PATHOLOGY OF THE CARDIOVASCULAR SYSTEM

Cardiovascular pathology is the study of diseases that affect the heart and vascular structures. Diseases of the myocardium, endocardium, pericardium, and vascular structures as well as congenital cardiovascular anomalies and tumors of the cardiovascular system will be discussed.

OBJECTIVES:
1. Review normal myocardial contraction and discuss toxic and infectious disease processes which may result in myocardial disease.
2. Become able to define and discuss the primary cardiomyopathies.
3. Develop an ability to describe common congenital heart anomalies and discuss the pathological disease processes which may be associated with these abnormalities.
4. Be knowledgeable about endocardial diseases, pericardial diseases and vascular diseases.
5. Gain ability to dissect a heart and discuss specific disease processes which may affect each particular area of the heart.

REFERENCES:


The Heart. Chapter 12, by Frederick J. Schoen, pp 555-618, in Robbins and Cotran Pathologic Basis of Disease 7th ed. (2005), Editors: Kumar, Abbas & Fausto. Elsevier and Saunders publishers

HEART - INTRODUCTION

Review - (pp 197-199 Thomson’s Special Vet Path)  
Normal Anatomy  
Normal physiology and normal blood flow (cardiac and general)  
Review changes associated with right and left heart failure  
Myofiber structure and function

Sectioning heart - There are as many ways to section a heart as there are pathologists! Most of the hearts which you will see have been sectioned in the echoplane sectioning – so that one can compare the gross pathological changes to the ultrasound taken in the live animal. In lab, we will review opening the heart in the more traditional manner by following the normal blood flow. Each of you will have the opportunity to open a heart in this manner. The echoplane technique will be demonstrated.
Postmortem Changes – It is important to recognize the normal from abnormal!

- Rigor mortis in heart results in contracted, rigid walls
  - The left ventricle may be empty of blood because of contraction.
  - Post mortem clots (including chicken fat clots) may be present within the atrium and right ventricle.
  - Seen in horses because of the ability of equine erythrocytes to undergo Rouleaux formation.
  - Also can be observed in septic animals
- “Fresh” hearts (before rigor mortis has set in) may appear “flabby and enlarged”.
  - This may resemble but is NOT diagnostic for dilative cardiomyopathy (DCM).
- Hemoglobin imbibition on endocardium and epicardium is seen with erythrocytes lyse after death – post mortem change...
- Young dogs and horses may have diffuse or patchy myocardial pallor grossly but do not have microscopic changes so are considered to be normal. Therefore one must be careful with interpretation of the gross findings and do histopathology.
- Intracardiac injections of euthanasia solution:
  - Can cause acute hemopericardium (so can other injections).
  - Can precipitate and have crystalline deposits on the endocardium or epicardium.
  - Can result in myocardial pallor at the site of injection due to the extreme toxicity of this material when injected into tissue.

DISEASES OF THE MYOCARDIUM

The myocardium is composed of cardiac muscle, (myocytes) similar to skeletal muscle, and therefore responds to injury much like skeletal muscle and vice versa. Cardiac muscle has a greater liability because of its continual activity and dependence on aerobic glycolysis. The papillary muscles of the left ventricle seem to be most sensitive believed to be due to the vascular supply of the heart.

Myocardial cells are considered to be a permanent cell population with extremely limited regenerative potential in the adult animal. Therefore myocardial cell death results in scar formation. The thickness of the left ventricular free wall is approximately 3 times that of the right ventricle when measured in a transverse section across the middle of the ventricles. This results from the greater pressures in systemic circulation than in the pulmonary circuit. In newborn animals the right and left ventricles are approximately equal in thickness.

*Myocardial Reaction to Injury (pp199-202 Thomson’s Special Vet Path)*

The following outline demonstrates the ability of the myocardium to undergo changes that you learned in general pathology. They are simply listed in these notes. If you have forgotten the pathological processes associated with these terms, please review last semester’s lecture notes.
Reversible morphologic alterations of myocardium. These changes are seen as a result of workload requirement.

- Atrophy
- Hypertrophy

Sublethal injuries – myofibres are still viable – but may be injured and not functioning properly.

- Fatty degeneration
- Lipofuscinosis
- Vacuolar degeneration
- Myocytolysis

Lethal cell injury [Myocardial Cell Death]

- Necrosis
  - Often followed by leukocytic invasion + phagocytosis of debris
    - This can look like myocarditis!
  - Macrophage invasion and phagocytosis of necrotic material
  - Fibroblasts proliferation results in fibrosis with collagen deposition
  - End result $\rightarrow$ fibrosis

Reminder: Myocardial regeneration is rare. It occurs in avian hearts and myofibres in the neonatal period. A leading area of research is in progress to develop methods to improve this regenerative capacity.

- Apoptosis
  - Myofibers shrink and form apoptotic bodies
  - No inflammation
  - Minimal fibrosis

NOTE: The Normal myocardium has a 3 - 5 fold functional reserve capacity, which will eventually be lost with cardiac disease and, subsequently, will result in impaired function. However, small lesions can be important if they involve the conduction system.

Compensatory mechanisms occur with an increase demand in workload include:

- Cardiac dilation
- Myocardial hypertrophy
- Peripheral resistance

Cardiac Decompensation (Failure) - (Congestive Heart Failure) (CHF)

**Definition:** The heart is not able to pump blood at a rate which can support the requirements of the metabolizing tissues. --or-- Cardiac output is less than venous return.

**Lesions:** The pathological manifestations of CHF are found in tissues other than the heart. You may want to review general pathology lecture notes if you aren’t feeling comfortable with pulmonary edema/congestion seen with left heart failure (LHF), centrilobular hepatic congestion and generalized edema resulting from increased hydrostatic pressure associated with right heart failure (RHF).
**CARDIAC HYPERTROPHY**

**Definition:** Reversible increase in the mass but **not number** of myocardial cells.

**Pathophysiological Process:**
1. 8 in size of myocardial fibres
2. 8 in # of myofibrils
3. Enlargement of mitochondria, but mitochondria are not 8 in # compared to the size of the myocardial fibre
4. 8 in # of ribosomes
5. Synthesis of abnormal proteins
6. 8 in collagen
7. 8 cell volume compared to vascular supply
8. 8 metabolic requirements and oxygen consumption

* 3 and 7 are two major reasons why a hypertrophic myocardium may fail

**Requirements:**
1. Time
2. Adequate nutrition
3. "Healthy" myocardium
4. If 1-3 are lacking the result is a dilated ventricular chamber and heart failure.

**Types of Hypertrophy**

**Concentric Cardiac Hypertrophy (Pressure Overload Hypertrophy)**

Defined as an increase in the mass of the ventricle without accompanying increase in end diastolic volume.

-Necropsy findings:
- Increased thickness of the ventricular wall and size of papillary muscles of affected chamber.
- Ventricular chamber size may be decreased.
- In general: 8 in the right side of heart (RVH) makes the Æ broader at it base; hypertrophy of the left side (LVH) increases Æ length, bilateral hypertrophy produces a more rounded shape than normal.

-Pathophysiology:
  Sarcomeres increase in number parallel to long axis of cells (wider).

-Causes: Pressure overloads

-Some Examples: Aortic stenosis (LVH), pulmonic stenosis (RVH), pulmonary hypertension (RVH), systemic hypertension (LVH), Hyperthyroidism - enhanced production of myocardial contractile proteins.

**Eccentric Cardiac Hypertrophy (Volume Overload Hypertrophy)**

Defined as an 8 in myocardial mass accompanied by an 8 end diastolic volume - 8 ventricular volume.
Pathophysiology:
Sarcomeres increase in number in length and width.
Necropsy findings: Enlarged cardiac chamber.
- Heart tends to be globose in shape, the ventricular walls are usually thin.
  - Papillary muscles may be attenuated.
  - Endocardium may be white due to fibrosis.

Causes:
Volume overloads
- Local pathological changes
  - Valvular insufficiency (valvular endocardiosis)
  - Congenital defect with shunt (VSD)
- 8 blood volume
- Develop in later stages of diseases that cause concentric hypertrophy.

Detection -
It is often difficult to determine whether a heart is hypertrophied, especially in eccentric hypertrophy where the walls may be thin. One good indicator is the percent heart weight relative to body weight. In a case of hypertrophy the percentage is increased. Normal heart weights vary from species to species and depend upon the amount of physical activity of the individual animal. The following normal percentages are reported: 1.0% dogs, 0.3-0.75% cats, and pigs 0.3%. Remember an increased percent heart weight to body weight may be due to normal physiological hypertrophy.

Three stages of myocardial hypertrophy
1. Initiation
2. Stable hyperfunction
3. Deterioration of function associated with degeneration of hypertrophied myocytes

Cardiac Dilation
1. Causes - same as those of hypertrophy in the presence of a myocardium which cannot undergo hypertrophy for one or more of the following reasons:
   - Insufficient time
   - Inadequate nutrition
   - Diseases myocardium
2. Detection at necropsy – can be difficult.
3. Terminal lesion in many cardiac disease processes.
**Primary Myocardial Disease = PRIMARY CARDIOMYOPATHY**

**Definition:** Acute, subacute, or chronic generalized disorder of heart muscle. An intrinsic disease of the myocardial fiber, the cause is usually not known. This is a primary disease process within the myocardium. In pathological terms, cardiomyopathy is diagnosed as gross or histological changes in the myocardium.

**NOTES:** “Arrhythmogenic cardiomyopathy” (ACM) is a clinical term used to refer to recurrent or persistent arrhythmia in the setting of a normal left ventricular ejection fraction. This clinical disease process often precedes pathological changes.

**Cause:** Usually etiology is unknown or obscure. Idiopathic - speculation of immune or viral etiologies

**Species affected:**
- Dogs - Possible genetic predisposition
  - Great Dane
  - Doberman Pinscher - congestive
  - Irish Wolfhound
  - Saint Bernard
  - English Bull Dogs -ACM
  - Bull Mastiff
  - Boston Terriers - HCOM
  - Springer Spaniels – Atrial Standstill
  - Portuguese Water Dog
  - Golden Retrievers with Muscular Dystrophy
- Incidence: male dogs > female dogs, often middle aged

   - Turkeys "round heart disease"
   - Chickens - ascites Syndrome
   - Cats- A genetic influence is suspected.
   - Cattle – Holstein/Friesian, Simmental/Red Holstein
   - Syrian hamsters
   - Human beings
     - Some forms are Autosomal Dominant with single gene mutation.

**Types**
Classification based on cardiac wall thickness measurements

**Hypertrophic (HCOM)**
- Increased heart weight as a percent body

  **Pathophysiology:**
  - Increased ventricular filling
  - Decreased compliance
  - Diastolic Dysfunction
  - "Concentric hypertrophy gone awry."
Hypertrophic Cardiomyopathy - continued

Primarily a left ventricular problem. But can involve both ventricles and occasionally only septal hypertrophy. This is referred to as eccentric Hypertrophic cardiomyopathy.

Histologically: myocardial fibers are irregular in size. Nuclei are large. Myofibers are disorganized – so called myofibre disarray.

Species: cat, dog, rat, pig, human beings
  Feline: Middle aged males, 10-20% posterior paralysis
  Breeds: Maine Coon, Rag Doll, British and American Shorthair, Rex, Persians
  [Link: http://www.newmanveterinary.com/felhcm.html]

**Congestive (dilated) (DCOM)**
- Progressive cardiac dilation and contractile dysfunction
- Low contractile force 8 end diastolic volume
- Taurine deficiency in cats and foxes
- L-carnitine deficiency may be related to DCOM

Histological: myocardial fibers are thin and wavy. Variable amounts of fibrosis may be present.

Species: cat, dog, hamster, turkey, pig, cow, foxes

Restrictive: least common type seen in domestic animals
- Due to restriction of ventricular filling
- Endocardial fibroelastosis (Will discuss with endocardial disease)
  - Species: cat – Burmese, human beings
- Excessove Moderator Bands – feline
- Endocardial Fibrosis - endomyocarditis

OTHER
- Arrhythmogenic Right Ventricular Fibroadipose Dysplasia - Adipose tissue and fibroblasts infiltrate and replace normal right ventricular myocardial tissue. Seen most often in Boxer dogs.

**Secondary Myocardial Diseases = SECONDARY CARDIOMYOPATHIES**

Definition: Any Disease of the myocardium that is the result of a metabolic, toxic, infectious, or neoplastic disease process. In other words, any myocardial disease that isn’t inflammatory or a primary cardiomyopathy can be referred to as a **secondary cardiomyopathy**. May be peracute, acute, subacute, or chronic. (Many are peracute.)

Note: There are an extensive number of compounds which can result in myocardial degeneration and necrosis. For a comprehensive list – see Thomson’s Special Veterinary Pathology, page 214, Box 4-2.
Secondary Myocardial Diseases (continued)

**Endocrine/Metabolic Diseases**

**Catecholamine Toxicity**
- CNS trauma: “Brain-Heart Syndrome” - myocardial damage secondary to release of endogenous catecholamine dump from trauma to the head and brainstem nuclei.
- Functional pheochromocytoma - tumor of the adrenal medulla
- Exogenous - overzealous administration of epinephrine, etc.
Lesions - Multifocal myocardial necrosis with concentration of damage on the left ventricular subendocardium and papillary muscles. Lesions consist of necrosis with contraction bands with subsequent macrophage invasion.
- Excess intracellular calcium, vasoconstriction and increased heart rate all occurring together may be responsible for these lesions.

**Hyperthyroidism** - Primarily feline
- Cardiac hypertrophy due to increased production of myocardial contractile proteins under the influence of excess concentration of circulating thyroid hormones. Heart rate and cardiac output are also increased.
- Reversible upon return to euthyroidism (?)

**Hypokalemia**
Causes - potassium deficient diets
- hemodialysis
Lesions - rats, pigs, dogs
- Left ventricular free wall and septum
- Multifocal myocytolysis, myodegeneration and necrosis

**Conduction Disorders**
- Unexpected death in Doberman pinscher dogs
  - Focal degeneration of the bundle of His
- Syncope in pup dogs
  - Degeneration of the bundle of His
- Intermittent sinus arrest in deaf dalmation dogs
  - Sinus Node lesions
- Sinoatrial syncope (sick sinus syndrome)
  - Miniature Schnauzers
  - Springer Spaniels
- Arrhythmias – dogs and horses
- Atrial fibrillation
- Heart block
  - associated with myocardial lesions

**Nutritional Deficiencies**

**Vitamin E/Selenium deficiency**
_Vitamin E/Selenium deficiency is an important disease process. The pathogenesis was discussed last semester in General Pathology. Dr. Lopez will discuss again in the Musculoskeletal portion of systemic pathology_
Synonyms for Vitamin E/Selenium deficiency
"White Muscle Disease" lambs and calves
"Mulberry Heart Disease" pigs

Incidence: Occurs in areas with soil deficient in Vitamin E/Selenium. Other tissues besides heart and skeletal muscle can be affected when Vit E and/or Selenium are deficient.

Domestic animals: (myocardial lesions) calves, lambs, pigs, turkey poults and ducklings

Zoo animals: Nyala antelopes, elephants, deer, baboons, camels, exotic birds

Etiology
- Low dietary levels of selenium, Vit E, and sulphur-containing amino acids
- High dietary concentrations of polyunsaturated fats
- Exposure to pro-oxidant compounds
  ozone, oxygen, iron, radiation injury, doxorubicin
- Intake of selenium antagonists such as silver salts and various other metals
  mercury, copper, cobalt, cadmium, tin, zinc

Pathogenesis: Vitamin E is an antioxidant that works synergistically with glutathione peroxidase to catalyze the conversion of $\ce{H2O2}$ to $\ce{H2O}$. Selenium is an integral structure of glutathione peroxidase (metalloenzyme).

Gross Lesions
Calves: extensive pale areas of necrosis and mineralization in the left ventricular free wall and ventricular septum.
Lambs: similar areas of necrosis and mineralization but lesions are present in the subendocardial myocardium of the right ventricle.
Pigs: widespread epicardial and myocardial hemorrhages, with pale streaks scattered throughout the heart. The myocardial lesions are present in the walls of all chambers, but tend to be most severe within the atria than the ventricular myocardium.

Histo Lesions
Calves and lambs: Areas of myocardial damage have hyaline necrosis with or without accompanying mineralization, macrophage invasion, with eventual stromal collapse and fibrosis.
Pigs: Vascular lesions consist of fibrinoid necrosis in intramyocardial arteries and arterioles. Numerous fibrin microthrombi in myocardial capillaries. Myocardial hemorrhage and edema. Muscular lesions include hyaline necrosis and mineralization with macrophage invasion and later fibrosis.

Copper and/or Iron Deficiency

Synonyms - “falling disease” cattle
Lesions:
Gross: pale, atrophic and flabby hearts
Micro: myocardial fibrosis

Experimental disease in pigs
Lesions: myocardial rupture = hemopericardium
Rupture of pulmonary or coronary arteries
Toxic Agents Producing Myocardial Disease

Gossypol Toxicity
Cause: Gossypol is found in cottonseed
Requires 2-3 months of continuous feeding
Species affected:
- Pigs are more susceptible than cattle
- Reported in dog
Lesions: Dilated heart with multiple areas of degeneration and necrosis.
Hepatotoxic - produces necrosis

Poisonous Plants http://library.thinkquest.org/C007974/intro.htm
Cardiac Glycosides
Oleander - sudden death no lesions
Foxglove
Lily of the Valley

Cardiotoxins
Myocardial depressants
Direct injury to myofibre
Hypersensitivity
Toad Poisoning – Bufo toxicosis (cardiac glycoside)

Chemotherapeutic agents

** Doxorubicin (Adriamycin)
- Antineoplastic compound prevents synthesis of DNA, RNA and proteins
  Gross: Dose-related chronic cardiotoxicity characterized by congestive heart failure - see lesions of congestive heart failure. Pale myocardium
  Micro: Sarcoplasmic vacuolization “Adria Cells”
  Myocytolysis, hyaline necrosis
  Hydropic degeneration

** Monensin
- Coccidiostat for poultry, and growth-promoting agent for cattle
- Toxicosis reported in horses, cattle, sheep, pigs, dogs, and poultry
  Lesions: necrosis of cardiac and skeletal muscle

Furazolidone - toxicoses in poultry
- Antibacterial drug
- Congestive heart failure myocytolysis - sublethal injury, pale eosinophilic
- Turkeys slightly more sensitive than other poultry

Other agents
Fluroacetate (1080) commercial rodenticide
Cantharidin intoxication from Blister Beetles
INFLAMMATORY DISEASES OF THE MYOCARDIUM – MYOCARDITIS

Infectious Agents

**Viral**
- Encephalomyocarditis virus of swine picornavirus
- Parvovirus in the dog
  - Usually affects pups <10 weeks of age
- West Nile Virus
- Foot and Mouth Disease - cattle
- Pseudorabies
- Canine distemper in very young puppies
- Bluetongue in sheep
- Coxsackie virus in man and mouse
- Avian encephalomyelitis, Newcastle Disease, Eastern and Western encephalitis
- Human causes – Coxsackievirus, influenza, HIV, cytomegalovirus

*Note*: Viral agents may produce acute necrosis with little inflammatory response, or if the animal survives, may be infiltrated by inflammatory cells.

**Bacterial**
- Direct extension of pericarditis (eg. "hardware disease")
- Direct extension of endocarditis (eg. erysipelas in pigs)
- Any septicemia
  - Breleria burgdorferi – Lyme disease
  - Histophilus somni - cattle
  - Blackleg - Clostridium chauvei in cattle
- Listeria monocytogenes especially in sheep
- Actinobacillus equuli and Strep equi in horses
- Arcanobacterium pyogenes sheep, cattle
- Staph aureus in sheep and goats
- Corynebacterium kutscheri - rodents
- Pseudomonas aeruginosa
- Tyzzer’s Disease Bacillus piliformis in horses, rabbits, puppies and birds.
- Necrobacillosis - Fusobacterium necrophorum

**Parasitic**
- Neospora caninum
  - Fatal fetal myocarditis and encephalitis
  - Fetal infections may result in abortion or weak calves
- Toxoplasma gondii
  - Occurs in any species in contact with cat feces
    - Public health hazard
    - Fatal fetal myocarditis can occur if infected during pregnancy
**Trypanosoma cruzi** (Chagas’s disease) Trypanosomiasis
- reported in dogs and people in Texas, Louisiana and Mexico
- fatal infection in young dogs (6-8 months)
  - "Assassin" bug inoculates organism while feeding
- *Sarcocystis cruzi, S. bovicanis* of little significance if present within the myocardial fibers of an adult animal.
- Trichinella (usually skeletal muscle but occasionally heart)
- Cysticercus ovis (Taenia ovis)

**Fungi**
- Does not commonly affect the myocardium – in fact relatively rare!
  
  *Blastomyces dermatitidis*
  *Coccidioides immitis*

**Miscellaneous and Interesting Myocardial Pathology**

**Porcine stress syndrome**
- associated with stress of shipment
  - Genetic defect in the ryanodine receptor gene which results in an increase in the “open” state of this Ca^{2+} channel
Sudden death in cattle - sometimes due to myocardial necrosis
  - etiology not known - associated with calcium containing inclusions in cardiac mitochondria - may be related to Vitamin E/Se deficiency
Canine gastric/dilatation or volvulus Y myocardial ischemia and necrosis
Senile Lipofuscinosis - aged animals and animals with severe cachexia
  - Hereditary disease in Ayrshire cattle - no clinical signs
Glycogen storage disease
Senile changes
  - Fibrosis - (horses)
  - Fatty infiltration (common in old sheep) or associated with obesity
  - Anichkov Cells – "caterpillar cells"
    - Origin not determined – may be macrophage or abortive attempt of myocyte regeneration.
  - Epithelial Inclusion Cysts - incidental finding seen in cows embryonic rests.
  - Primates - have bizarre myocardial cells.
CONGENITAL ABNORMALITIES OF THE HEART AND LARGE VESSELS

Causes of Abnormal Cardiovascular Development

1. Genetic
   - Single or multiple gene effects
   - Most species have a low background frequency of spontaneous cardiac malformations

2. Infectious agents:
   - Parvovirus infections in cats and dogs
   - Bluetongue virus in sheep
   - BVD in cattle

3. Teratogens:
   - Deficiencies: Vitamin A, pantothenic acid, riboflavin, or zinc
   - Excesses: Vitamin A, retinoic acid, or copper
   - In utero exposure to x-irradiation or fetal hypoxia
   - Teratogenic compounds: thalidomide, ethanol, salicylates, griseofulvin, cortisone

Cardiovascular Malformations by Species and Breed

NOTE: Most Common Defects are identified with an asterisk *

**Canine -**

<table>
<thead>
<tr>
<th>Malformation</th>
<th>BREEDS</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patent Ductus Arteriosus (PDA)</td>
<td>(Poodle, Collie, Pomeranian, Chihuahua, Maltese)</td>
<td>25.0%</td>
</tr>
<tr>
<td>Pulmonic Stenosis</td>
<td>(Bulldog, Fox terrier, Beagle, Samoyed, Chihuahua, Mastiff, Chow Chow, Cocker Spaniel, Labrador retriever, Terriers, Boxer, Schnauzer, Newfoundland)</td>
<td>17.5%</td>
</tr>
<tr>
<td>Subaortic stenosis</td>
<td>(German Shepherd, German Shorthair pointer, Bull terrier)</td>
<td>12.0%</td>
</tr>
<tr>
<td>Persistent Right Aortic Arch (PRAA)</td>
<td>(German Shepherd, Irish Setter, Great Dane)</td>
<td>7.0%</td>
</tr>
<tr>
<td>Ventricular Septal Defect (VSD)</td>
<td>(Bull dog)</td>
<td>6.0%</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>(Keeshound, English Bull Dog)</td>
<td>3.4%</td>
</tr>
<tr>
<td>Atrial Septal Defect (ASD)</td>
<td>(Samoyed, Boxer, Doberman, Samoyed)</td>
<td>3.7%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td>74.6%</td>
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</tbody>
</table>

**Feline**

Siamese cats have a higher incidence of congenital heart disease.

<table>
<thead>
<tr>
<th>Malformation</th>
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<tbody>
<tr>
<td>Endocardial cushion defects</td>
<td></td>
</tr>
<tr>
<td>Mitral Valve dysplasia</td>
<td>19/96</td>
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<tr>
<td>Tricuspid valve dysplasia</td>
<td>18/96</td>
</tr>
<tr>
<td>VSD</td>
<td>13/96</td>
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<tr>
<td>Aortic Stenosis</td>
<td>12/96</td>
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<tr>
<td>Persistent Common Atrioventricular canal</td>
<td>11/96</td>
</tr>
<tr>
<td>PDA</td>
<td>7/96</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>6/96</td>
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</tbody>
</table>

**Bovine**

<table>
<thead>
<tr>
<th>Malformation</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>Valvular hematocysts (see endocardial disease)</td>
<td></td>
</tr>
<tr>
<td>VSD - may be associated with microphthalmia</td>
<td>ASD</td>
</tr>
<tr>
<td>Transposition of great vessels</td>
<td>Hypoplasia of the left ventricle</td>
</tr>
<tr>
<td>Anomalous coronary artery</td>
<td>Persistent common truncus arteriosus</td>
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**Equine**

<table>
<thead>
<tr>
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<th>Percentage</th>
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<tbody>
<tr>
<td>VSD</td>
<td>Aortic Stenosis</td>
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<tr>
<td>ASD</td>
<td>Persistent Common Truncus Arteriosus</td>
</tr>
<tr>
<td>PDA</td>
<td>Tetralogy of Fallot</td>
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**Porcine**

<table>
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<th>Malformation</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>Aortic subaortic stenosis</td>
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<tr>
<td>Endocardial cushion defects</td>
<td></td>
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**Ovine**

<table>
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<th>Malformation</th>
<th>Percentage</th>
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<tr>
<td>VSD</td>
<td>Endocardial cushion defects</td>
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Abnormalities of the atrial septum

Foramen Ovale (normal)
- Functionally closes at birth
- Anatomically closes later

**Atrial Septal Defect (ASD)**
Common Atrium
Haemodynamics: 8 flow from L to R atrium 6 8
Vol Rt Â 6 8 CVP (may reverse flow with pulmonary hypertension)
Dogs Breeds
Boxer
Doberman Pinscher
Samoyed

Abnormalities of the atrioventricular canal

Normal Development:
The endocardial cushions come together and form mitral valve, tricuspid valve, annulus pulposis and separation of four chambers.

** Persistent common atrioventricular canal - Endocardial cushion defect 
- Failure of endocardial cushions to form.
- Often associated with concomitant ASD and VSD.
- May not be fatal but there will be mixing of systemic and pulmonary blood.
- Few cases have been seen in lambs at AVC.

** Tricuspid dysplasia 
- Seen most commonly in Labrador retrievers and cats.
- Septal valve leaflets are attached to the septum.
- Chordae tendinae are short and thick.
- Downward displacement of base of the valves.

Mitral Valvular Insufficiency
Malformations of mitral valve complex
Relatively common in cats

Tricuspid atresia
a. Obliteration of the right atrioventricular orifice
b. If present, often other defects may be present and essential for in utero survival.
These may include patent foramen ovale, ventricular septal defect, right ventricular hypoplasia and left ventricular hypertrophy.

1 Please note: All Figures have been adapted from: Jubb, Kennedy, Palmer, Volume 3, 4th ed, Pathology of Domestic Animals
Abnormalities of the Interventricular Septum

Normal Development
1. Primary R and L ventricles of the embryo communicate via the primary interventricular foramen;
   - bordered above by bulboventricular flange and below by muscular interventricular septum.
2. Primitive ventricles dilate and myocardium grows continually.
   - the medial walls of the dilating and growing ventricular myocardium fuse.
   - muscular interventricular septum.
3. Membranous portion of the interventricular septum is formed by the R and L truncoconal ridges and superior atrioventricular cushion.
4. Muscular portion grows up - membranous portion grows down.

** Interventricular Septal Defect - VSD
- Defect of membranous septum (most common).
- Occasionally the muscular part of the septum is also affected (less common).
- Sometimes associated with other anomalies of the truncoconal region (overriding aorta).
- Haemodynamics: overload RV 6 8 RV psi 6 RVH 6 RVF [blood shunts L 6 R but could reverse later]

Abnormalities of the truncus arteriosus and conus cordia

Normal Development
- Spiral ridges fuse to divide truncus arteriosus and conus cordis into aorta and pulmonary artery.

** Tetralogy of Fallot
Caused by unequal division of conus cordis due to anterior displacement of the truncoconal septum.
Consists of the following (4):
1. Pulmonic stenosis
2. Ventricular septal defect - membranous
3. Overriding aorta (rightward displacement)
4. RVH – Results from 1-3 anomalies
Haemodynamics of Tetralogy of Fallot
Pulmonic Stenosis 6 psi overload RV 6 RVH 6 RHF + 8 RV psi 6 blood shunts RV to LV 6 venous blood into systemic circulation 6 cyanosis.
Keeshonds – inherited
English bull dogs

** Eisenmenger’s Complex/Syndrome
Tetralogy of Fallot without pulmonic Stenosis.

** Eisenmenger’s Physiology is the result of a right to left shunt.
Persistent truncus arteriosus
- Failure of the spiral ridge to divide the truncus arteriosus and conus cordis into the pulmonary artery and aorta.
- Seen most frequently in the horse.
- Animal may survive up to a year.

Transposition of the great vessels
- Truncoconal septum fails to spiral therefore it descends straight downward.
- Aorta exits from right ventricle and pulmonary artery exits from left ventricle.
- Sometimes associated with VSD.

Abnormalities of the Semilunar Valves

**Pulmonic Stenosis** (> dogs)
Inherited: Beagles, English bulldogs, and Chihuahua.
Seen in many dog breeds.
Partial fusion of pulmonic valve.
- Circumference of pulmonic valve < aorta.
- RVH occurs 2E to 8 resistance.

Supravalvular Pulmonic Stenosis
6 Fibrous narrowing above valve.
Results in Right ventricular hypertrophy (RVH).

Subvalvular Pulmonic Stenosis
6 Hypertrophy of crista supraventricular muscle ridge.
6 Fibrous connective tissue below valve.

Haemodynamics
Psi overload RV 6 concentric cardiac hypertrophy 6 RHF.

Post stenotic Dilation of Pulmonary Artery
Dilation of pulmonary artery near stenotic valve due to turbulence.
Secondary problem associated with the stenotic valve.

complete fusion = pulmonary valvular atresia
- RV fails to develop normally due to nonfunctional pulmonic valve.
- If nonfatal in utero must be an associated with ASD or patent foramen ovale
  (only outlet from right heart) and PDA (only access to pulmonary circulation).
- Usually fatal at birth or shortly thereafter.

** Aortic Stenosis
- Partial fusion of aortic valve.
- Circumference of the aortic valve < pulmonary valve.
- LVH occurs secondarily to increased pressure.
  Result is concentric hypertrophy.
- Most common defect in swine.
- Inherited: Newfoundland, boxer, German shepherd.
  Seen in many other dog breeds.
**Subvalvular Aortic Stenosis**
- Is much more common than a valvular aortic stenosis.
- May occur secondary to severe LVH.
- May occur with hypertrophy of the interventricular septum.
- Could result from inflammatory response due to irritation of mitral valve.
- Pigs most often have this type of aortic stenosis.

Haemodynamics: 8 psi of LV 6 concentric LVH 6 LVF.
Post stenotic dilation of aorta
- Occurs due to turbulence of blood flow through stenotic valve.

Total fusion = aortic atresia
- Left ventricle fails to develop normally due to lack of aortic valve.
- If not fatal in utero there must be associated large PDA and usually patent foramen ovale.
- Usually fatal at or shortly after birth.

**Abnormalities of the Position of the Heart**

**Ectopia cordis**
1. Most frequently seen in cattle - (subcutis, neck).
2. Heart is outside of the thoracic cavity.
3. Usually secondary to sternal cleft or failure of thoracic cavity to close normally.
4. Some animals can survive for several days to months.

**Dextrocardia**
1. Heart is on the right side rather than the left.
2. Often associated with total or partial situs inversus (mirror image of normal left to right symmetry) - Ciliary Dyskinesia, Kartagener’s Syndrome.
   a. Dextrocardia and situs inversus.
   b. Total lack of ciliary movement.
   c. Usually fatal.

**Abnormalities of Great Vessels**
- Anomalies from failure of closure of fetal cardiovascular shunts.

Normal Development:
Fetus has two right to left shunts to bypass the lung:
1. Foramen ovale
2. Ductus arteriosus
   - In the dog there is functional closure by 9 hours and anatomic closure with formation of the ligamentum arteriosum by 15-18 days.
   - Closure in the pig by 12-14 days.
   - Closure in the guinea pig by 24 hours.
   - Ruminants - may be probe patent in "young".

Aorta develops from the left aortic arches.
Right subclavian artery arises from the brachiocephalic trunk.
Abnormal Development or Great Vessels

** Patent Ductus Arteriosus (PDA)**
1. Failure of the ductus arteriosus to close by 3 weeks - normally forms ligamentum arteriosum.
2. Inverse relationship between lung expansion at birth and closure of the ductus.
3. Some cardiac malformations (see above) require a PDA for survival (eg. pulmonary atresia and aortic atresia).
4. Haemodynamics
   Blood shunts L → R (from aorta to pulmonary artery, circulation through lungs excess work load on left heart (LVH, LVF) may reverse in severe, chronic cases (volume).
5. Breeds affected:
   - Poodles, Collies, Pomeranians, Chihuahua, Maltese
   - Other breeds

** Persistent Right Aortic Arch (PRAA)**
A. Right 4 th aortic arch remain, left arch disappears.
B. Aorta comes down the dorsum of the esophagus.
C. Ductus arteriosus attaches to the aorta behind the esophagus.
D. "Vascular ring anomaly" results due to entrapment of esophagus and trachea by (MARKED ON PHOTO BELOW):
   1. Descending aorta
   2. Ligamentum arteriosum (closed or open)
   3. Main pulmonary artery (not visible on photograph)
   4. Heart base (not visible is beneath the esophagus (E)
E. Esophageal dilatation proximal to the constriction can result in regurgitation.
F. Death most often due to aspiration pneumonia.
G. German shepherd, Great Dane, Irish setter dogs are predisposed.
DISEASES OF THE PERICARDIUM

Comments: Primary pericardial disease is rare. However, the pericardium is frequently involved secondarily by direct extension from diseases of the myocardium, pleura, lungs, or systemic disease processes. The good news is you heard of many of the disease processes in general pathology!

The pericardial sac is basically a fibrous sac surrounding the heart, and therefore reacts to injury in a limited manner. The contents within the pericardium may provide clues relating to the pathogenesis of the disease process. The entire surface of the pericardial cavity is covered by mesothelium. Visceral pericardium is another name for the epicardium. The pericardial sac can be expanded over time.

Non-Inflammatory Disease Processes

**Hydropericardium**
Definition: excessive accumulation of serous fluid in the pericardial space.
Causes:
- Hypoproteinemia (generalized edema)
- Congestive heart failure,(usually right heart failure)
  "Ascites syndrome" - poultry
  Dilated cardiomyopathy - dogs and cats
  Pulmonary Hypertension - "Brisket Disease" "high altitude disease."
- Tumors - both primary and secondary.
- Systemic Diseases (may have some protein and may clot)
  Good site to swab for bacterial isolation.
  Mulberry heart disease (swine)  Viral Diseases
  Bacterial Septicemias in swine
  Heartwater (rickettsial disease
  in small ruminants)
Significance of hydropericardium:
  Acute - cardiac tamponade.
  Chronic - pericardium can distend/stretch to accommodate.
  Reversible - if 1 cause can be removed.

**Hemopericardium**
Definition: accumulation of blood in pericardial space.
Causes:
- Aortic rupture within pericardial sac (horse, turkey).
- Atrial rupture (dog).
- Rupture of the pulmonary artery.
- Iatrogenic - intracardiac injections.
- Bleeding from a tumor within pericardial sac.
Sequela:
  Acute:
  Produces 9 cardiac filling and 9 cardiac output
  (cardiac shock).
  "Cardiac tamponade" terminology used for acute filling of pericardial sac.
  Right heart more sensitive than left heart.
  Atria collapse and are unable to fill with blood.
  Chronic – pericardial sac can expand and accommodate blood.
**Hemorrhagic pericardial effusion** of dogs
- etio: unknown cause can occur with bleeding tumor within heart/epicardium.
- Seen most often in large breed dogs, especially Golden retriever.

### Serous atrophy of fat

**Definition:** degenerative of adipose tissue with replacement by loose connective tissue.

**Causes:**
- Inadequate nutritional supply.
- Secondary to starvation or inanition.
- Occurs relatively rapidly in a sick cow.

**Significance:**
- Indicates the condition of health of the animal.
- Little effect on local function and is reversible.
- Usually seen on epicardial surface of heart near or in coronary grooves.

### Congenital and Miscellaneous Disorders

**Absence**
- Diaphragmatic-Pericardial Hernia (> cats)
- Visceral Gout (avian and reptile) - urate deposits
- Epicardial mineralization - (cardiac calcinosis)
- Inbred strains of mice
  (photo in Thomson's Special Path).

### **Inflammatory Diseases of the Pericardium**

**Infectious Process**
- Extending **outward** from the myocardium.
- Extending **inward** from the pleural space or mediastinum.

**Septicemias** – most common.

**Fibrinous Pericarditis** – usually hematogenous.

**Lesions:**
- **Gross:** Accumulation of fluid and fibrin within pericardial space. Surfaces of epicardium and pericardial sac may be slightly opaque. Fibrinous adhesions which can be broken down (torn apart) easily are occasionally referred to as "Bread and Butter."
- **Micro:** Usually only mild inflammation with fibrin on the surface involving the pericardial sac and epicardium in acute disease processes. Variable numbers of neutrophils and macrophages are seen.

**Cause:** (usually hematogenous infections)
- **Cow:** Mannheimiosis, blackleg, coliform septicemias
- **Fetus:** *Brucella sp*, *Arcanobacter pyogenes*
- **Pig:** Glasser's disease (*Haemophilus parasuis*), Streptococcus, Mannheimiosis, Mycoplasma pneumonia, Salmonellosis
Horses: Streptococcal infections
Birds: Psittacosis
Cats: Feline infectious peritonitis (FIP)
Sheep: Pasteurella and Streptococcal infections

Outcome:
- Early death – often not directly related to the pericarditis, but rather due to the pathogenicity of the organism and resultant septicemia.
- Fibrous adhesions may occur.

**Purulent or Suppurative Pericarditis**

Lesions:
- **Gross:** Fluid and liquefied inflammatory debris accumulated within the pericardial sac, usually very malodorous.
- **Micro:** Moderate accumulations of neutrophils and other inflammatory cells on the surface of the pericardial sac and epicardium. Fibrous connective tissue present beneath the layer of inflammatory cells, but dependent upon the time frame of the disease process.

Cause: Associated with various pyogenic bacteria. Often complication of traumatic reticulopericarditis.

Note: Either fibrinous or suppurative pericarditis may undergo organization which produces fibrous adhesions of the pericardium to the epicardium.

**"Bovine Traumatic Reticulopericarditis"

("Hardware Disease")
- Due to extension of a sharp object through the wall of the reticulum, diaphragm and pericardium.
- Mixture, or "garbage can" of bacteria isolated.

*/Sequela of Pericarditis*

- Effective resolution depends on the extent of the disease and severity.
  - **Mild fibrinous pericarditis** - May resolve completely.
  - **Severe fibrinous pericarditis** - May resolve with only focal or diffuse thickenings of the pericardium or epicardium or focal adhesions may occur.
  - **Suppurative pericarditis** - Seldom resolves completely. The resolution that occurs often results in massive adhesions of pericardium to heart (restrictive or constrictive pericarditis) producing congestive heart failure.

*/Constrictive Pericarditis*

**Definition:** Chronic inflammatory lesion of pericardium accompanied by extensive fibrous proliferation and eventual formation of fibrous adhesions across the pericardial space.

**Results:** Compensatory myocardial hypertrophy due to interference of cardiac filling results in eventual congestive heart failure (usually right heart).
DISEASES OF THE ENDOCARDIUM

The endocardium is the innermost layer of the heart. It lines the chambers and extends over projecting structures such as the valves, chordae tendineae, and papillary muscles. The atrial endocardium is thicker than the ventricular endocardium. Purkinje fibers are distributed throughout the ventricles in the subendocardium. **Primary Endocardial Disease** is not common and is defined as a non-inflammatory disease in which the exact cause is not known, eg: Endocardial fibroelastosis and endocardiosis.

*Endocardial fibroelastosis*

**Cause:**
- Familial disease in the Burmese cat.
- Viral infections:
  - Parvovirus (dog)
  - Encephalomyocarditis virus (man, mouse, pig)
- Hypoxemia
- Trauma
- Extreme dilatation of ventricular chamber.

**Pathogenesis:** Progressive edema of endocardium results in fibroblast proliferation and an 8 amount of collagen and elastin within and/or immediately beneath endocardium.

**Effects:**
- Restricted myocardial motion produces a i in output and may lead to CHF
- May incarcerate subendocardial Purkinje fibers which could result in a left bundle branch block.

Form of restrictive cardiomyopathy.

**NOTE:** Focal subendocardial fibrosis is occasionally seen in the atria and intima of large vessels. These changes are a reaction of the endocardium/ endothelium to abnormal jets of blood or to turbulence following congenital or acquired valvular disorders. These structures are frequently termed, "jet lesions."

**Valvular Endocardiosis**

**Synonyms:**
- Chronic valvular fibrosis.
- Valvular mucoid degeneration.
- Myxomatous degeneration of the valves.
- Myxomatous transformation of atrioventricular valves.

**Pathogenesis:** Degenerative lesion characterized by proliferations of loose, fibroblastic tissue in the spongiosa with deposition of acid mucopolysaccharides. Collagen within the fibrosa region of valve becomes degenerative. A genetic predisposition is recognized, but the mode of inheritance is unknown and complex.
Lesions

**Gross** - glistening smooth nodular thickenings on the valve leaflets margins and chordae tendineae.

**Micro** - spongiosa is greatly thickened by proliferation of loose fibroblasts with an accumulation of poorly staining mucopolysaccharide material.

Incidence:
- Occurs most frequently in dogs > 5 years of age, males > females.
- In one study of 4831 dogs, 11.3% had cardiac disease.
  - 72% had endocardiosis
- Cavalier King Charles >50% affected at 4 years of age 100% >10 years.
- Cocker spaniels more commonly than expected.
- German Shepherds less commonly than expected.
- Other breeds: beagle, dachshund, poodle, schnauzer, Chihuahua, fox terrier, Boston terrier, Pekinese, Wolfhound, deerhound, Doberman pinscher.

Valves affected

<table>
<thead>
<tr>
<th>Valve Type</th>
<th>Percentage</th>
</tr>
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<tbody>
<tr>
<td>Left atrioventricular alone (mitral)</td>
<td>60%</td>
</tr>
<tr>
<td>LAV and RAV</td>
<td>30%</td>
</tr>
</tbody>
</table>

Aortic valve and pulmonic valves may be affected but not as frequent.

Sequela
- Valvular insufficiency:
  - Due to contracture of chordae tendineae resulting in volume overload:
    - LVH
    - LVF
    - Congestive failure.
- Rupture of chordae tendineae:
  - Acute left heart failure
  - Pulmonary edema
  - Death.
- Chronic left heart failure
  - Pulmonary fibrosis.
- Rupture of left atrium
  - Cardiac tamponade.

Blood Cysts

**Synonyms** = Hematocyst, Valvular hematoma.

**Incidence:** Occurs most commonly on valves of calves and cattle, most frequently affect the AV valves. Incidental finding. My also be seen in foals, puppies, and dogs.

Lymphocyst - Valvular cystic structures filled with clear fluid.

Secondary Endocardial Disease

**Definition** = Disease of the endocardium resulting from metabolic, toxic, infectious, or neoplastic disease.

**Mineralization**

Pathogenesis: The cardiovascular system is unusually susceptible to mineralization because of its high elastin content. This is particularly true in the endocardium and tunica intima of vessels, (internal elastic lamina). Mineralization is the most common degenerative change seen in the endocardium.
Lesions: gritty and granular plaques generally subendocardial in location.

Gross: White, elevated, firm.

Micro: Accumulations of basophilic, acellular material usually with little or no inflammation (stains positive with Von Kossa stain for calcium).

Causes: Any disease which will lead to an imbalance of Ca:P ratio.

- **Endocrine/Metabolic Diseases**
  - Pseudohyperparathyroidism
  - Hyperphosphatemia
  - Nutritional (excess phosphate diet)
  - Renal failure

- **Toxic Substances**
  - Vitamin D poisoning
  - Plants containing Vitamin D analogs
    - "Manchester wasting disease" Jamaica
    - "Naalehu disease" Hawaii
    - "Entique Seco" Argentina
  - Plants in the potato family
    - Solanum malacoxylon
    - Cestrum Diurnum
  - Miscellaneous Causes of endocardial mineralization.
    - May accompany endocardial fibrosis when chambers are acutely dilated.
    - Chronic debilitating disease, ruminants.
    - Jet lesions may become mineralized.

**Endocarditis**

Definition: Inflammation of the endocardium. Usually bacterial in origin.

Pathogenesis: Usually valves are affected, (valvular endocarditis). However, occasionally valves are spared and there is involvement of the non-valvular endocardium, (mural endocarditis). The manner in which the bacteria lodge on the valves is not clear. Valves are thought to be predisposed because of their lack of blood supply. Nutrition and immune effector cells come primarily from passing blood. These areas may be predisposed because of continual trauma and/or lack of blood vessels within the valve. Bacteria tend to localize along appositional surfaces. Recurrent bacteremia is frequent. The following valves are affected most commonly:

  Mitral (LAV) > Aortic > Tricuspid (RAV) > Pulmonary

**Organisms**

**PIG**

*Erysipelothrix rhusiopathiae*, chronic usually LAV

Strep suis usually LAV +/- aortic valve

*Staph aureus*

**COW & SHEEP**

*Arcanobacterium pyogenes* - (can originate from mastitis, metritis, or hepatic abscesses)

*Strep sp* - lambs with polyarthritis
HORSE - uncommon
   *Streptococcus equi*
   *Actinobacillus equuli*
   *E. coli*
   *Pseudomonas aeruginosa*

CAT & DOG
   beta hemolytic *Streptococcus sp*
   *Erysipelothrix rhusiopathiae*
   *Bartonella sp* - canine

Lesions
   **Gross**: Proliferative lesions (vegetations), which are yellow-red or yellow-gray and usually covered by a thin clot of blood, which can be easily peeled off at necropsy. The surface is friable, small lesions can be broken off leaving a granular, eroded surface on the valve. Extension into the adjacent mural endocardium is frequent.

   **Micro**: Bacterial colonies are numerous. Accumulations of fibrin, neutrophils and variable amounts of granulation material dependent upon the length of time the lesion has been present.

   *Sequela:* - Chronic lesions may organize by granulation from the base of the valve.
   - May undergo mineralization. Complete resolution is uncommon.

   **Right Heart**
   - Valvular distortion = right heart failure
   - Pulmonary thrombosis and abscessation (embolic pneumonia)

   **Left Heart**
   - Valvular distortion = left heart failure
   - Thromboemboli (kidney, spleen, myocardium, brain, joints)
   - Inflammatory induced septal defects or pericarditis can occur

**Miscellaneous Endocardial/Valvular Diseases**

- *Atrial thrombosis* may be associated with failing ventricle.
  Dogs & cats with cardiomyopathies.
  Syrian Hamsters and certain strains of mice

- *Uremia* (usually acute): **Uremic Endocarditis**
  - ulcerative endocarditis of left atrium causes endocardial mineralization, inflammation and thrombosis. Mechanism unknown.

- *Subendocardial hemorrhage*
  - Bacterial septicemias
  - Bluetongue (sheep) - hemorrhage at the base of the pulmonary artery
  - Infectious canine hepatitis
  - Toxemias - ruminants
  - Agonal findings - adult bovines

- **Strongylus vulgaris** larvae occasionally migrate aberrantly through the endocardium, eliciting an inflammatory response.
- Mycotic infections are rare
Degenerative Diseases

NOTE: Degenerative changes are relatively uncommon in animals compared to human beings. The following is a brief summary of disease processes seen most often in humans, but occasionally in “non-human mammals”.

Arteriosclerosis

**Synonym:** arteriolarsclerosis, arteriolosclerosis.

**Definition:** “hardening of the arteries”. Includes all chronic arterial metamorphoses which consist of induration, loss of elasticity and narrowing which are the result of proliferative and degenerative (NOT inflammatory) changes of the media and intima.

Atherosclerosis

A subtype of arteriosclerosis.

Greek derivation “gruel or mush.”

**Definition:** refers to degeneration in the wall of an artery in which lipids, (cholesterol, triglycerides, etc), are the primary components of the degenerative response.

Occurs chiefly in large elastic arteries in man:
- aorta and its branches
- coronary arteries
- cerebral arteries
- Atheromatous plaques may occlude the vessel producing ischemic necrosis of the tissue supplied by the involved artery.

<table>
<thead>
<tr>
<th>Medial Necrosis (of Monckeberg)</th>
<th>Arteriolosclerosis</th>
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<tbody>
<tr>
<td>Affects the media of medium-sized arteries</td>
<td>Affects arterioles</td>
</tr>
<tr>
<td>Lesions (within media)</td>
<td>Lesions</td>
</tr>
<tr>
<td>- Hyaline degeneration</td>
<td>Concentric</td>
</tr>
<tr>
<td>- Fatty degeneration</td>
<td>lamellar intimal</td>
</tr>
<tr>
<td>- Fibrinoid necrosis</td>
<td>proliferations</td>
</tr>
<tr>
<td>- Mineralization</td>
<td></td>
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</tbody>
</table>

Degenerative Vascular Disease in Animals

**Dog**

- **Atherosclerosis**: Lipid accumulation occurs in the intima or media of muscular arteries secondary to hypercholesterolemia usually due to hypothyroidism, diabetes mellitus.
- **Medial sclerosis** and **amyloidosis** occurs in association with endocardiosis.
- Little or no evidence that these lesions have clinical or pathologic significance.
- Renal amyloidosis and Cushing’s disease predisposes to thromboembolic phenomena, especially **pulmonary artery thrombosis**.
- **Amyloidosis** – pulmonary and coronary arteries significance unknown.

**Pig**

- **Atherosclerosis** pattern mimics that of human beings.
- Proposed as best species for experimental study.
- Little or no evidence that these lesions have any clinical or pathologic Significance.

**Cow**

- **Intimal mineralization**
  - Johne’s disease
  - Aortic arch
  - Vitamin D and Vitamin D analog poisoning
  Widespread mineralization
Cat
- **Medial hypertrophy of the muscular pulmonary arteries**
  - May be associated with reaction to parasites such as *Paragonimus* or *Toxocara*. Has been reported in specific pathogen free cats.

*Inflammatory Diseases of the Vasculature*

**Definitions:**
- Periarteritis - inflammation of the adventitia.
- Polyarteritis - inflammation of many arteries.
- Phlebitis - inflammation of a vein.
- Vasculitis - inflammation of vessels (arteries and veins).

**Causes:**

*Viral*

Equine Viral Arteritis:
- Polyarteritis affecting the media and adventitia.
- Widespread petechiae, subcutaneous edema, hydrothorax, hydroperitoneum, hydropericardium.

Malignant Catarrhal Fever (MCF) of cattle:
- Polyarteritis and periarteritis.

Equine Infectious Anemia:
- Polyarteritis and periarteritis.

Bluetongue of sheep:
- Polyarteritis centered around the intima.
- See hemorrhage in at the origin (base) of pulmonary artery (intimal surface).

Feline Infectious Peritonitis - pyogranulomatous vasculitis.

Aleutian disease of Mink.

Hog Cholera of pigs.

*Bacterial*

*Salmonella sp* produces vasculitis.

*Histophilus somni* (thromboembolic meningoencephalitis, (TEM)).

*Parasitic*

*Strongylus vulgaris* in the horse.
- Fourth stage larval forms live in the intestinal arteries. Causes and intense focal inflammatory reaction in the walls of the larger arteries that results in aneurysmal dilation, thrombosis, and infarction distal to involved site (thromboembolic colic of horses).

*Dirofilaria immitis* in the dog.
- Adult worms live in the pulmonary arteries of dogs.
- Microfilaria are found throughout the circulation.
- Lesions:
  - villous pulmonary endoarteritis (inflammation of the intima).
  - medial sclerosis and hypertrophy.
  - obstruction and narrowing of the lumina produces increased pulmonary resistance causing right ventricular hypertrophy which many progress to right heart failure.

Other Parasites:
- Angiostrongylus vasorum
- Spirocerca lupi
- Schistosomiasis
- Elaeophoriasis
- Onchocerciasis
- Aelurostrongylus sp

Fungi (infrequent) fungi disseminate via vasculature (eg. guttural pouch mycosis).

Immune Mediated Vascular Disease
Systemic lupus erythematosis
Rheumatoid Arthritis
Polyarteritis nodosa
Staphloccocal hypersensitivity

Miscellaneous Diseases
Aneurysm:
- Dilatation and attenuation of a vessel wall.
- Often only the stretched intima and adventitia remain due to rupture of the media.
Significance: May occur 2E to degenerative or inflammatory disease of a vessel wall.
  - Aneurysms may rupture resulting in hemorrhage by rhexis.

Varix (varices)
Definition: An uneven, permanent dilatation of a vein.

Telangiectasia:
Definition: Marked dilatation of clusters of capillaries forming focal, red areas.
  - Occurs in livers – often cattle
  - Sometimes referred to as peliosis – especially in rat livers.

Post Caval Thrombosis: (Previously discussed in circulatory disturbances).
  - Occurs in cattle - usually feedlot - hepatic abscess ruptures into the post cava.

*Omphalophlebitis:
Definition: Inflammation of the umbilical arteries and vein.
Lesions: Inflammation of the umbilical stump, umbilical arteries, hepatic abscesses, peritonitis, arthritis.

Post-stenotic dilation: a phenomenon that regularly occurs when a large elastic artery becomes constricted in circumference. This is a sac like dilation that forms distal to the stenotic lesion.

Jet Lesions - secondary to excessive turbulence (see congenital anomaly notes).
NEOPLASMS OF THE CARDIOVASCULAR SYSTEM

Primary

*Hemangiosarcoma

Synonyms: hemangioendotheliasarcoma
Angioendotheliasarcoma

Locations:
Right atrium
- German Shepherd - often see metastasis to lung.
Spleen
- Most common site – but may occur anywhere.

Pronosis: May be locally invasive and often metastasize widely.

*Hemangioma

Synonyms: hemangioendothelioma
angioendothelioma

Benign neoplasm arising from endothelial cells.
Most commonly found within the skin.

*Heart Base Tumour

Synonyms: chemodectoma, aortic body tumor.
Incidence: usually brachycephalic dog breeds.
(Boxer, Boston Terriers) - rare in cat.
- Arises from the chemoreceptor cells in the adventitia of the aorta, pulmonary artery or base of heart.
- Ectopic thyroid tumors are also reported at this site.

Schwannoma (Neurofibroma)
- seen in cattle
- arises from nerves of epicardium, usually multicentric

Others
6 rhabdomyomas/rhabdomyosarcomas
6 myxoma/myxosarcoma

Secondary Neoplasms

*Lymphosarcoma - Often occurs within the left atrium of cattle. Heart is occasionally affected in dogs and cats with lymphosarcoma.

*Hemangiopericytoma - Tumor originating from pericytes.
- Spindle cell tumour (common name), dermal or subcutis in dogs.
### ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>ACM</td>
<td>Arrhythmogenic Cardiomyopathy</td>
</tr>
<tr>
<td>ASD</td>
<td>Atrial Septal Defect</td>
</tr>
<tr>
<td>CHF</td>
<td>Congestive Heart Failure</td>
</tr>
<tr>
<td>CVP</td>
<td>Central Venous Pressure</td>
</tr>
<tr>
<td>DCOM</td>
<td>Dilated Cardiomyopathy</td>
</tr>
<tr>
<td>HCOM</td>
<td>Hypertrophic Cardiomyopathy</td>
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<tr>
<td>L</td>
<td>Left</td>
</tr>
<tr>
<td>LV</td>
<td>Left Ventricle</td>
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<tr>
<td>LVF</td>
<td>Left Ventricular Failure</td>
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<tr>
<td>LVH</td>
<td>Left Ventricular Hypertrophy</td>
</tr>
<tr>
<td>PDA</td>
<td>Persistent Ductus Arteriosus</td>
</tr>
<tr>
<td>PRAA</td>
<td>Persistent Right Aortic Arch</td>
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<tr>
<td>R</td>
<td>Right</td>
</tr>
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<td>Interventricular Septal Defect</td>
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