

## Degenerative valvular disease – What’s New?

Chronic degenerative valvular disease is the most common cardiovascular disease in small animals, and is also known as endocardiosis or myxomatous valve degeneration.

It is seen more commonly in older small breed dogs, and males appear to be predisposed and develop more severe disease.

### **Anatomy:**

Mitral valve (dog) – large anterior leaflet, smaller posterior leaflet and small commissural cusps between the two leaflets. Both leaflets are semicircular and are attached to the mitral valve annulus (fibrous ring) and to the papillary muscles (through chordae tendineae).

Tricuspid valve – two primary leaflets and multiple commissural cusps. The mural leaflet is significantly larger than the septal leaflet.

Aortic valve – three semi-lunar cusps.

### **Pathology:**

- Mitral valve lesions are the most common, but the tricuspid valve and rarely the aortic valve can also be affected.

- Gross inspection reveals thickened and redundant valve leaflets. The free margins of the leaflets are the most commonly affected areas, which have nodules and are opaque.

Mitral valve prolapse – a portion of the body of the leaflet protrudes into the left atrium.

Structures involved: leaflets and chordae tendineae

Lesions:

- Redundancy of the leaflets
- Lengthening of the chordae tendineae
- Thickened and/or fenestrated leaflets

### **Histopathology:**

The atrioventricular valves have four layers:

- Atrialis (endocardial)
- Spongiosa (collagen, fibroblasts, elastic fibers and mucopolysaccharides)
- Fibrosa (collagen bundles)
- Ventricularis (endocardial)

In myxomatous AV degeneration the spongiosa increases in size and the fibrosa degenerates. A significant increase in extracellular matrix is observed and fibroblasts proliferate forming nodules.

### **Etiology:**

- Likely hereditary

- Evidence suggests that degenerative valvular disease is inherited as a polygenic threshold trait in Cavalier King Charles Spaniels.

**Pathophysiology:**

- Valvular regurgitation → increased atrial volume → atrial dilatation
- Eccentric hypertrophy → annular dilation → worsening regurgitation
- Increased atrial pressure → CHF
- Severe regurgitation → decreased forward flow
- Decreased forward flow → Renin-angiotensin-aldosterone system (RAAS) stimulation
  
- Pulmonary hypertension can develop secondary to chronic pulmonary venous hypertension.
  
- Myocardial failure usually occurs in the latter stages of the disease and is more commonly identified in large breed dogs.
  
- Atrial tears/rupture can cause cardiac tamponade secondary to acute hemopericardium.

**History/clinical signs:** coughing, labored breathing, syncope, distended abdomen

**Physical exam:** mucous membrane color and CRT, jugular veins, thoracic auscultation and peripheral pulses

Common PE findings:

- Heart murmur
- Increased intensity of the first heart sound
- Systolic click
- Third heart sound
- Arrhythmias
- Tachypnea/tachycardia
- Crackles and wheezes
- Distended jugular veins
- Brisk femoral pulses

**Diagnostic tests:**

Thoracic radiographs  
Electrocardiogram  
Echocardiogram  
Blood pressure.

**Therapy:**

Asymptomatic disease:

- ACE inhibitors?
- $\beta$ -blockers?

Heart failure:

- Diuretics
- Vasodilators
- Positive inotropes

## What's new?...

### **Etiology:**

Genomic expression patterns and possible genetic biomarkers for canine degenerative mitral valve disease.

### **Diagnosis:**

#### **Laboratory Tests**

- B-type natriuretic factor/peptide (BNP)?
- Atrial natriuretic factor peptide (ANP)?
- Cardiac troponin-I (TnI)?

### **Imaging:**

#### **Real time 3D echocardiography**

Real time three-dimensional echocardiography is a new ultrasound modality that provides comprehensive views of cardiac valves.

This technique potentially provides a more accurate echocardiographic means of evaluating cardiac chamber volumes and a more precise postoperative tool.

Other possible future applications and benefits in veterinary cardiology will also be discussed.

### **Treatment :**

Recent clinical trials concerning the use of ACE inhibitors,  $\beta$ -blockers and pimobendan in asymptomatic disease and heart failure will be reviewed.

QUEST – Pimobendan vs benazepril trial in patients with congestive heart failure secondary to degenerative mitral valve disease.

VETPROOF (follow up) – Enalapril trial in asymptomatic patients with degenerative mitral valve disease.

Benazepril study - University of Alford – Benazepril trial in asymptomatic patients with degenerative mitral valve disease.

### **References:**

- Braunwald E, Zipes D, Libby P. Heart Disease: a textbook of cardiovascular medicine. WB Saunders Co, New York. 2007.
- Kittleson MD, and Kienle, R. Small Animal Cardiovascular Medicine. St. Louis, Mosby. 1998

# Geriatric Dentistry

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## Geriatric Anesthesia:

A discussion of geriatric dentistry must begin with a discussion of anesthesia. All too often we hear “my pet is too old for anesthesia”. Sometimes this is from clients, but often they are hearing it from their veterinarian. Generally, there are no specific issues just that the patient is over 10.

Let me begin by saying, age is not a disease. In fact, when all other factors are equal (meaning the patient has no other medical problems) age was PROVEN to not be a negative indicator in anesthetic complications. While it does increase the odds that the patient does have some systemic (cardiopulmonary or metabolic) derangement, it DOES NOT guarantee it. Therefore, you cannot tell if a patient is (or is not) an anesthetic candidate until a minimum database is performed.

- A. Complete blood panel (renal, hepatic, CBC, T4)
- B. Urinalysis (critical for evaluation of renal values in cats)
- C. Chest radiographs
  - HCM is often not ausculted
  - Over 50% of patients over 6 have significant findings on chest films
- D. Blood pressure in felines (early indicator of disease)
- E. Cardiac ultrasound in cats?

Once the baseline health of the patient is established, you (or we) can determine the appropriate risk: benefit ratio *for each patient*. Even if there is mild to moderate metabolic derangements, the vast majority of patients would benefit from good oral health. Even patients with severe systemic derangements can be treated, especially if their level of disease is significant.

Utilize balanced anesthesia to minimize anesthetic complications. Heavy premedication with opiates (morphine, hydromorphone, or buprenorphine) and diazepam will decrease the amount of induction drugs and inhalational anesthesia needed. Proper pain management including regional blocks and MiLK will further decrease inhalational anesthesia needs. In addition, micodoses of dormitor or ketamine will support blood pressure (but should be avoided in cardiac patients). Finally, have hetastarch and dopamine available for hypotensive situations. A good link for emergency drug dosages is available at .

Patient monitoring should include regular blood pressure evaluation. This can be done manually with a Doppler or automatically with an ossilometric measuring unit. Finally, maintenance of body temperature is critical in older patients, especially small breed dogs and cats. Bair huggers are invaluable during dental surgery.

Finally, avoid lengthy anaesthetics if at all possible. 2 short anesthetic episodes are much safer than 1 long one. Try to avoid any patient being under anesthesia more than 3 hours!

### **Intrinsically stained teeth:**

Intrinsic staining is another clinical sign of tooth death and secondary infection. Affected teeth can appear as pink, purple, yellow, or grey. Additionally, some teeth may just appear more white or opaque. Additional non-vital teeth can be detected by means of transillumination. I have noticed increased intrinsic staining with increased age. This is especially true of the incisor teeth (as well as mandibular third molar teeth) in small breed dogs. In addition, the canine teeth of older large breed dogs are commonly affected. The most common place, however, is in the mandibular incisor teeth of small brachycephalic breeds (Lhasa Apso, Shih Tzu).

A study by Hale showed that while only 40% of intrinsically stained teeth had radiographic signs of endodontic disease, 92.7% of these teeth are actually non-vital. Non-vital teeth lose their natural defence ability and are often subsequently infected via the bloodstream, which is known as *anachorisis*. Therefore, it is important that practitioners do not rely on radiographic appearance to determine vitality. All intrinsically stained teeth should be definitively treated via root canal therapy or extraction.

**Feline Tooth Resorption (TRs) (Previously known as resorptive lesions):** TRs are a very common problem in geriatric patients. Reports vary as to their incidence from 30 to 60% of cats being affected. The main risk factors are increasing age and the presence of other dental disease (including additional TR lesions). TRs are caused by odontoclasts, which are cells that are responsible for the normal remodelling of tooth structure. These cells are activated and then do not down-regulate, resulting in tooth destruction.

There are currently two recognized forms of resorptive lesions, type 1 and type 2. Clinically they appear very similar, as dental defects that are first noted at the gingival margin. However, advanced cases show significant tooth destruction and actually look like a fractured tooth.

Dental radiology is the best diagnostic tool for differentiating the types of TRs. With type 1 lesions, there is no replacement of the lost root structure by bone, whereas with type 2 there is generally marked replacement of the lost tooth structure.

Type 1 TRs are typically associated with inflammation such as L/P stomatitis or periodontal disease. In these cases, it is thought that the soft tissue inflammation may have activated the odontoclasts. The weakened crown will eventually fracture, while the root canal system stays intact resulting in continued pain and infection for the patient.

Type 2 lesions are generally seen in otherwise healthy mouths; however, these lesions will create local gingivitis. The etiology of type 2 TRs remains unproven. The two major current theories include abfraction injuries from eating hard food and excess vitamin D in the diet.

Historically, restoration was a recommended therapy, especially of early lesions. However due to the progressive nature of the disease; this carries a poor long term prognosis and is rarely performed today. Therefore, extraction is now the treatment of choice. Extractions can be very difficult in these cases due to tooth weakening and ankylosis. In cases with significant weakening and or ankylosis, performing the extractions via a surgical approach is recommended to speed the procedure and decrease the incidence of fractured and retained roots (see extraction article).

Recently, crown amputation has been suggested as an acceptable treatment option for advanced type 2 lesions as it results in significantly less trauma and faster healing than complete extraction. This procedure, although widely accepted, is still controversial. Veterinary dentists typically employ this treatment option only when there is significant or complete root replacement by bone. In contrast, the majority of general practitioners use this technique far too often. Crown amputation should only be performed on teeth with radiographically confirmed advanced type 2 TRs which show no peri-apical or periodontal bone loss. Crown amputation should not be performed on teeth with type TRs, radiographic or clinical evidence of endodontic or periodontal pathology, associated inflammation or infection, or in patients with L/P stomatitis. Practitioners without dental radiology capability SHOULD NOT perform crown amputation. In these cases, the teeth should either be fully extracted or the patient referred to a facility with dental radiology.

### **Idiopathic root resorption in dogs:**

This has been described only fairly recently in veterinary literature. The etiology is currently unknown. It is only generally seen in older patients. The most common teeth affected are the mandibular premolars followed by the maxillary premolars. It is occasionally seen in other teeth. Clinical signs are rare and occur late in the course of disease. They consist most commonly of a lesion at the gingival margin, most commonly on the lingual aspect. They may be mistaken for a furcational defect, as they are commonly associated with periodontal disease.

Radiographically they will appear very similar to feline TRs. Generally there is a loss of the periodontal ligament space (dentoalveolar ankylosis). This may become quite severe over time. The major difference is that the root canal system typically is intact. This ankylosis makes extraction exceedingly difficult, however if the root canal system is intact, complete extraction is necessary. In addition, unfortunately, this condition is commonly seen concurrent with periodontal disease. Again, this makes extraction necessary.

Therefore, dental radiographs are invaluable when treating geriatric dogs. The information gained will save valuable time (and frustration) during extraction.

### **Oral neoplasia:**

The oral cavity is the fourth most common place for neoplasia to occur. Obviously, increasing age is the biggest risk factor for oral neoplasia. A newly discovered risk factor for oral neoplasia is periodontal disease and its significant inflammation. This inflammation acts as a promoter allowing the neoplastic cells to grow. Controlling periodontal disease is therefore even more critical in geriatric patients.

Small, apparently innocuous growth should be evaluated more critically in geriatric patients, as the incidence of neoplasia is increased. Biopsy anything abnormal no matter how benign it appears.

### **Extractions:**

Over the life of the patient, extractions will become more difficult. This may be due to ankylosis, which may or may not be radiographically visible. However, it is also true that the alveolar bone hardens over time. This will make extractions more difficult regardless of ankylosis. Therefore, when performing extractions in geriatric patients, allot more time and consider performing surgical extractions initially, rather than waiting for a fractured root. In addition, I would recommend increasing your charges, as it will be more difficult.



# The Incidentally Discovered Adrenal Mass

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## Adrenal “incidentalomas”

- The use of non-invasive imaging techniques has increased
- Adrenal masses are being detected in animals without clinical signs of endocrine disease
- Adrenal masses are discovered in 1-10% of humans being imaged for non-endocrine diseases

## Differential Diagnoses:

- Adrenal Cortex
  - Nodular hyperplasia
  - Adenoma
  - Adenocarcinoma
- Adrenal Medulla
  - Pheochromocytoma
  - Ganglioneuroma
- Other Adrenal masses
  - Myelolipoma
  - Granulomatous disease
  - Teratoma
  - Adrenal cyst
  - Hematoma

## Differential Diagnoses

- Metastasis
  - Lymphoma
  - Mammary gland tumors
  - Leukemia
  - Pulmonary adenocarcinoma
  - Other carcinomas (prostatic, gastric, bladder)
- Pseudoadrenal Masses
  - Kidney
  - Spleen
  - Pancreas
  - Lymph nodes
  - Blood vessels
- Technical artifacts

## Functional Adrenal Tumors

- Hyperadrenocorticism (Cushing's syndrome)
- Pheochromocytoma
- Hyperaldosteronism (Conn's syndrome)
- Hypersecretion of sex hormones

## Realistically...

- Is the tumor secreting glucocorticoids?
- Is the tumor secreting catecholamines?
- Is the tumor causing the clinical signs the pet is presenting with?
- Now what?

## Diagnostic approach

- Review history, PE, clinicopathologic findings (CBC, Chemistry panel, UA)
- Assess contralateral adrenal gland
- Evaluate for hyperadrenocorticism

## Hyperadrenocorticism

- Most common adrenal tumor
- Adenoma or adenocarcinoma
- May have minimal classic clinical signs !
- Contralateral adrenal atrophy
- Adrenal function tests

## Question to be answered

- Is this tumor secreting glucocorticoids?  
This question can be answered with:
  - HDDST
  - endogenous ACTH

## Other tests for Cushing's

- Screening tests for Cushing's may not be helpful
- 40% of dogs with adrenal dependent HAC will have a normal ACTH stimulation test
- Lack of suppression on LDDST does not rule out PDH

## Adrenal-dependent Cushing's

- High dose dexamethasone suppression test
  - Most specific test to r/o adrenal dependent Cushing's

## Adrenal-dependent Cushing's

- HDDST fails to suppress
- Contralateral adrenal atrophy
- Undetectable endogenous ACTH
- Consistent with cortisol secreting tumor

## What if the contralateral gland is large?

- Adrenal hyperplasia due to PDH
  - 25% of dogs with PDH will not suppress on HDDST
  - Endogenous ACTH should differentiate
- Second tumor (pheo, non-functional, other)
- Adrenal function tests can be confusing if both PDH and adrenal dependent Cushing's are present

## Adrenal-dependent Cushing's

- HDDST suppresses adequately:
  - Rules out cortisol secreting tumor
  - PDH with asymmetrical hyperplasia
  - Other types of tumors
- If Cushing's is still suspected clinically, pursue work-up for PDH
  - LDDST, ACTH stim, Uco:cr

## Pheochromocytoma

- Hypersecretion of catecholamines
- Clinical signs due to episodic hypertension
  - panting, weakness, anorexia, weight loss, syncope, "anxiety", tachycardia, skin flushing
- May invade local vasculature

## Pheochromocytoma

- Demonstrate hypertension
- Hypertension present with many adrenal tumors
  - Hyperadrenocorticism
  - Pheochromocytoma
  - Hyperaldosteronism
- Repeat blood pressure several times
  - Normal blood pressure does not r/o a pheo!

## Pheochromocytoma

- Usually incidental finding at necropsy
- Urine and serum catecholamines
  - Abstract at ACVIM in June showed correlation with urine normetanephrine: creatinine ratio
  - Commercial test is not yet validated in veterinary species
- Diagnosis of exclusion
- Often incidental finding at necropsy

## Hypersecretion of sex hormones

- Androgens, estrogens, progesterones, precursors
- Not common in dogs and cats (common in ferrets)
- Clinical signs include endocrine pattern alopecia, PU/PD, or may be non-specific
- Diagnostic tests
  - Adrenal sex hormone levels pre and post ACTH

## Hyperaldosteronism

- Very rare in veterinary patients
- Clinical signs due to hypokalemia and hypertension
  - Weakness, lethargy, ventral neck flexion
- Diagnostic tests
  - Hyponatremia, hypokalemia
  - Aldosterone levels pre and post ACTH

## Surgical treatment

- Treatment of choice if when:
  - Tumors are functional
  - Tumors are large (>3cm)
  - Tumors are invading local blood vessels

## Adjunctive medical therapy

- Adrenergic blockers for suspected pheos
  - Phenoxybenzamine for 2 weeks prior to surgery
- Glucocorticoids for cortisol secreting tumors
- Glucocorticoids and mineralocorticoids for bilateral adrenalectomies

## Medical treatment

- Trilostane or Lysodren (O'PDDD, Mitotane)
  - Occasionally effective
  - May need higher doses
  - Monitor therapy with ACTH stimulation test
- **ONLY TREAT IF THERE ARE CLINICAL SIGNS!**

## Benign neglect

- No clinical signs of adrenal disease
- Owner declines surgery
- Very old or debilitated patients
- Monitor for clinical signs of adrenal disease
- Re-evaluate ultrasonographically every 1-3 months
  - Rapid growth
  - Invasion of vessels

## When an adrenal mass is found:

- Rule out Cushing's syndrome
- Pre-treat with phenoxybenzamine if a pheo is suspected (i.e. Cushing's is ruled out)
- Surgery for functional, large, or invasive tumors
- Wait and watch if surgery is not performed