be an unexpected clinical finding. Many older Small breed dogs develop new murmurs as the result of mitral valve degeneration. If a new murmur is heard in a dog that is not feeling well, a PCV should be checked as well as anemia may result in a mumur. An echocardiogram is never a bad test, but if the dog is otherwise well, (s)he may also be safely monitored. Thoracic radiographs are useful to look for left atrial enlargement, and an NT-proBNP blood test can be easily used for evidence of impending heart failure (or at least left atrial stretch). If owners are not interested in pursuing an echo or if echo unavailable (and honestly even if echo confirms mitral valve disease), the owner should continue to monitor the dog at home, ideally with resting respiratory rate, and consider dietary transition. Weight loss *unplanned can be an early warning of progression to heart failure. Other clues from a clinical perspective of impending heart failure include loss of sinus arrhythmia and increasing grade of murmur. Owners should be advised that many small breed dogs have mitral valve disease, and never progress to CHF.

- Murmur intensity usually proportionate to severity of mitral value disease
- Generally slow progression

New Murmur- Large breed dog

Larger breed dogs certainly may develop mitral valve disease, but it is far less common. Other forms of heart disease are more common, such as DCM or pericardial effusion. Otherwise, similar guidelines are reasonable as for smaller dogs.

New Murmur- cats

Cats are incredibly tricky, as a cat can have a super loud murmur and no significant heart disease or no murmur and very advanced heart disease. So, it is always reasonable to advise an echo in a cat with a murmur, however, it is far less easy to predict the presence of significant heart disease than in small dogs. The presence of a gallop or irregular rhythm will perhaps more strongly support cardiac disease, and if these are heard, you should push more strongly for evaluation. NT pro-BNP is also super helpful in cats, perhaps more so that in dogs as a screening test. These are available as both a SNAP test in cats, and a send-out (IDEXX) test in dogs and cats. Chest radiographs are helpful, but as much of the acquired cat heart disease is hypertrophic cardiomyopathy, cardiomegaly may or may not be easily detected. If it is present, an echo is clearly advised.

- Murmur not related to severity of disease
- Much harder to predict

So, what about is a “Pet ok for anesthesia?”

Anesthesia has inherent risks, and cardiac disease can increase those. Your first questions are typically “What do I want to do?” “How long will it take” and “How important is it” For dogs not on heart medications, typically anesthesia will be well-tolerated. Dogs with mitral valve disease tend to tolerate anesthesia fairly well, as do cats with hypertrophic cardiomyopathy not in failure. Animals with dilated cardiomyopathy have a much harder time recovering from anesthesia. So, in general, if animals in active heart failure, anesthesia should be avoided if at all possible.

In anesthesia is needed, opioids and midazolam/diazepam are relatively cardiac safe. Intubation and ventilation is usually safer than heavy sedation. Close monitoring (ECG/pulse ox/ETCO2) is advised. Propofol (and alfaxalone) , while fast-acting, are potent cardiac /respiratory depressants and should not be used alone. Procedure time should be limited if possible, and consideration should be given to keeping the procedure as short as possible.

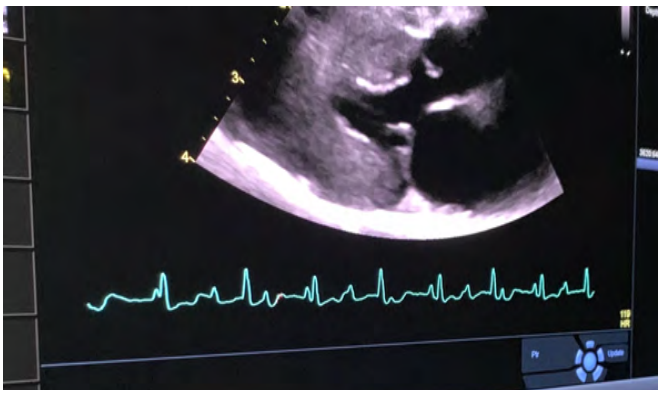
Dental care is hard decision, severe dental disease can affect the quality of life; but lengthy procedures may be hard to recover from and the pro and cons should be balanced out cautiously.

I hear something funny

If you hear an abnormality during an exam, it is helpful to try to get an EKG to better interpret what is happening. AliveCor is a nice option or if you have a regular EKG that can be very helpful as well. Try to get a long strip within limited artifact (easier said than done!). It can be helpful to remember that ventricular abnormalities are more common extra-cardiac (eg. Splenic masses) unless they are seen in Boxer, Dobies or maybe English Bulldogs.

Summary

Cardiology can be a very rewarding aspect of practice, and partnering with a cardiologist can be fun and help with patient care and understanding!



Management of Hypertension in Dogs and Cats

Dr. William Whitehouse - Kansas State University

Update on Management of Systemic Hypertension in Dogs and Cats

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Pathophysiology

Overview

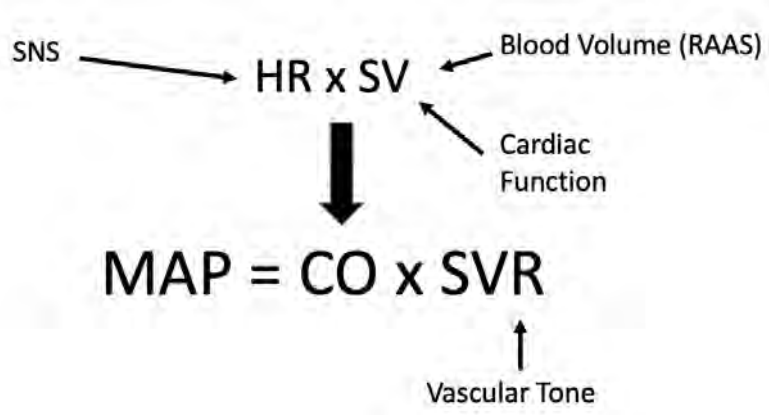
- A few initial notes about systemic hypertension (SHT)
  - It is not a disease → it is a complication of a disease and a clinical finding
  - Most causes are not resolvable
  - Management is focused on end-organ survival
- Systemic hypertension in people
  - Systemic hypertension is a major risk factor for heart disease and stroke
    - In 2017, heart disease was the #1 and stroke was the #5 cause of death in the United states[1]
    - Worldwide, hypertensive cardiac disease has increased by approximately 50% from 2007 to 2017[2]
  - Systemic hypertension is commonly heritable in people → primary (essential) hypertension[3]
    - Associations with gender, race, obesity, high sodium diets
- Systemic hypertension in dogs and cats
  - More likely secondary to another disease process (>80%)[4]
    - Chronic kidney disease
    - Protein-losing nephropathy
    - Certain endocrinopathies
  - Primary (essential) systemic hypertension is thought to be uncommon
    - Most of our patients have underlying kidney disease, so could we be missing a diagnosis of subclinical chronic kidney disease?
    - Consequently, the term "idiopathic" is preferred over "primary" or "essential" in veterinary medicine
- How is systemic hypertension similar amongst dogs, cats, and people?
  - Chronic increases in blood pressure cause injury to tissues
  - Target-organ damage (TOD)
    - Ocular
    - Renal
    - Neurologic
    - Cardiac
  - Many of the underlying diseases that cause systemic hypertension in dogs and cats also cause systemic hypertension in people
  - Our patients have a similar response to medications

Hypertension and Kidney Disease

- In people, there are multiple risk factors for the development of systemic hypertension (gender, race, obesity)
- Additionally, people may be considered "salt sensitive" where a high salt diet could lead to chronic increases in blood pressure

- A main determinant of this is abnormal renal sodium handling from kidney disease, but many people without kidney disease are found to be salt sensitive with genetics, changes in sodium accumulation in the interstitium, and alterations in endothelial cell function as some of the proposed mechanisms[5]
- Our small animal patients are not considered to be salt sensitive
  - Canine models were used in the original studies describing pressure natriuresis and diuresis[6]
  - Renal disease was the determining factor that caused an increase in systemic blood pressure despite the same sodium intake
- Kidney disease causes systemic hypertension, and systemic hypertension worsens kidney disease in a vicious spiral; however, the kidney disease must come first
  - Defects in renal excretory function appear to be a prerequisite for chronically increased blood pressure
  - Idiopathic hypertension implies the presence of kidney disease (could these patients have early kidney disease?)
  - If the kidneys were normal, they would excrete "excess" salt and water to decrease blood pressure

Control of Blood Pressure



Measurement of Blood Pressure

Types of BP Devices

- Direct BP measurement → arterial catheterization with measurement by an electronic pressure transducer
  - Gold standard
  - Impractical
- Indirect BP measurement
  - Doppler sphygmomanometry → transmitting and receiving transducer emits ultrasound waves that detect motion of a blood vessel wall or RBC movement causing a frequency change from ultrasonic to audible that is amplified by the doppler machine
    - Measures systolic arterial pressure (SAP)
  - Oscillometric → measures oscillations in a cuff bladder
    - Less reliable with tachycardia and arrhythmias

    - High definition oscillometric → processor is considerably more powerful allowing real time analysis of the oscillometric curve and exact control of the valves that determine inflation and deflation making it more accurate over a wider range of pressures
        - Limited studies in awake animals
        - Difficult to get repeatable measurements in cats
- Recommendations on what to use[4]
    - Indirect device designed for veterinary use
    - Validated in conscious dogs and cats
    - Systolic BP should be used for clinical decision making

<u>ACVIM Consensus Guidelines</u>[4]

- Operator
    - Skilled and experienced
    - Most skilled person usually a tech
- Location
    - Quiet area
    - Away from other animals
- Timing → reduce "White Coat Effect"[7,8]
    - Before other procedures
    - Allow patients to become acclimated for 5-10 min
    - Owner should ideally be present
- Cuff selection
    - 30-40% of circumference of extremity at site of cuff placement
- First measurement discarded
- Average 5-7 consecutive readings
- Repeat if significant variation
- BP trends downward → wait until plateau then take 5-7 readings
- Document
    - Patient position and attitude
    - Device used
    - Cuff site
    - Cuff site circumference
    - Cuff size
    - Individual taking measurements
- What type of device to use?
    - Dogs[9,10]
        - Medium and large – oscillometric
        - Small – either (Doppler)
    - Cats[11]
        - Doppler on forelimb
        - Oscillometric on tail
- Who gets screened?
    - 1. Patients with clinical abnormalities consistent with hypertensive TOD
        - Progressive CKD → progressive azotemia or proteinuria
        - Retinopathy/choroidopathy → acute blindness, retinal edema/hemorrhage, tortuous vessels, retinal detachment
        - Encephalopathy → white matter edema and vascular lesions (hemorrhage and infarction), clinical signs may vary

        - Left ventricular hypertrophy → left ventricular hypertrophy, systolic murmurs gallop, development of congestive heart failure is infrequent but may occur
        - Vascular abnormalities → epistaxis
    - 2. Presence of diseases associated with systemic hypertension
        - Dogs
            - Chronic kidney disease (9-93%)
                - PLN (80-90%)
            - Acute kidney injury (87%)
            - Hyperadrenocorticism (73-80%)
            - Diabetes mellitus (24-46%)
            - Pheochromocytoma (43-86%)
        - Cats
            - Kidney disease of any type (19-65%)
                - May be higher with proteinuria
            - Hyperthyroidism (5-87%)
            - Hyperaldosteronism (50-100%)
    - 3. Treatment with pharmacological agents or exposure to toxins that may increase BP
        - Medications
            - Glucocorticoids
            - Mineralocorticoids
            - Erythropoiesis-stimulating agents
            - Phenylpropanolamine
            - Toceranib phosphate
        - Toxins
            - Methamphetamine/amphetamine
            - 5-hydroxytryptophan

Diagnosis

Classification Based on Risk of TOD[4,12]

Systolic Blood Pressure mmHg	Blood Pressure Substage	Risk of Future Target Organ Damage
<140	**Normotensive**	Minimal
140 – 159	**Prehypertensive**	Low
160 – 179	**Hypertensive**	Moderate
≥ 180	**Severely hypertensive**	High

Assessment of SHT

- Typically based on SBP (systolic blood pressure)
- SHT should be confirmed on multiple (>2) occasions
    - Rules out situational hypertension
    - Except in presence of TOD
- Patients with "prehypertension" typically not treated → monitor
    - Those who are normotensive but have diseases known to cause SHT → monitor
- Those with "hypertension" or "severe hypertension" → start treatment

Work Up

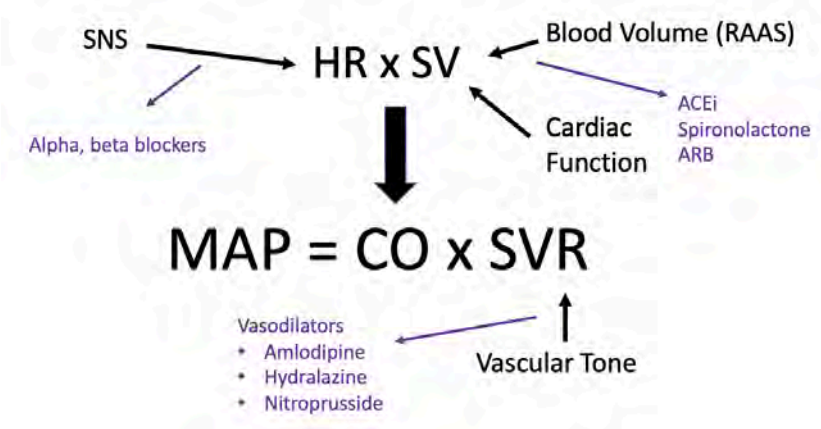
- Once SHT is diagnosed, search for possible underlying disease
- Treat underlying condition
  - Most patients fail to become normotensive
  - May still have some reduction in BP
  - May make SHT more amenable to treatment
- Dogs and cats
  - CBC/chem/UA
  - UPC
  - Abdominal ultrasound
- Dogs
  - Testing for hyperadrenocorticism
  - Urinary metanephrines
- Cats
  - Thyroid
  - Aldosterone

Treatment

General Goals

- Therapy must be individualized to the patient
- Once daily meds is ideal
- Gradual persistent reduction in BP
- Acute, severe decrease should be avoided
- Reduce TOD
- Goal is to reduce BP to <160 mmHg (ideally <140 mmHg)
  - Ultimate goal is to decrease magnitude, severity, and likelihood of TOD

Antihypertensive Therapy in Dogs[4]



- RAAS inhibitors
  - Examples
    - Angiotensin converting enzyme inhibitors
    - Angiotensin receptor blockers
    - Aldosterone antagonists → NOT first-choice
  - Typically, first line therapy
    - Enalapril or benazepril 0.5 mg/kg PO q12h
    - Telmisartan 1 mg/kg PO q24h

- Usually SBP reduction of 10-15 mmHg (at least with ACEi)
- Calcium channel blockers (CCB)
  - Smooth muscle dilator
  - Amlodipine 0.1-0.5 mg/kg PO q24h
  - Should be avoided as monotherapy in dogs (may exacerbate proteinuria)

<u>Antihypertensive Therapy in Cats</u>

- CCB is the first line therapy in cats
  - Amlodipine[13]
    - 0.625 mg/cat PO q24h if SBP <200 mmHg
    - 1.25 mg/cat PO q24h if SBP >200 mmHg
  - Mean reduction in SBP of 28-55 mmHg[13-17]
  - Transdermal amlodipine less preferred route[18]
- Hypertension and proteinuria in cats
  - Survival in CKD cats appears to be linked to proteinuria and not hypertension[14,19,20]
  - Amlodipine can reduce proteinuria[19]
  - Amlodipine + ACEi or ARB appears to be well tolerated[14,21]
  - No current studies showing an additional benefit to RAAS inhibition
- Telmisartan
  - ARB licensed in Europe for treatment of proteinuria in cats since 2013
  - Approved by the FDA in 2018 for treatment of SHT in cats
  - Two recent, fairly similar multicenter studies have come out evaluating the use of telmisartan for the treatment of feline SHT (both funded by Boerhinger Ingelheim)
    - The first study was out of Europe and evaluated 285 cats with a SBP between 160-200 mmHg and no TOD[22]
      - ❖ They were divided into a telmisartan group that received 2 mg/kg PO q24h versus placebo
      - ❖ 252 cats completed the first part of the study which was a 1-month evaluation of efficacy, and the telmisartan group had a mean reduction in SBP of 19.2 mmHg by day 14 and 24.6 mmHg by day 28 of treatment
      - ❖ Approximately 50% of telmisartan-treated cats reached a SBP <150 mmHg or had >15% decrease in SBP from baseline at day 28
      - ❖ 144 cats completed the second part of the study where they were followed for 120 days of treatment, and approximately 60% of telmisartan-treated cats reached a SBP <150 mmHg or had >15% decrease in SBP from baseline at day 120
      - ❖ Only 2% of cats treated with telmisartan were reported to become hypotensive
    - The second study out of UGA evaluated 221 cats also with a SBP between 160-200 mmHg and no TOD[23]
      - ❖ They were also divided into a telmisartan group that received 1.5 mg/kg PO q12h for 14 days followed by 2 mg/kg PO q24h
      - ❖ 173 cats completed the first part of the study which was a 1-month evaluation of efficacy, and the telmisartan group had a mean reduction in SBP of 23.3 mmHg by day 14 and 23.9 mmHg by day 28 of treatment
      - ❖ Approximately 20% of cats treated with telmisartan needed to be rescued with another therapy because of persistent severe hypertension (SBP ≥180 mmHg)
      - ❖ 13.4% of cats in the telmisartan group needed dose-reduction due to hypotension, and these cats weren't included in the day 28 analysis
      - ❖ Similar to the other study, approximately 50% of telmisartan-treated cats reached a SBP <150 mmHg or had >15% decrease in SBP from baseline at day 28

        - ❖ 73 cats completed the secondary part of the study where they were followed for 182 days of treatment, and 63% of the telmisartan-treated cats reached a SBP <150 mmHg or had >15% decrease from baseline at day 182
    - Dose: 1.5 mg/kg PO q12h x 14d then 2 mg/kg PO q24h
    - Reduction of BP by 20-25 mmHg

Other Antihypertensives

- Hydralazine
    - Direct vasodilator
    - Dog: 0.5-2 mg/kg PO q12h
    - Cat: 2.5 mg/cat PO q24-12h
- Diuretics – not routinely used in vet med (most of our patients have CKD)
    - Hydrochlorothiazide 2-4 mg/kg PO q12-24h
    - Furosemide 1-4 mg/kg PO q8-24h

Specific Diseases

- Feline hyperthyroidism
    - Atenolol for tachycardia
    - 6.25-12.5 mg/cat PO q12h
- Hyperaldosteronism
    - Spironolactone 1-2 mg/kg PO q12h
    - May still need amlodipine
- Pheochromocytoma
    - α_1 blocker – phenoxybenzamine 0.5 mg/kg PO q24h
    - May need β blocker and amlodipine

Follow Up

- BP, fundic exam
- Renal panel, UA/UPC, etc.
- Frequency and nature of BP rechecks will vary
    - TOD → 1-3d
    - Change in therapy → 7-10d
    - Routine → 1-4m

Hypertensive Emergencies

- Marked elevations in SBP and signs of ongoing acute TOD
    - Most likely ocular or neurologic
- Therapeutic target is incremental reduction of SBP, not acute normalization
- SBP should be reduced by 10% in first hour then 15% over the following few hours[24]
- Amlodipine
- Hydralazine 1-2 (up to 4) mg/kg PO q12h (rapid onset)
- If parenteral meds are used, arterial catherization for continuous BP monitoring is recommended
    - Hydralazine 0.1 mg/kg IV over 2 min then 1.5-5 ug/kg/min
    - Nitroprusside 0.5-3.5 ug/kg/min
    - Fenoldepam 0.1-1.6 ug/kg/min
    - Labetalol 0.25 mg/kg IV over 2 min repeated up to 3.75 mg/kg then 25 ug/kg/min

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The emergence of Atypical and Indolent Lymphomas in Dogs

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The Emergence of Atypical and Indolent Lymphomas in Dogs

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Veterinary Health Center
At Kansas State University
To discover. To teach. To heal.

Outline

- Introduction
- Cases
- Classification
- History and clinical presentation
- Cytology
- Flow cytometry
  - B-cell / T-cell testing
- Clonality testing
  - PARR
- Histopathology
- Specific types of indolent lymphomas
  - T zone
  - Mantle cell
  - Marginal zone
  - Follicular

Incidence

- Hematopoietic neoplasia is second only to skin neoplasms in dogs
- 7-24% all canine neoplasms
- 83% all hematopoietic neoplasms
- DLBL is the most common
- Incidence is increasing

Case 1 - Casey

- Signalment
- History
- PE
- Diagnosis
- Treatment
- Outcome

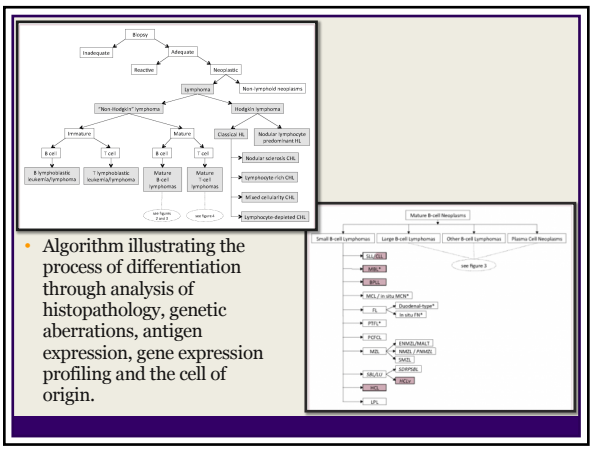
Case 2 - Sadie

- Signalment
- History
- PE
- Diagnosis
- Treatment
- Outcome

Human classification

- >50 different types of lymphoma or leukemia as classified by the World Health Organization (WHO)

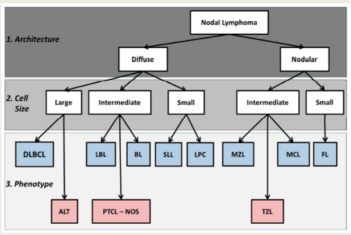
Swerdlow et al. Blood. 2016; 127(20): 2375-2390



- Algorithm illustrating the process of differentiation through analysis of histopathology, genetic aberrations, antigen expression, gene expression profiling and the cell of origin.

Canine classification

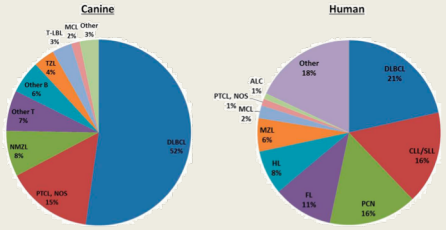
- In dogs the process is similar



Seelig et al, Vet Sci ,2016; 3: 11.

Differences

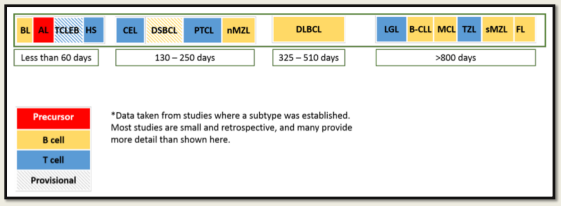
- Comparison of sub-types in dogs and people



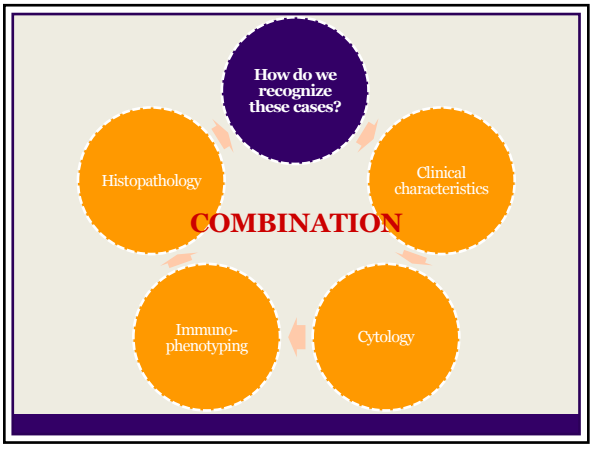
Seelig et al. Vet Sci. 2016; 3: 11

Why does this matter?

- Frequency, prognosis and treatment

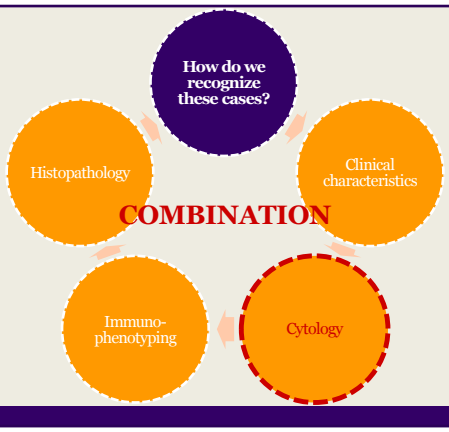


Avery, Workshop Proceedings of the Fourth Meeting of the European Canine Lymphoma Group, CH-Lugano, June 22nd 2019



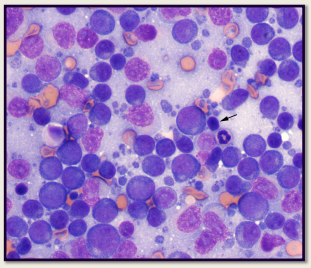
History and clinical presentation

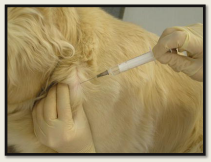
- Historical clues
  - Timeframe
    - *Not* rapidly progressive
  - Well dog
    - Incidental finding
- Physical examination
  - *Only* mild to moderate (and only slowly progressive) lymphadenomegaly
  - A single node
  - An atypical location
  - Solitary splenic involvement



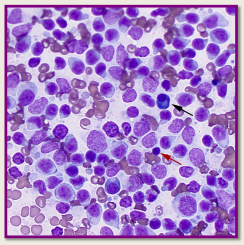
Cytology

- Cytomorphologic characteristics





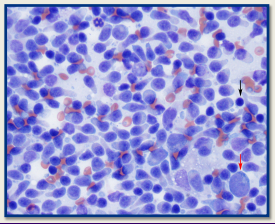
courtesy of Cornell University's College of Veterinary Medicine's eClinpath



High grade T-cell lymphoma

courtesy of Cornell University's College of Veterinary Medicine's eClinpath

Indolent T zone lymphoma



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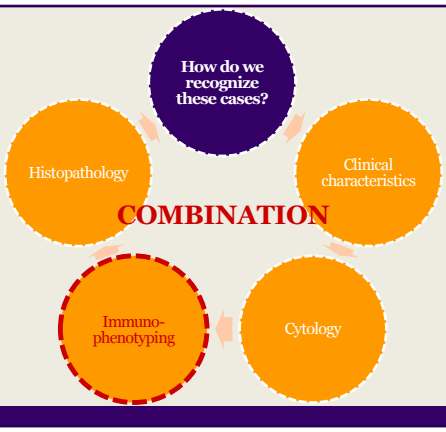
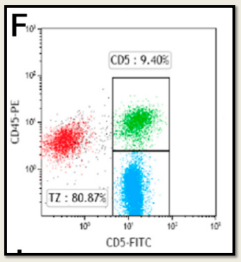


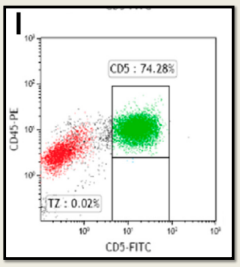
Immunophenotyping

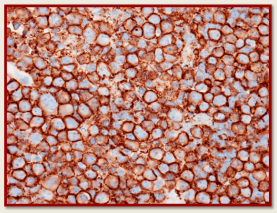
- B-cell and T-cell testing
- Samples
  - Immunocytochemistry (ICC)
    - Unstained cytology smears for microscopy (CSU)
    - Cellular suspension (FNA or blood) in EDTA saline for flow cytometry (KSU)
  - Immunohistochemistry (IHC)
    - 10% formalin-fixed tissue sample



T zone

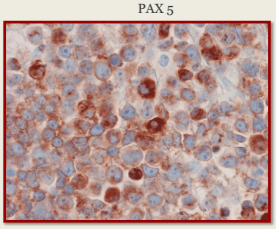
Normal LN





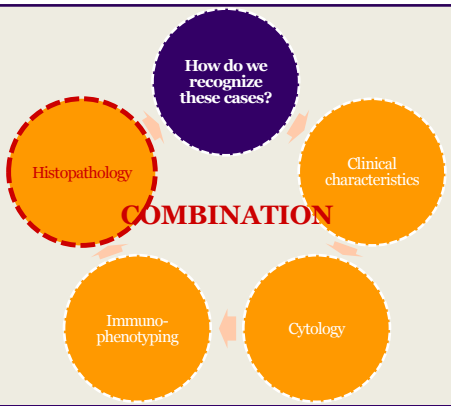
T-cell markers
CD 3
(CD 5)

B-cell markers
CD79a
CD 20 / 21
PAX 5

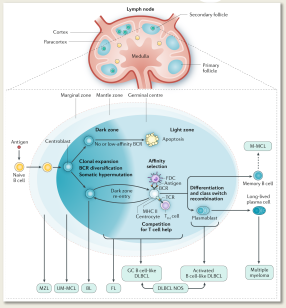


PARR

- Polymerase chain reaction (PCR) for antigen receptor rearrangement
- Clonality testing
- Samples
  - Cytology unstained smear preps (CSU)
  - Cytology cellular suspension in EDTA sterile saline (NCSU)
  - Histology formalin-fixed tissue samples (MSU)



Histopathology

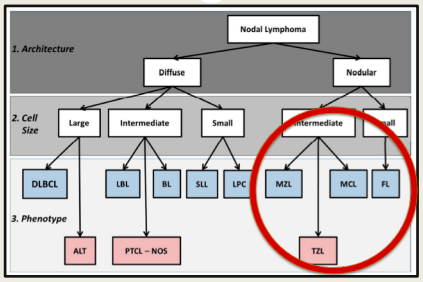


Carbone, A., Roulland, S., Gloghini, A. *et al.* Follicular lymphoma. *Nat Rev Dis Primers* 2019; 5. 83. https://doi.org/10.1038/s41572-019-0132-x

Common types of indolent lymphoma

- T zone lymphoma
- Marginal zone lymphoma
- Mantle cell lymphoma
- Follicular lymphoma
  - Nodal
  - Splenic
  - GI

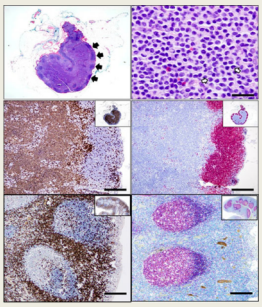
Common types of canine indolent lymphomas



Seelig et al, Vet Sci ,2016; 3: 11.

T zone lymphoma (TZL)

- Histopathology



Valli et al. Vet Pathol. 2011; 48(1): 198-211

T zone lymphoma (TZL)

- Cytology
  - Clear cell type
- Immunophenotyping
  - T cell markers
  - CD45-, CD3+
  - May be exceptions to this flow cytometric pattern
- Hematology (CBC)
  - Often circulating atypical cells

T zone lymphoma (TZL)

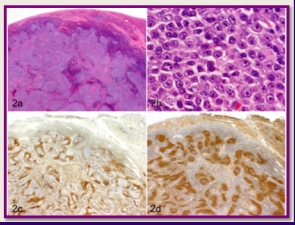
- Clinical history and presentation
  - Enlarged peripheral lymph nodes
  - No other PE abnormalities
  - Well dogs
- Staging

T zone lymphoma (TZL)

- Therapy
  - Active surveillance
  - Criteria for instituting treatment
    - Unwell dog
    - Progressive disease, with additional organ involvement
  - What therapy???
    - CHOP versus less aggressive protocols
- Outcomes
  - MST: ~14-33 months

Marginal zone lymphoma (MZL)

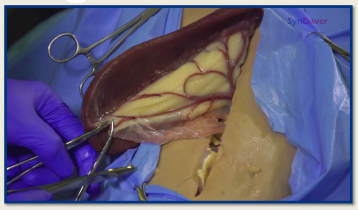
- Histologic characteristics
- Immunophenotypic characteristics
  - B cell markers
  - CD45+, CD20+, CD79a+, Pax-5+, Bcl-2+, MUM-1+, Mcl-1+ (1/3)



Valli et al. Vet Pathol. 2011; 48(1): 198-211

Splenic marginal zone lymphoma (MZL)

- Staging
- Therapy
  - Splenectomy
- Outcome
  - "Cure" with splenectomy
  - MST: ~38 months

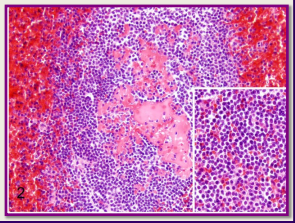


Nodal marginal zone lymphoma (MZL)

- Staging
- Therapy
  - Best approach to management is unknown
- Poorer prognosis
  - PFI: ~5 months
  - MST: ~8.5 months

Mantle cell lymphoma (MCL)

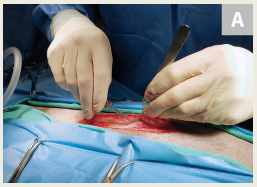
- Histopathology
- Immunophenotyping
  - B cell markers
  - CD45+, CD20+, CD79a+, Pax-5+
  - Negative for MUM-1, Mcl-1



Stein et al. Vet Pathol. 2019; 56(3): 350-357

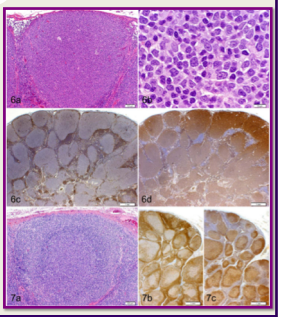
Mantle cell lymphoma (MCL)

- Staging
- Therapy
  - Splenectomy
- Outcomes
  - Preliminarily good outcomes, albeit small numbers reported
  - Less common than MZL
  - Contrasts with the disease in humans



Follicular lymphoma (FL)

- Histology
- Immunophenotyping
  - B cell markers



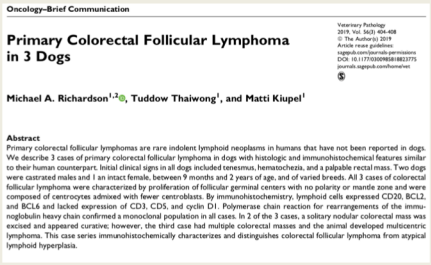
Valli et al. Vet Pathol. 2011; 48(1): 198-211

Follicular lymphoma (FL)

- Therapy
  - Best approach is unknown
  - Surgery a consideration
- Outcomes
  - Few cases reported in the literature
  - Preliminarily reasonable outcomes
  - Transformation to a more aggressive form is possible

Colorectal FL

- Single paper



Oncology–Brief Communication

Primary Colorectal Follicular Lymphoma in 3 Dogs

Veterinary Pathology
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Abstract

Primary colorectal follicular lymphomas are rare indolent lymphoid neoplasms in humans that have not been reported in dogs. We describe 3 cases of primary colorectal follicular lymphoma in dogs with histologic and immunohistochemical features similar to their human counterpart. Initial clinical signs in all dogs included tenesmus, hematochezia, and a palpable rectal mass. Two dogs were castrated males and 1 an intact female, between 9 months and 2 years of age, and of varied breeds. All 3 cases of colorectal follicular lymphoma were characterized by proliferation of follicular germinal centers with no polarity or mantle zone and were composed of centrocytes admixed with fewer centroblasts. By immunohistochemistry, lymphoid cells expressed CD20, BCL2, and BCL6 and lacked expression of CD3, CD5, and cyclin D1. Polymerase chain reaction for rearrangements of the immunoglobulin heavy chain confirmed a monoclonal population in all cases. In 2 of the 3 cases, a solitary nodular colorectal mass was excised and appeared curative; however, the third case had multiple colorectal masses and the animal developed multicentric lymphoma. This case series immunohistochemically characterizes and distinguishes colorectal follicular lymphoma from atypical lymphoid hyperplasia.

Key points

- Indolent lymphomas make up ~30% of all canine lymphomas
- Diagnosis will likely require not only a FNA, but a combination of an astute clinical assessment, cytology, immunophenotyping, and in most cases histology
- Important to recognize these cases because they may have different approaches to treatment and differing prognoses
- Further work is needed to delineate the best treatment / management approaches

Questions?

Approach to the Trauma Patient

Dr. Liz Rozanski - Tufts University

Thoracic Trauma

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Trauma, including thoracic trauma, is common presenting complaint in small animal emergency medicine. The basic tenets of emergency medicine focus on the primary survey, including the evaluation and stabilization of the major body systems (heart, brain, lungs) and then the secondary survey with complete patient evaluation.

In the Try not to miss something category, the following tips are potentially useful.

1) **Look at the total solids,** particularly in dogs. Major trauma is a common cause significant blood loss, but this can occasionally be hard to fully appreciate due to splenic contraction which will temporarily raise the hematocrit. Specific common sites of bleeding include hemoabdomen, and retroperitoneum, and bleeding into fracture sites. Total solids of less than 6 gm/dl warrant further evaluation, especially if coupled with persistent tachycardia. Occasionally, pre-existing diseases may result in hypoproteinemia (PLE/PLN etc) but the safest approach is to assume hemorrhage.
2) **Learn Focused Assessment with Sonography for Trauma (FAST)**. Abdominal and thoracic ultrasonography is very useful for determining if there is free blood in the abdominal cavity or free blood or pneumothorax in the chest. The concept of Point of care scanning in veterinary medicine was further described by Boysen and colleagues, and since that time, multiple studies have documented its utility. Following initial patient survey, and institution of resuscitative measures, a standardized approach to a using ultrasound will help identify fluid more quickly. Most ER practices recommend a brief (< 2 hour) training seminar to cover the basics of imaging, and then a short period of a supervised experience. Point of care ultrasound involves a <u>limited use</u> of ultrasound to answer the question 'Is there fluid or not?" and is not designed to evaluate the entire cavity being scanned. The ER clinician should learn specific sites to scan and record these in the medical record. It is harder to appreciate pneumothorax than effusion. Recall that significant volumes of pleural effusion are more likely to result in signs of hypovolemia than respiratory embarrassment as pleural effusion in trauma is typically blood, although injury associated urinothorax, and bilothorax have rarely been reported.
3) **Learn to use lactate and lactate clearance** to help monitor patients. Lactate elevations in the emergency service primarily reflect tissue hypoperfusion, although other causes such as neoplasia and some drugs may also result in a type B (non-hypoperfusion related) increase in lactate. Recall that seizures or

struggles during blood collection (eg. Muscular activity; normal behavior in Jack Russell Terrorists) may result in increased lactate values. Changes in lactate, including failure to normalize, can help alert emergency personnel to dynamics in patient status. A reasonable protocol includes

a> Baseline lactate at admission- if < 2.0 mmol/l; treat as clinically warranted and monitor for evidence of hypoperfusion such as tachycardia, tachypnea or altered mentation. Recheck lactate in clinical signs suggests changes.

b> Baseline lactate at admission > 2.0 mmol/L.

c> Lactate clearance studies

4) **Chest radiographs are a really good idea. This is specifically true in thoracic trauma.** Abdominal films are unlikely to add more information than ultrasound and clinical examination unless there is evidence to the contrary (eg. Ruptured bladder suspect). Chest radiographs are of importance to look for evidence of diaphragmatic hernia, which should be promptly repaired surgically, and for the evaluation of pneumothorax and pulmonary contusion. There is occasionally debate in the literature as to the timing of the chest film, due to the idea that pulmonary contusions get worse in the first 12-24 hours as pulmonary hemorrhage recruits some inflammatory infiltrates. However, clinically, the changes in radiographic appearance are of limited significance. Dogs and cats with severe (eg.life-threatening) contusions will have marked distress shortly after injury.
5) **Remember to see if the legs work.** Spinal fractures are often associated with a poor prognosis, and long bone fractures may be very expensive to repair. It is wise to ask the client if they have seen the patient walk since the accident, if they did walk, it is much less likely there is severe orthopedic disease. Animals laterally recumbent with a Schiff-Sherington position should be quickly evaluated for neurological function in the hind limbs, as this posture is characteristic of spinal fracture. Dogs with spinal fractures with intact sensation have a good to excellent prognosis but dogs with the loss of deep pain sensation have a grave prognosis for return to function.
6) **Don't be afraid of the transfusion or transfuse early/transfuse often!** Recall in trauma that the day started out good, meaning that in the vast majority of animals with trauma, a PCV that is less than 30% is suggestive of severe loss. Thus, if you see a 3 year old lab with a PCV of 27% on presentation, this likely represents close to a 40% blood loss! The impact of fluid dilution is hard to predict. In a non-bleeding dog, the addition of 30 ml per kg of crystalloid could be expected to lower the PCV from 50 to 37% until redistribution occurs and the PCV returns to normal. If you transfuse early, you may be able to avoid playing "catch-up" later. Additionally, avoiding microvascular hypoperfusion may prevent disseminated intravascular coagulation or systemic inflammatory response syndrome. Type specific blood is required in cats, and appealing but not required in first transfusions in dogs. Plasma transfusions are ideally given at a rate of 1:1 with pRBCs, however, this may not be financially or practically

feasible. Some practices are evaluating the use of thromboelastrography in trauma patients as a marker of coagulopathy.

7) **The number one rule-out for oliguria** is inadequate volume resuscitation. While ruptured urinary tracts are possible, it is much more likely to under-estimate the fluid demands of a trauma patient in the first 8 hours. Urine production should be considered in the face of the volume of fluids given, not just in the > 2 ml/kr/hr. Hypovolemia should be excluded prior to extensive diagnostic efforts.

SPECIFIC INJURIES

8) **Slow rollovers** have a higher likelihood of ruptured bladders and diaphragmatic hernias as these injuries are more common when the pressure on the structure is slowly increased and maintained. Ruptured bladders may be diagnosed via imaging, or through evaluation of abdominal fluid. Abdominal fluid in specific will have higher potassium and creatinine values than serum. Abdominal effusion from ruptured bladders tend to cause marked pain from chemical peritonitis, seemingly more so than from straight hemoabdomens. After the presence of urine in the abdominal cavity is established, imaging studies are warranted to localize the site of the tear. Most uroabdomens result from bladder tears, but other sources of leakage are also possible, such as the urethra or the ureters. It is of course desirable to know the sources of the leakage prior to surgery, as some repairs are much more challenging than others. For diaphragmatic hernias, radiographs are often adequate to highlight the tear; but when there are not, it is more challenging to establish the diagnosis, and may include ultrasonography, contrast studies or surgical exploration.
   a) **Ruptured bladders** should be repaired when the patient is stable. There is not a benefit to delaying surgery; however, if the patient is unstable (hyperkalemic or shocky) simple placement of an abdominal drainage catheter is often very successful in controlling the electrolyte disturbances and uremia. Exchanges are not required but can be perused if desired.
   b) **Diaphragmatic hernia** should be repaired in a timely fashion. Older literature suggested to wait 24 hours until surgery, however, this was based on retrospective data that was flawed as dogs that were more severely injured often went to surgery urgently, and then died, so that non-survival was a side effect of severity of injury, not the timing of surgery.

9) **Degloving wounds looks bad, but most heal well.** Animals often have rapid healing in response very severe loss of skin and soft tissue. Hocks in particular with often look horrid; but will heal with bandage changes, often not requiring surgical stabilization (particularly in cats). Following cardiovascular stabilization, the patient should be anesthetized and the wound completely cleaned of debris and dead tissues. Wet to dry bandages should be applied until there is a clear tissue bed – recall that the role of the wet to dry bandage is to pull debris off with each bandage change. Therefore, when the wound is clean and beginning to granulate, a non-adherent bandage should be applied. The frequency of bandage change should be daily at first, then may be extended to every 2 or 3rd

day. Bandage material can be pricy, and often time-consuming to replace the bandages, but wounds do tend to heal very well. It is important to ward off the urge to close wound that will be under "just a little" tension; these will invariably break down, and be more frustrating and expensive to treat. Antimicrobials are indicated until the tissue bed is healthy.

10) **It is hard to keep a young cat from healing.** Pelvic and distal limb fractures will heal with cage result. Orthopedic injures are often most appropriately treated with surgical fixation. However, in many cases, rest with result in a very functional outcome. Cats are MUCH less likely than dogs to suffer thoracic trauma and to subsequently survive to reach the hospital.

One concept that is well worth implementing is protocol-driven resuscitation. This involves creating a standing list of orders for each trauma patient, and to mandate that each step (goal) is reached prior to patient discharge. Veterinarians as a group tend to prefer to be independent; however, emergency practices are often staffed by newer or less experienced clinicians and technicians. However, even experienced clinicians are prone to fatigue, distraction and complacency. Minimizing errors is a vital step in improving patient care and has been shown to be life-saving and fiscally wise in human medicine. The role of specific protocols in emergency medicine remains to be determined. However, a simple option might be as follows:

Proposed Trauma protocol

1) Alert senior clinician and technician of impending arrival and ETA if known. If possible, have ready area prepared

Arrival

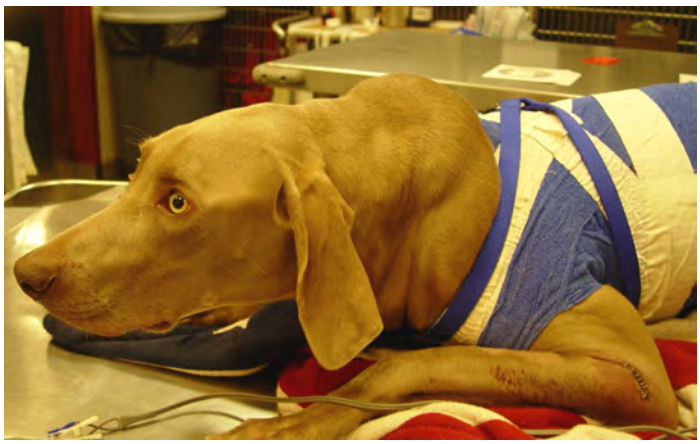
a) Immediate primary survey- heart, brain, lungs, obvious fractures/degloving. Place continuous EKG
b) Place 1-2 large bore IV catheters. Begin IV fluids at 20 ml/kg (crystalloids).
c) Collect samples for point of care testing- PCV/TS/Dextrose/Azo/Lactate at minimum, ideally to include more extensive testing.
d) If solids < 6 gm/dl (60 gm/l) actively look for hemorrhage; perform FAST exam of abdomen and chest, look for fluid, and pneumothorax.
e) Continue fluid resuscitation until HR < 140 bpm.
f) Titrate analgesics (pure opoids) to provide adequate pain relief.

Try not to make anything worse

The major ways to make trauma worse are 1) over-zealous use of fluids 2) failure to look adequately for all injuries/make sure they make sense and 3) Inappropriate repair of injuries or use of medications

Fluid therapy is critical for adequate recovery from major injuries. It is essential to balance out intravascular volume requirements with the potential to create a dilutional coagulopathy and to have fluids extravascate. a) Early recommendation of "shock" dose fluids are currently considered flawed, as dilutional coagulopathy is a considered a real

risk. Additionally, temporarily increasing intravascular volume may "pop" off the clot of a major bleed, resulting in hemorrhage and subsequent inability to clot. From a lung perspective, extravasated fluid affects gas exchange and may contribute to hypoxemia and to pulmonary failure. b) recall that animal should walk with most injuries. If you detect a femoral fracture, but the dog will not walk on the other 3 legs, there may also be a pelvic fracture. If the front foot is dragging and the dog can't position it correctly, it is unlikely the buprenorphine he got 3 hours ago. The figure below showed a HBC dog with a temporary tracheostomy that was closed after the tracheostomy was removed. Note the SQ emphysema. c) NSAIDs, steroids and furosemide are excellent medications, but have little to no role in the acutely injured patient in whom cardiovascular stability can't be assured.



SPECIFIC RECOMMENDATIONS ON THORACIC TRAUMA

Pneumothorax refers to the development of free air within the pleural space. The air gets to the pleural space either from the outside or via air leakage from the pulmonary parenchyma. Radiography, thoracocentesis or auscultation may identify pneumothorax. Auscultation of dogs with pneumothorax may be misleading if respiratory sounds are louder than average. In many emergency practices, dogs showing respiratory distress may undergo thoracocentesis based upon trauma history and increased respiratory effort. Approximately 25-30 ml/kg of air generally needs to be removed to provide significant improvement to respiratory status. Occasionally, a thoracostomy tube is required to prevent either continuous or intermittent chest drainage. Generally, a dog is considered a candidate for a chest tube if it requires greater than three needle thoracocentesis in less than 12- 18 hours or if no end-point is reached during thoracocentesis.

Animals will breathe with a restrictive pattern (short shallow breaths).

Pulmonary contusion is another common traumatic thoracic injury. Pulmonary contusion occurs when blunt trauma to the chest causes alveoli to fill with blood and

fluid (inflammation). Pulmonary contusion occurs in a large percentage of animals with thoracic trauma. Contusion may be identified radiographically as interstitial to alveolar infiltrates or clinically by tachypnea/increased respiratory effort in dogs following trauma. Therapy for pulmonary contusion is supportive and includes oxygen and fluid therapy as needed to maintain adequate circulating volume. Some clinicians vividly recall dogs with pulmonary contusion that appeared to rapidly deteriorate following a large volume of intravenous crystalloids. Most dogs with pulmonary contusion improve significantly in 2-3 days and recover completely in less than one week.

Hemothorax is another common sequalae of thoracic trauma. The impact of hemothorax is more likely from hypovolemia from the blood loss, than from the pleural effusion. Hemothorax is usually a presumptive diagnosis after identification of pleural effusion on chest radiographs from a trauma patient. Treatment is supportive. Thoracocentesis is avoided unless otherwise indicated. Surgical exploration is a last resort.

Rib fractures are also common in the patient with thoracic trauma. Rib fractures appear to be painful, particularly on inspiration. Individual fractured ribs do not themselves typically affect lung function, but reflect a severe injury to the chest. Therapy for rib fractures typically is conservative and includes pain management (opoids and local blocks). Some clinicians advocate loosely applied support bandages. If multiple ribs are fractured at several sites, an unstable piece or flail segment may be formed. This “flail” segment moves paradoxically with respiration. Various methods of stabilization have been described; however, frequently the underlying contusions may actually be more detrimental to lung function.

Diaphragmatic hernias may also occur in animals with significant chest injuries. The muscular portion of the diaphragm is the area most frequently torn. In general, animals with traumatic diaphragmatic hernias have other significant intrathoracic injuries try to better clarify the margins of the diaphragm. The timing of surgery may be equally important in successful patient outcome. Surgery should be undertaken when the patient is cardiovascularly stable. This should be within 12-24 hours of the injury. However, if the stomach is in the chest cavity, this is a surgical emergency because the stomach may distend with air and severely compromise ventilation. In general, in the patient with an acute diaphragmatic hernia, associated injuries may also play a significant role in deciding the time of surgery. Remember that surgery and anesthesia (and the recovery period) may be stressful to the critically injured dog. Safe anesthesia requires a rapid intubation and positive pressure ventilation from the time of entry into the abdominal cavity until the integrity of the diaphragm is restored. All efforts should be made to limit anesthesia and surgery time.

Post-operative care usually involves standard attention to adequate intravascular volume, oxygen supplementation and pain relief (local and opoids). Most dogs with acute traumatic diaphragmatic hernias recover well from surgery, but the adequate monitoring and support in the post-operative period is critical.

Cardiac arrhythmia are common following trauma in dogs and are generally self-limiting. However, severe tachycardias may occasionally require therapy (such as

lidocaine). Animals having sustained severe trauma should be monitored for cardiac complicatons as well.

Most dogs with traumatic thoracic injuries recover uneventfully from their injuries with no lasting complications. The standard course is for the patient to look the worse for the first 24 hours after presentation and then to make relatively rapid recovery. Successful management includes appropriate identification of injuries and well-timed interventions.

Respiratory Distress Cases

Dr. Liz Rozanski - Tufts University

Respiratory Distress

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Respiratory distress of any origin represents a true emergency as rapid treatment is warranted to identify the underlying cause, to limit the sensation of difficulty breathing and to provide diagnostic and therapeutic information for clients of affected animals. Clearly, there are a variety of potential underlying reasons for the development of respiratory distress. For the clinician in emergency practice, success in patient management revolves around developing a knowledge base of potential causes of respiratory distress and "pattern recognition" of common emergent problems affecting dogs and cats. The goals of this article is to review the function of the respiratory system, to describe pathophysiological causes for hypoxemia, to illustrate various methods for classifying respiratory distress, to highlight common emergency conditions resulting in respiratory distress and to provide guidelines for emergent management.

As a review, the primary goal of the respiratory system is to promote gas exchange. Air enters the upper respiratory system via either the nose or the mouth. The air is warmed and humidified and then passes down the respiratory tree. Larger particles of debris are filtered out. The trachea branches into successive generation of smaller and smaller airways. Gas exchange occurs at the level of the alveoli. While the flow of air (Ventilation-V) is obviously essential, the other aspect of gas exchange involves the delivery of blood (perfusion-Q) to the level of the alveoli. Blood makes it way to the capillaries adjacent to the alveoli by way of the pulmonary vasculature. The blood leaves the right heart via the pulmonary outflow tract and then flows through the pulmonary arteries. The arteries branch into smaller and smaller vessels until the level of the capillary. Red cells go through the capillaries in single file and during their time in contact with the alveoli, the oxygen diffuses out of the alveoli and binds to the hemoglobin as the CO2 (waste gas) will diffuse off the hemoglobin molecule into the alveoli for expiration. In normal animals, the capillary endothelium is very impermeable to larger molecules such as albumin.

In animals that develop hypoxemia there are five possible broad causes. Each disease observed clinically will fit into one of more of the following categories. These causes include a low inspired oxygen concentration, hypoventilation, shunt, V-Q mismatch and diffusion impairment. Normal oxygen concentration in room air is 21%, thus a low inspired oxygen concentration (Termed FiO2) is very uncommon. However, this may occur at higher altitudes or due to an anesthesia machine dysfunction. Low FiO2 may be corrected by giving a higher concentration of oxygen. Hypoventilation results from absent or ineffective (low) tidal volumes due to causes such as drug therapy (opoids), or loss of central respiratory drive. Hypoventilation is treated by either mechanical ventilation or by reversing the cause that triggered the hypoventilation. Importantly, hypoventilation may not be appreciated as respiratory distress as respiratory attempts are limited. Shunt is a term that refers to the complete by-passing of the lungs by the unoxygenated blood. The classic example of a shunt is the right to left shunt that accompanies the cardiac defect of Tetralogy of Fallot (right ventricular hypertrophy, ventricular septal defect, pulmonary stenosis and over-riding aorta). In these animals, the severity of the PS results in the shunting of the un-oxygenated blood into the left ventricle. Pure shunts will not respond to oxygen supplementation. V-Q mismatch means that there is poor coordination of the areas of the lung that are getting perfused versus those that are getting ventilated. Normally, there is a good match between areas that get good air flow versus those that get good perfusion. However, in some cases due to inflammatory products (eg pulmonary edema, pneumonia) areas of the lungs receive blood flow but no ventilation, while others receive ventilation but no blood flow. This mismatching results in the development of low blood oxygen. V-Q mismatch will respond to oxygen supplementation. The final area that may result in hypoxemia is diffusion impairment. Typically during transition phase through the capillary bed, the oxygen diffuses within the first third of the length of the capillary. However if the alveolar-capillary membrane is very thickened, there may be diffusion impairment.

While an understanding of potential causes of respiratory distress are mandatory, the first steps in the clinical evaluation of a patient presenting with respiratory distress are to provide a supplemental source of oxygen and to obtain a brief history from the client. All emergency facilities should have a form of supplemental oxygen available. Supplemental oxygen may be provided via a variety of options, including flow-by, face mask, nasal oxygen, e-collar and cellophane wrap ("oxygen hood"), oxygen cage and intubation with intermittent positive pressure ventilation (IPPV). Flow-by oxygen is provided by holding oxygen tubing near the mouth and nostrils of the affected patient. The flow rate is usually set at 100ml/kg/minute or greater. Flow-by oxygen is an easy and rapid solution; however, the actual increase over room air's 21% oxygen content may be minimal, particularly with an anxious or uncooperative pet. Oxygen may also be provided with a face mask, with the oxygen tubing attached to a cone that is placed over the nose and mouth of the

patient. The FiO_2 with a face mask is also variable, although with very weak animals a high percentage may be reached. Both flow-by and face mask oxygen may require veterinary personnel to hold both the pet and the oxygen supply. Nasal oxygen involves placement of flexible catheter into the nasal passages and insufflation of humidified oxygen. Nasal oxygen is particularly useful in pets that are not either panting or open-mouth breathing. Nasal oxygen is commonly placed after patient stabilization, rather than urgently in the emergency setting. A home-made oxygen hood may be created with an Elizabethan Collar and cellophane wrap, or may be commercially purchased (Jorgenson Laboratories). An oxygen cage is also frequently used to provide supplemental oxygen. Oxygen cages are commonly well-tolerated by both cats and dogs, and are also capable of reaching high concentration of oxygen, however, if the cage door is open for patient manipulation, the FiO_2 will rapidly fall. Finally, intubation and IPPV is the best option for providing high levels of supplemental oxygen, removing respiratory fatigue, and eliminating patient fear and anxiety.

During the initial stabilization of the pet, a history should be obtained from the owner. In some cases, the precipitating case of the respiratory distress is straight-forward, such as with traumatic injuries, while in other cases, the onset may be more insidious. Animals with pre-existing medical conditions, such as cardiac disease, neoplasia or megaesophagus may also be predisposed to the development of respiratory distress. Owners should be questioned as to past medical conditions, history of routine veterinary care, including heartworm prophylaxis, and finally, the progression of the signs of respiratory distress should be described. Specifically, distress may be acute in onset, or more progressive. In cats in particular, the development of respiratory distress may be preceded by anorexia, lethargy or abnormal behavior.

Respiratory distress may be further characterized by the location of the lesion or the underlying pathophysiological condition. Often, localization of the lesion can help to guide the clinician to the most likely cause. Specifically, respiratory distress may be localized to upper airway, lower airway, parenchymal or pleural space disease. Common pathophysiological causes for respiratory distress include anatomical abnormalities, airway collapse, pulmonary edema of cardiac and non-cardiac causes, infection, inflammatory and trauma. For the emergency clinician, the most appropriate first step is to localize the lesion and then to review specific differentials based upon signalment, history and other physical examination findings.

Upper airway diseases may be appreciated by loud stridorous breathing, with an increased inspiratory time. Many dogs are hyperthermic on initial presentation due to decreased ability to cool. The upper airways represent the primary source of resistance to airflow. Upper airway obstructions can be either dynamic or fixed. Dynamic obstructions are characterized by the paradoxical movement of tissues into the lumen of airway during inspiratory inspiration. Common dynamic obstructions include laryngeal paralysis and tracheal collapse, while fixed obstruction include extraluminal obstructions such as neoplasia or cellulitis and intraluminal obstructions such as laryngeal tumors or nasopharyngeal polyps. Both dynamic and fixed severe upper airway obstructions will also result in the development of airway mucosal edema and possibly everted laryngeal saccules due to irritation from the increased air flow rates through a narrow lumen.

Emergently, upper airway obstruction should be suspected in a dog with loud, noisy breathing. Therapy for a suspected dynamic obstruction should include sedation and supplemental oxygen. Sedation is beneficial in the dynamic obstruction in reducing the anxiety associated with inspiration because with increased inspiratory efforts there is a resulting paradoxical decline in airway diameter. Low doses of acepromazine (0.03-0.05 mg.kg intravenously) alone or in combination with butorphanol (0.1 mg/kg intravenously) are often effective. Hyperthermia should be treated by active cooling with room temperature (not cold!) intravenous fluids, and by placing the dog in a cool area. Due to airway swelling and edema, a single dose of short acting anti-inflammatory glucocorticoid is advisable. If the dog has not improved within 15-30 minutes or if distress is worsening, more aggressive therapy is warranted. The dog should be heavily sedated or anesthetized and intubated. The emergency clinician should be competent to evaluate airway function and anatomy and to perform a tracheostomy if needed. Additionally, as many upper airway conditions require management and/or surgical intervention, the emergency clinician should be fluent in discussions with clients concerning long-term outcomes. The most common causes of upper airway obstruction may vary depending on location but in our practice include laryngeal paralysis, tracheal collapse, brachycephalic airway syndrome and severe cellulitis. While a complete discussion on the management of these conditions is beyond the scope of this chapter; however, despite their similarities, some differences due to the underlying disease do exist concerning optimal management of affected patients.

Laryngeal paralysis primarily affected older large breed dogs, particularly retrievers. Usually, the clinical signs of noisy breathing have been present for some length of time prior to a crisis. Crises often occur during the first hot and humid days of the spring or summer. Dogs will commonly respond well to sedation. Dogs that do not rapidly improve should be sedated and have laryngeal function evaluated and be intubated. If palliative surgery is not readily available, dogs may have a tracheostomy performed or may be keep briefly

sedated/intubated until normothermia and eupnea ensue. In our practice, we will commonly maintain dogs on a propofol CRI in 0.9% saline (20 ml of propofol in 1 liter of NaCl) titrated to effect for 30-60 minutes. If after this time period a dog can not be extubated, due to the higher risk of aspiration pneumonia, it is better to perform a tracheostomy than keep a patient intubated.

Conversely, in dogs with severe brachycephalic airway syndrome or tracheal collapse, it may be impossible to remove a tracheostomy tube after placement. This means that it avoidance of a tracheostomy is preferable in these dogs as compared with dogs with laryngeal paralysis. If a tracheostomy is unavoidable; plans should be made for surgical correction of the obstruction as soon as feasible. Brachycephalic dogs may also develop laryngeal collapse, which is not amenable to laryngoplasty, and may ultimately necessitate a permanent tracheostomy.

In cats, upper airway obstructions are less common, but may be caused by nasopharyngeal polyps or infiltrative laryngeal diseases (neoplasia or granulomatous). Occasionally cats with severe pleural effusion will have the appearance of severe inspiratory distress. If sedation for an oral examination for a cat with a suspected upper airway obstruction is planned, then supplies should be collected ahead of time for an emergency tracheostomy. The laryngeal lumen of affected cats can be only a millimeter or two in diameter and may require an urgent tracheostomy. If a biopsy of a laryngeal mass is performed in a cat, a tracheostomy is almost always required due to subsequent airway swelling. Cats may also have a permanent tracheostomy placed, although it less well tolerated than it is in dogs. (Fig Arnie)

Respiratory distress may also result from lower airway disease, parenchymal lung disease or pleural space disease. Thoracic radiographs are essential to help clarify the degree of pulmonary or pleural space involvement. However, it is important to recall that radiography can be stressful, particularly in cats that are experiencing respiratory distress. Lower airway diseases include chronic bronchitis and feline asthma. Chronic bronchitis rarely presents emergently, although flare-ups in some patients do occur. Chronic bronchitis is defined as the presence of a cough on most days for the preceding two months, without evidence of other underlying cause. Canine chronic bronchitis commonly affects small breed dogs. On auscultation, a mitral murmur is commonly heard. Conversely, feline lower airway disease may present as an emergency. In cats, airway disease appears to represent a continuum with some cats having primarily inflammatory airway disease with cough and excessive mucus production, while other cats are the more prototypical “asthmatics” with reversible bronchoconstriction. Cats with severe bronchoconstriction will often present emergently. It is important to distinguish the airway disease from congestive heart failure. Cats with airway disease typically are normothermic and have had a history of cough. Both heart failure and airway disease may be accompanied by crackles.

Parenchymal lung disease is often responsible for respiratory distress. Common causes of parenchymal lung disease include pulmonary edema (cardiogenic and non-cardiogenic), pulmonary contusion, pneumonia and neoplasia. Heart failure in cats is usually appreciated by hypothermia combined with an increased respiratory rate and effort. Jugular venous distension may be present. A gallop or a murmur may be ausculted. Due to the hypothermia, cats with congestive heart failure (CHF) will commonly have slow heart rates (130-140 bpm). Heart disease in dogs is usually either chronic valvular disease or dilated cardiomyopathy. Animals with a history of trauma or possible trauma and that are presenting with respiratory distress can be assumed to have some component of pulmonary contusion (and/or pneumothorax). Therapy for respiratory distress associated with pulmonary infiltrates include supplemental oxygen and therapeutic agents directed towards the presumptive underlying cause. The distribution of the pulmonary infiltrates may be very useful to help determine the underlying problem. In dogs, cardiogenic pulmonary infiltrates will most often surround the perihilar region, while in cats the distribution of pulmonary edema may vary. Bacterial pneumonias will typically have a cranioventral distribution. Neoplasia will usually result in a nodular pattern, although metastatic disease may appear variable.

Animals with suspected cardiogenic pulmonary edema should be treated with initially with diuretics (furosemide 1-4 mg/kg iv or im, q 1-6 hours), cage rest and supplemental oxygen. If a rapid improvement is not observed, additional therapy with vasodilators (nitroprusside titrated to effect) is warranted. In practice, despite published guidelines, measurement of blood pressure during infusion of nitroprusside is usually not performed in order to limit patient stress, loss of supplemental oxygen (by opening cage door) and technical difficulties in getting accurate numbers. Specifically, it may be challenging or impossible to place an arterial line for direct blood pressure determinations in an animal with congestive heart failure, oscillometric techniques are commonly inaccurate with small dogs or cats or during ectopy and Doppler techniques are time-consuming and require a patient with respiratory distress to be restrained. Dobutamine, as a continuous rate infusion (CRI) is very useful in dogs with dilated cardiomyopathies. Intravenous fluids should not be administered to a patient with heart failure, although they should be permitted *ad lib* access to water.

Hemodynamically significant arrhythmias should be treated. Patients should be transitioned to long-term medications after stabilization. While an echocardiogram is not considered an emergent procedure, it is useful for if an emergency clinician has access to an ultrasound machine for that individual to gain basic knowledge of echocardiography, including assessment of left atrial size, contractility and presence or absence of pericardial effusion.

Non-cardiogenic pulmonary edema may occur for a variety of reasons. In the emergency room; upper airway obstruction, seizures and electric cord injury are common triggers for the development of non-cardiogenic edema. Non-cardiogenic pulmonary edema is typically high-protein and results due to permeability shifts in the capillaries rather than hydrostatic forces as with cardiogenic edema. There is not specific therapy that has been proven beneficial for hastening recovery from non-cardiogenic edema. Treatment recommendations include cage rest and supplemental oxygen. More specific therapy with either diuretics or colloids has been advocated by various clinicians although no consensus statement exists. The vast majority of dogs with non-cardiogenic pulmonary edema will rapidly improve within 24-48 hours.

Pulmonary contusions are common after traumatic injury, particularly in dogs. Animals severe affected with pulmonary contusion will be short of breath rapidly after the injury, although radiographically infiltrates will often worsen over the first 12-24 hours. Dogs with contusions commonly have small to moderate volume pneumothoraces as well. Contusions will generally heal rapidly. One study in dogs was unable to support the use of either prophylactic antibiotics or steroids. (powell JVECCS) Diuretics are also not indicated for animals with pulmonary contusion.

Dogs with pneumonia may present to the emergency room with respiratory distress. Bacterial pneumonia is very rare in cats. Pneumonia can be sub-divided in to community-acquired such as severe bronchopneumonia (infectious kennel cough complex) or aspiration pneumonia in a dog with laryngeal paralysis or megaesophagus or hospital-acquired for a dog that develops pneumonia while hospitalized for treatment of another condition. Therapy for pneumonia includes broad-spectrum antibiotics, physiotherapy and intravenous fluids. Ideally, a bacterial culture is performed prior to the institution of antibiotics.

Animals will infrequently present on emergency with dyspnea due to metastatic disease, although cough and lethargy are common. Spontaneous pneumothorax may occasionally develop in a patient with pulmonary neoplasia. Treatment of suspected neoplastic disease is directed at supportive care. Common metastatic tumors include hemangiosarcoma and mammary gland adenocarcinoma. Occasionally, further imaging is indicated to attempt to localize a primary tumor; however, generally this is futile. Pulmonary lymphoma may respond well to therapy. It is also important to exclude a recent travel history in dogs with a nodular pulmonary pattern as the systemic mycoses can mimic metastatic disease. Other less common causes of pulmonary infiltrates include eosinophilic pneumonitis and pulmonary fibrosis.

Pleural space disease will commonly result in marked respiratory distress. Common causes are pleural effusion, pneumothorax and diaphragmatic hernia. Pleural space disease may often be suspected clinically based upon a restrictive (short and shallow) breathing pattern. Thoracic radiographs are very useful in documenting the extent of the pleural space disease. Diaphragmatic hernia should be corrected as soon as the patient is considered stable enough for surgery. In traumatic injuries, concurrent pulmonary contusion may markedly worsen gas exchange, thus anesthesia and surgery may be postponed until clinical improvement. However, if significant herniation exists, including the presence of the stomach intra-thoracically, surgical repair become urgent. Anesthesia may still be safely performed with pulmonary contusions, although in addition to the positive pressure ventilation required due to the loss of diaphragmatic integrity, a small amount of positive end-expiratory pressure (PEEP) may be beneficial to help recruit collapsed alveoli. In chronic hernias, re-expansion pulmonary edema may result in severe respiratory failure. Thus correction of chronic hernias should be undertaken with care and gradual re-inflation of the lung.

Pneumothorax may be classified as either traumatic or spontaneous. Traumatic is the most common. For animals with a known history of injury, needle thoracocentesis may be performed as guided clinically. Due to the high density of tissue thromboplastin, the previously healthy injured lung will heal rapidly, thus chest tubes are not commonly required in the trauma patient. A good guideline is that three or more thoracocenteses (“Three strike rule”) within 24 hours is sufficient justification for placement of the chest tube in the traumatic pneumothorax. It is exceedingly rare to have a patient with trauma require a thoracotomy for resection of the traumatized lung. Conversely, most cases of spontaneous pneumothorax require surgical resection of the affected lobe. Spontaneous pneumothorax is defined as pneumothorax occurring without trauma. Common causes include bulla/blebs and neoplasia (primary or metastatic). Additionally, cats with lower airway disease may occasionally develop spontaneous pneumothoraces. (White) For affected dogs, rapid surgical exploration and resection has been associated with decreased morbidity and expense. (Puerto)

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Pediatric Dentistry

Dr. Douglas Winter

Pediatric Dentistry

Overview

This presentation will focus on the most common pediatric dental and oral conditions encountered in a puppy and kitten during the first 12 mont. During the time of development of the oral cavity, there are many problems that can occur which may lead to significant pain and disease long term. As with most other oral conditions, our patients rarely show outward signs of pain and disease. It is the responsibility of the veterinarian and support team to recognize normal versus abnormal development, their significance, and be able to understand that appropriate treatment is necessary.

Objectives

- Review normal anatomy of the puppy and kitten.
- Understand eruption dates and when to intervene with treatment
- Review non-occlusal pathology (microglossia, cleft palates, etc.)
- Review of malocclusions (deciduous, permanent) and treatment options and timing.
- Address fractured primary and permanent teeth.

Discussion

Review oral anatomy of the puppy and kitten.

Every patient should have a thorough oral exam with each visit. It is essential to know the deciduous and permanent dental formulas for the dog and cat as well as approximate eruption times of all teeth.

Dental formulas:

Dog

- Deciduous (primary): 2 (3/3 i, 1/1 c, 3/3 pm) = 28
- Permanent (secondary): 2 (3/3 I, 1/1 C, 4/4 PM, 2 /3 M) = 42

Cat

- Deciduous (primary): 2 (3/3 i, 1/1 c, 3/2 pm) = 26
- Permanent (secondary): 2 (3/3 I, 1/1 C, 3/2 PM, 1/1 M) = 30

Eruption times:

Dog

- Deciduous (primary)(weeks): i 3 -4, c 3, pm 4 -12
- Permanent (secondary)(months): I 3 -5, C 4 -6, PM 4 -6, M 5 -7

Cat

- Deciduous (primary)(weeks): i 2 -3, c 3 -4, pm 3 -6
- Permanent (secondary)(months): I 3 -4, C 4 -5, PM 4 -6, M 4 -5

There are no deciduous (primary) molars.
The deciduous (primary) fourth premolar functions as the mandibular first molar.
At 8 weeks of age, most primary teeth should be erupted and all visible by 12 weeks of age.

1. Newborn exam (first few weeks)

Microglossia (small tongue)
This is an uncommon, hereditary and lethal defect of the tongue. Puppies are not able to nurse appropriately due to an abnormally small tongue and lack a normal swallowing reflex. This is a complex multi-system birth defect (autosomal recessive) with abnormalities noted in the tongue, pharynx, musculoskeletal system and the brain. Euthanasia is recommended and parents should not be used for breeding.

Cleft palates (primary or secondary)

Clefts of the primary palates (harelip) are rostral to the incisive foramen and including the lips. They can be unilateral (left) or bilateral. Depending on the severity, they may only be cosmetic causing no problems with nursing or respiration. **Clefts of the secondary palate (hard palate caudal to the incisive foramen usually involving the soft palate)** are most often on the midline and usually involve the soft palate. These clefts result in a direct communication between the oral and nasal cavities. Affected animals are at great risk of developing aspiration pneumonia. The prognosis is guarded without surgical correction of the cleft to reestablish a functional separation between oral and nasal cavities. With successful closure of the defects, the prognosis is excellent. Ideally, secondary clefts are repaired about 12 weeks of age. Cleft palates are excellent cases to refer due to high risk of complications and difficulty of subsequent procedures being successful. Clefts of the soft palate may be midline, unilateral or bilateral. Absence of the soft palate carries a hopeless prognosis as the pharyngeal sphincter is incomplete. As this is considered congenital with possibly genetic involvement, affected individuals should be taken out of the breeding pool.

2. First and second wellness visits (8 and 12 weeks)

Delayed eruption of primary (deciduous) teeth.
Most primary (deciduous) teeth should be erupted and all visible between 8 -12 weeks of age. Primary teeth that fail to erupt are in most cases impacted below dense, fibrous gingival tissue. This is called soft tissue impaction. Small breed dogs seem particularly prone to this condition. If the deciduous teeth fail to erupt, there may be insufficient room within the mandible and maxilla for normal development of the permanent teeth. After dental radiographs, an operculectomy is performed to allow normal eruption. Remember, molars and first premolars have no primary precursors. Closely monitor these permanent teeth, as they may also have soft tissue impaction. Lastly, if a primary tooth is congenitally missing, then the permanent tooth will also be absent.

Review of malocclusions
Ideal or normal occlusion (MAL0) can be described as perfect interdigitation of the upper and

lower teeth. The ideal relationship with the mouth closed can be defined by the following: The maxillary incisors should just slightly overlap the mandibular incisors. The mandibular canine tooth should be placed between the maxillary lateral incisor and the maxillary canine tooth. The crown cusps of the mandibular premolar teeth bisect the interproximal (interdental) spaces rostral to the corresponding maxillary premolar teeth.

Up to day 50, mandibular growth in length is from the rostral mandible (Hennet, Harvey 1992). After day 50, mandibular length is from the caudal mandible (mandibular ramus). This means that there is no change in mandibular length between the incisors to the first molars between 3 and 6 months. As the growth of the mandible and maxilla are under separate genetic control, the growth of one only influences the growth of the other. Their growth is influenced by dental interdigitation. As the maxilla grows, it pulls the mandible forward. The upper canine pushes the lower canine forward. As the mandible grows, it pushes the maxilla forward with the lower incisors pushing the upper incisors. This relationship is maintained into adulthood. Dentition affects jaw movement. If the patient is genetically programmed for a normal bite (MAL 0) but mechanical impediments (abnormal dental interlock) exist, then the jaw cannot grow.

Class 1 Malocclusion (MAL1) – normal relationship of maxilla/mandible but 1 or more teeth out of position (dental malocclusion)
Class 2 Malocclusion (MAL2) - (mandibular distoclusion)– mandible is short in relation to the maxilla (skeletal malocclusion)
Class 3 Malocclusion (Mal3) - (mandibular mesioclusion)– mandible is long in relation to the maxilla (skeletal malocclusion)

Malocclusion of the primary (deciduous) dentition
Malocclusion of the primary dentition are fairly common and are likely to occur in the adult dentition as well. Treating malocclusion of the primary dentition is to alleviate oral pain and abnormal/adverse dental interlock to allow the shorter jaw to continue growing if genetically programmed. By surgically extracting the teeth causing the dental interlock, the primary canines or primary canines and incisors, in the shorter jaw or the jaw that needs to grow. This is called interceptive orthodontics. Interceptive orthodontics should be performed as soon as possible (6-8 weeks) for maximum effect. For example in a class 1 or class 2 malocclusion, the patient may have trauma to the palatal mucosa from the mandibular deciduous canine(s). The extractions will alleviate this discomfort as well as allow the jaw to reach its full genetic potential. Then the puppy or kitten needs to be evaluated for malocclusions of the adult dentition. Early recognition and proper therapy is essential to proper outcomes.

Fractured primary (deciduous) teeth
Primary teeth are long and thin when compared to permanent teeth making them more susceptible to wear and fracture. Since our juvenile patients love to chew these combined facts make tooth fracture, with pulp exposure, a common occurrence. These teeth are painful; ignoring them is NOT an option. Careful and immediate surgical extraction is required. Do not wait until neutering to remove them as they are painful to the patient and may adversely affect the developing permanent tooth. Some general comments about primary tooth extraction: 1) Always take pre and post extraction dental radiographs. 2) Primary teeth are long (20% crown, 80% root). 3) The permanent successors must can be seriously damaged with instruments due to poor

technique. 4) Use appropriately sized (small and delicate) elevators and forceps.

3. Third wellness visit (16 weeks)

Persistent deciduous teeth
This problem can occur in any sized dog, but most often associated with toy breed dogs. It can be seen occasionally in cats. If the deciduous tooth is still in place, it should be removed as soon as possible. The most common primary teeth that are persistent are the canines followed by the incisors and then premolars. Leaving a persistent deciduous tooth in place until six months (spay/neuter time) is inappropriate predisposing the tooth to premature periodontal disease and forcing the permanent tooth to erupt into an abnormal location causing a malocclusion.

RULE: Two teeth of the same type (primary and secondary) should occupy the same spot at the same time.

NOTE: There is a lot of development time between 16 weeks and the spay/neuter visit at 6-months. A lot of changes occur during time. Ideally, seeing the puppy/kitten at five and six months to check for any developmental abnormalities (persistent deciduous teeth, embedded teeth, and malocclusions) so they can be diagnosed and treated in a timely manner.

4. Fourth wellness visit, spay/neuter (6-month) With the animal being under general anesthesia, this allows for a good oral exam under anesthesia to look for the following.

Embedded teeth
Missing teeth, especially the mandibular PM1 (305 and 405), should be documented with dental radiographs. If the radiograph reveals an unerupted (embedded) permanent tooth, this tooth should be surgically extracted or referred pending the location and comfort of the veterinarian. Failure to not extract an embedded tooth will often result in the formation of a dentigerous cyst. Over time these cysts well expand and damage adjacent teeth and possible jaw fracture.

Soft tissue impaction
Permanent teeth, just as deciduous, can become impacted below the thick and dense soft tissue. Teeth with no deciduous precursor, first premolars and molars, are more likely to have soft tissue impaction. Following dental radiographs, operculectomy surgery is done.

Dental crowding
Brachycephalic and small breeds suffer from severe crowding and rotation of teeth which lead to early periodontal disease and tooth loss. Strategic extractions can relieve crowding improve periodontal health.

Malocclusions
The key time to correct any malocclusion of the permanent dentition is between 5-7 months. The treatment varies on the type of malocclusion, but may include selective extractions, orthodontic movement, or crown reduction (coronectomy) with vital pulp therapy. The goal is to make the

puppy's occlusion functional and pain-free. Case-based examples will be discussed in the presentation.

6. Other

Maxillofacial trauma
Dog fights are the most common cause followed by HBC. Case-based examples will be discussed in the presentation.

Electric cord trauma
The severity of injury is dependent on the type of circuit, voltage, amperage and duration of contact, tissue resistance, tissue relationship with the current. Most dogs and cats present with low-voltage (<1000 V) injuries from alternating household current. The heart and respiratory centers are more sensitive to alternating electrical current. Diagnosis and treatment for cardiovascular and pulmonary injuries are outside of the scope of this presentation. Oral burns due to low-voltage involve the lips (commissure), gums, tongue, and palate. Burns present as pale yellow or tan to gray. Mild injuries will slough and heal in 3 weeks without the need for surgical debridement and repair. Oronasal fistulas will require closure when tissues are healthy to make mucosal flaps. Teeth in the young patient need to be monitored clinically and with dental radiographs to ensure that the teeth in the area of the burn remain vital. It is important to have patience to ensure the severity of wound is apparent before surgically deriding and closing the wound(s). The excellent blood supply of the oral cavity can allow for some significant healing, given a chance. Major thermal injures may require multiple or staged surgeries.

Oral mass

Oral papillary squamous cell carcinoma (OPSCC) occurs in young dogs. Tumors appear locally aggressive with no reports of metastasis. Recent study shows that OPSCC occurs in older dogs also. Wide surgical resection with clean margins is usually curative.

Canine oral papillomatosis (COPV) are single, but most often multiple cauliflower-like growths. Outbreaks of COPV usually occur in groups of dogs, affecting up to 25% and is species specific. To date there are 15 types of canines papilloma virus. Most oral warts last about 4-6 weeks and then totally regress in an additional 4 weeks. When warts are noted on the footpad(s) (CPV2), lesions can persist for up to 6 months. Lesions rarely cause clinical concern and require no treatment. Because the canine papillomavirus is non-enveloped, it is fairly stable in the environment.

Odontoma is a space-occupying benign tumor or harmartoma (normal dental tissue in an abnormal location) in young dogs and cats usually appearing around 6-18 months. There are two types that distinct radiographic appearance: 1) Complex odontoma - sharply defined mass of mineralized material surrounded by a narrow radiolucent band. 2) Compound odontoma - numerous tiny tooth-like structures within the mass. Odontomas are not neoplasms. Treatment is with marginal excision and/or curettage to remove the harmartoma, which is usually curative.

Tight lip syndrome
Most commonly seen in Shar-pei's where the lip can curl over the rostral mandible and lower

incisors and canines, interfering with occlusion, and inhibiting mandibular growth. The lip becomes traumatized. Teeth of the rostral mandible can become lingoverted. Owners report a reluctance to eat, halitosis, and bleeding. Surgery is required to create a labial vestibule and release the tension on the rostral mandible.

Summary

Early identification of pathology with aggressive and timely action by the general practitioner can help prevent a lifetime of oral pain and discomfort for their patient.

Notes