CARDIOVASCULAR PATHOLOGY

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Unit 1 - Heart Failure

There is a web lesson, “Heart failure,” that accompanies these notes.

Normal:

Heart failure is the inability of the heart to maintain adequate circulation. It occurs when blood returning to the heart cannot be pumped out at a rate sufficient to meet the metabolic demands of the body. Period. It is the leading cause of death in people, largely due to atherosclerosis and the high prevalence of hypertension. Want more salt on them fries? Heart failure occurs in animals as well, but the incidence is much much lower.

Heart failure can be broken down into two broad categories, acute heart failure and chronic (or congestive) heart failure.

Acute heart failure refers to sudden, significant loss of cardiac function. It is a precipitous event leading to “brain death” within minutes. Causes of acute heart failure include:

- Destruction of big part of myocardium - infectious, toxic, vascular
- Dilated or hypertrophic cardiomyopathy
- Cardiac tamponade
- Severe electrolyte imbalance

With all of these causes, often there are no premonitory signs of disease. Myocardial ischemia, which is the cause of acute heart failure (“heart attack”) in humans, is seen much less frequently in animals.
**Congestive heart failure**, also known as **chronic heart failure**, is the result of failure of the heart to maintain adequate circulation over a period of time. General causes of chronic heart failure include:

- **sustained systolic pressure overload**, such as occurs in systemic or pulmonary hypertension, or stenosis of one of the heart valves.
- **sustained volume overload** on one or both ventricles imposed by conditions such as mitral valvular insufficiency (mitral valve is leaky) or ventricular septal defects (hole in the wall).
- **loss of myocardial contractile capacity** through various mechanisms, such as a disease of the myocardium.
- **interference with ventricular filling during diastole**, such as may occur with cardiac tamponade, severe pericardial fibrosis, or hypertrophic cardiomyopathy.

In addition, congestive heart failure is often further divided into right-sided or left-sided heart failure, depending upon which ventricle is preferentially affected. However, failure on one side usually leads to failure on the other, so it doesn’t matter all that much.

The body makes many **adjustments**, or **compensations**, when faced with chronic heart failure. These adjustments to chronic heart failure can be divided into two main categories - *peripheral* and *cardiac*.

**Peripheral adjustments** for insufficient cardiac output would include trying to maintain blood flow to vital organs, increasing the amount of red blood cells to help with oxygenation, and controlling the blood volume to decrease the load on the heart. In more detail,

**Cardiac adjustments** to heart failure are those compensations that allow the heart to respond to circulatory demands over and above those of the normal animal at rest. Components of this cardiac reserve are the following -

- **Increased heart rate** increases cardiac output. The time required to empty the ventricles during systole is relatively fixed and cannot be shortened significantly, so an increased heart rate is accomplished primarily by shortening the diastolic interval.
At very rapid heart rates, there may not be enough time for complete ventricular filling during diastole and the cardiac output may suffer.

♥ Chamber dilation allows increased filling during diastole, increased stretching of myocardial fibers, resulting in increased stroke volume. At some point, however, chamber dilation becomes excessive - the myofibers are “overstretched,” and the efficiency of contraction decreases.

♥ Myocardial hypertrophy is a common adaptation to chronically increased tension on myocardial fibers caused by systolic mechanical overloading or impaired contractility. The increased muscle mass is due to increased size, length, and diameter of myofibers. Sooner or later they enlarge to the point that they are nonfunctional (can’t get oxygen through to where it needs to go). There are two patterns of ventricular hypertrophy.

*****The expected response of the heart to systolic pressure overload is concentric hypertrophy, while the response to diastolic volume overload is eccentric hypertrophy.*****

Eccentric hypertrophy (volume overload hypertrophy) occurs in response to chronic volume-overloading such as may be caused by, for example, valvular insufficiencies or septal defects. Eccentric hypertrophy is characterized by increased ventricular chamber volume with normal or decreased ventricular wall thickness. It seems odd to even call this a hypertrophy, because the walls are either normal thickness or thinner than normal. However, the total cardiac weight is increased due to a greater muscle mass surrounding the dilated chamber.
Concentric hypertrophy (pressure overload hypertrophy) occurs in response to chronic pressure-overloading caused by, for example, systemic or pulmonary hypertension or aortic/pulmonic stenosis. Concentric myocardial hypertrophy is characterized by increased ventricular wall thickness and decreased ventricular chamber volume. Because decreased chamber size reduces the diastolic volume, concentric hypertrophy must be accompanied by a compensatory increase in heart rate in order to maintain cardiac output.

Also, remember that failure of one side of the heart eventually leads to failure of the other side, if the animal lives long enough. So, both sides could end up hypertrophied.

Signs and lesions of cardiac disease

As mentioned previously, acute heart failure may have no premonitory signs. The only history may be sudden death. In addition, in very acute heart failure, although death was due to myocardial dysfunction, there may be no morphologic lesions to accompany the functional changes. This is because it takes several hours for physical changes to be visible in the cells after they have stopped working. This happens commonly in anesthetic overdose deaths - an animal dies under anesthesia due to heart failure yet there are no lesions to confirm this cause of death.

The lesions associated with congestive heart failure depend on whether the primary defect is in the right side or the left side of the heart.

Right heart failure (RHF) results in

- **Chronic passive hepatic congestion** is typified by a large, congested liver, occasionally with fibrin on the surface. The lobular pattern is enhanced and so this appearance is often termed “nutmeg liver.” The enhanced lobular pattern is due to blood backing up in the centrilobular area of each lobule - this causes necrosis of the centrilobular hepatocytes, more room for blood to back up, and a distinct dark area grossly at the central portion of each lobule. Chronically affected livers occasionally develop centrilobular fibrosis, a change known as “cardiac cirrhosis.” Other congested tissues can include spleen,
stomach, and intestines. Gastrointestinal congestion may impair function, resulting in diarrhea.

- **Edema and body cavity effusions** are often seen in right heart failure. In cattle and horses, edema is seen most commonly in dependent subcutaneous tissues, resulting in “bottle jaw edema” or brisket edema. In dogs and cats, edema of RHF shows up as ascites.

**Left heart failure** (LHF) shows up as pulmonary congestion and edema. This can produce severe dyspnea. Grossly the lungs are heavy, wet, and red. White foam may fill the airways. Chronic passive congestion of the lung can result in brownish discoloration of the lungs due to the presence of hemosiderin-laden alveolar macrophages (“heart failure cells”). What do we call this? – hemosiderosis.
Unit 2 - Lesions and Conditions involving the Pericardium

There is a web lesson, “Lesions and conditions involving the pericardium,” that accompanies these notes.

Nomenclature
The pericardial sac surrounds the heart. The mesothelial lining of the inside of the sac is the parietal pericardium and the lining of the outside surface of the heart is the visceral pericardium. Visceral pericardium is synonymous with epicardium. The space between the parietal pericardium and visceral pericardium is normally lubricated by a small quantity of fluid produced by the mesothelial cells. (Mesothelial cells are cells that line the mesentery, pleura and pericardium.)

Pericardial effusions and exudates
Materials accumulating rapidly within the pericardial sac can impinge on venous return leading to cardiovascular collapse and rapid death, a situation known as “cardiac tamponade.” Materials accumulating slowly are accommodated by stretching of the pericardium and may achieve tremendous volumes without significantly affecting venous return.

How much fluid is NORMAL in the pericardial sac? After all, there must be SOME fluid in there or the pericardium would scrape against the heart wall every time it beats. Ouch. An adult human has about 25 ml of clear pericardial fluid. You can use an imaginative sliding scale to figure out about how much is normal in animals of varying weights.
Types of effusions include:

- **Hydropericardium** is a serous/serosanguinous effusion within the pericardial sac. The fluid is clear, straw-colored, with low protein content, without flecks of fibrin, and does not clot upon exposure to air. Causes include congestive heart failure, pulmonary hypertension (e.g., high altitude disease of cattle), hypoproteinemia (cachexia, parasitism), and neoplasia (tumor causing lymphatic obstruction).

- **Hemopericardium** is an effusion of pure blood into the pericardial sac. Causes include: cardiac puncture, such as might occur with a fractured rib or gunshot wound; aortic rupture, which happens uncommonly in horses; ulcerative atrial endocarditis with mural perforation and rupture of a coronary artery occasionally occurs in dogs secondary to uremia; rupture of a cardiac hemangiosarcoma or heart base tumor. Hemopericardium is usually life-threatening. The sudden escape of blood into the pericardial sac leads to interference with cardiac filling and heart failure, which is referred to as cardiac tamponade.

- **Fibrinous effusions and fibrinous pericarditis** consist of fluid within the pericardial sac that clots soon after exposure to air. This is an indication of fibrinogen-rich exudate and therefore reflective of vascular damage. Fibrinous pericarditis is the accumulation of clotted fibrin on pericardial surfaces. The amount of fibrin can vary from a fine net to great masses that fill the pericardial sac and give the pericardial surfaces a shaggy appearance. There’s almost always a bacteria responsible (except cats where it is usually FIP). The bacteria differ according to the animal species.

*What happens to these fibrinous effusions?* Slight fibrinous exudates are removed by fibrinolysis and the mesothelial lining regenerates. More extensive fibrinous exudates are organized into granulation tissue, eventually forming fibrous adhesions between the visceral and parietal pericardium. There is generally little deleterious effect from the presence of small amounts of fibrin or fibrous connective tissues involving the pericardial surfaces. Extensive pericardial fibrosis can prohibit diastolic filling of the heart, which will cause heart failure.
Purulent pericarditis is the accumulation of pus (and generally fibrin as well) in the pericardial sac. It indicates the presence of pyogenic organisms and occurs with frequency only in cattle with traumatic reticulo-pericarditis. For a refresher, take a cerebral journey back to General Pathology where we covered how this happened. Cow eats nail. Nail lands in reticulum. Reticulum contracts. Nail pokes through reticulum and diaphragm and into pericardium. Bergey’s Manual (which is a huge volume categorizing all known species of bacteria) gets free ride on nail. The amount of exudate can vary from a thin film to more than a gallon. The color can vary from a yellow to gray to green to brown depending on the type of bacteria and erythrocyte contamination. The consistency can vary from watery to a thick fluid.

**What is the fate of purulent pericarditis?** The exudate is organized into granulation tissue which matures into fibrous connective tissue that may encase the heart in a layer several millimeters thick. Complete obliteration of the pericardial space by fibrous adhesions may occur and that is bad news. The progressive fibrous constriction of the heart interferes with diastolic filling and can eventually lead to congestive heart failure and death.

**Other epicardial lesions**

*Serous atrophy of epicardial fat* occurs in starvation. The fat along the coronary grooves becomes a clear to translucent gelatinous substance that may be flecked with small white foci of fat necrosis. This lesion is seen with cachexia of any cause. There is progressive mobilization of depot fat, which is accompanied by an increase in interstitial fluid within the fat.

*Epicardial hemorrhages* are commonly seen in horses, cattle, sheep, and swine dying from a variety of diseases (the lesion is common and often nonspecific with regard to cause). Pericardial hemorrhages are less commonly seen in dogs and cats. The type of hemorrhage varies from petechial to suffusive. They may occur anywhere over the surface of the heart but are most common at the base and along the course of the major coronary vessels. Specific causes known to engender epicardial hemorrhage include: trauma in any species; mulberry heart disease in swine (lack of Vitamin E decreases vascular integrity); warfarin toxicity which impairs coagulation; septicemia which is associated with bacterial toxins that damage blood vessels; hypoxia.

*Epicardial urate deposition* (visceral gout) occurs most commonly in birds and reptiles and is recognized grossly as white to gray granular epicardial precipitates.
Unit 3 - Endocardial Lesions

There is a web lesson, “Endocardial lesions,” that accompanies these notes.

**Endocardial hemorrhages** occur frequently in horses, sheep, and cattle, but less frequently in dogs and cats. Sometimes these hemorrhages are fairly extensive, but in general they are considered to be agonal, i.e., occurring at the time of death and not necessarily the reason for death.

**Endocardial mineralization** may occur in association with a number of conditions. As part of uremic endocarditis in dogs, the left atrial endocardium can develop firm, gritty, roughened areas which are almost pathognomonic for uremia. Debilitating conditions in cattle such as Johne’s disease cause mineralization in the aorta and also in the left heart. Myocardial degeneration in lambs with white muscle disease, which is due to a vitamin E/selenium deficiency, may result in endocardial mineralization. **Hypervitaminosis D**, due to dietary supplementation or plant poisoning, may lead to mineralization in multiple sites, including endocardium.

**Valvular hematocysts** are soft to turgid, red (occasionally pale), blood-filled cysts within valve leaflets. They occur commonly in young dogs and calves and less often in other animals. They are usually of no significance; rarely are they large enough to interfere with valve function.

**Valvular endocardiosis**, also known as *myxomatous degeneration*, is the most common cardiovascular lesion encountered in dogs. The incidence increases with age and the left atrioventricular valve is most frequently affected. Grossly, the lesion appears as smooth, sometimes nodular thickenings of valve leaflets.

****Valvular endocardiosis is the most common cause of mitral systolic murmurs in dogs****
In endocardiosis, the valve leaflets, instead of being nice and flat and thin at the ends, become bulbous and rounded – this keeps the valves from closing completely and uh-oh, insufficiency….

Atrial or ventricular thrombosis can occur in dogs and cats with cardiomyopathy. Turbulent blood flow in the abnormally contracting heart predisposes to thrombus formation.

Endocarditis is endocardial inflammation. It occurs rarely on the wall of the heart chambers ("mural endocarditis") and far more frequently on the valves ("valvular endocarditis") to the point where the term endocarditis usually implies inflammation of the valves. Why is inflammation so much more common on the valves? - probably because they are subjected to much more trauma and turbulent blood flow.

Inciting causes include trauma (valvular abnormalities), irritants (uremic toxins), bacteria, or parasites (especially strongyles). *Streptococcus* or *Staphylococcus* are common infecting agents in most species.

Lesions frequently develop on the edges of valve leaflets at sites of endothelial loss secondary to trauma caused by the apposing leaflets. They usually develop on the valve surface exposed to the forward flow of blood ("dorsal" surface of a-v valves, "ventral" surface of semilunar valves). Gross lesions begin as small ulcers (reddened defects in the endothelial lining) that become covered with fibrin. Yellow-gray to yellow-red friable (crumbly) debris then accumulates. Endocarditis is most common on atrioventricular valves in large animals. In dogs, it is usually seen on the aortic valve.

Sustained or recurring bacteremia is generally required for lesion development. Bacteria sequestered within the lesion are safe from antibiotic therapy because they are protected
by their furry wall of fibrin and thrombosis. Lesions that are initially nonseptic (such as uremic ulcers) are prone to bacterial colonization.

The critical observation used to distinguish the endocarditis lesion from other valvular changes (especially valvular endocardiosis in dogs) is the roughened surface, which indicates loss of the overlying endothelial layer.

What are the consequences of valvular endocarditis?

- Valvular insufficiency could lead to heart failure.
- Stenosis of the orifice could occur in one of two ways. The valves could become scarred and contracted, or a large vegetative mass could partially occlude the valve opening.
- Embolism could result as dislodgement of the thrombotic material gets carried downstream. Emboli from right heart lesions arrest in the lungs to form pulmonary abscesses. Pulmonary infarcts may occur (although the lung’s dual blood supply makes this organ relatively difficult to infarct). Emboli from left heart lesions disseminate into the systemic circulation, frequently producing myocardial, renal and/or splenic infarcts or abscesses.

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There are a number of congenital cardiac anomalies that are included in this unit, not because they are specifically endocardial in nature, but because they are usually encountered during examination of the endocardium.

Valvular stenosis is seen as a congenital problem, especially in dogs. It may be the aorta, or it may be the pulmonic valve. The opening is too small (this is what stenosis means) and the animal is just born that way. These dogs usually die by about 6 months of age.

Septal defects would also be identified during evaluation of the endocardial aspect of the heart.
Ventricular septal defect (VSD) is a small opening in the dorsal part of the interventricular septum, caused by malformation of structures that normally fuse to form the septum. VSD’s occur most commonly “high” under the septal cusp of the right atrioventricular valve when viewed from the right side and in the left ventricular outlet when viewed from the left side (i.e., subaortic interventricular septal defect). Small VSD’s may be missed if one does not look under the septal cusps of the atrioventricular valves.

Blood usually flows from left to right through a VSD due to higher LV pressures. This will cause right ventricular overload, with resulting eccentric hypertrophy. Following right ventricular hypertrophy, blood flow may reverse to a right to left direction (causing cyanosis). Effects of a VSD usually are not apparent at birth. If the defect is large enough, clinical signs are progressive. A precipitous decline occurs as the right ventricle hypertrophies to the point that blood goes from right to left, contributing unoxygenated venous blood to the systemic circulation.

Atrial septal defect (ASD) usually occurs at the location of the foramen ovale. Remember? - the foramen ovale is the channel between the two atria which in fetal life, allows oxygenated blood to flow from the right atrium to the left atrium because of greater pressure in the right atrium. After birth, with a reversal of the pressure gradient, the valvular opening is closed and the two membranes usually fuse. Simple lack of fusion of the interatrial septa does not produce a functional defect because pressure differences keep the flap shut. Such a situation is encountered very commonly in neonatal calves. A more serious form of ASD can occur due to failure in
closing of the ostium primum or failure in the separation of the pulmonary veins and the anterior vena cava. Either one of these can leave a bona fide hole in the interatrial septum. Because of higher pressure on the left side, blood gets shunted left to right, ultimately causing right ventricular hypertrophy and all its sequelae.

Associated changes can include systolic murmurs, ventricular hypertrophy (eventually biventricular) and areas of subendocardial fibrotic thickening ("jet lesions") at sites where shunting blood collides with the ventricular wall.
Unit 4 - Abnormalities of Myocardium

There is a web lesson, “Abnormalities of myocardium,” that accompanies these notes.

Abnormalities of cardiac size and shape

Normal - Some time after death cardiac muscle undergoes rigor mortis, which tends to expel most of the blood from the heavily muscled left ventricle but usually leaves blood in the right ventricular chamber. Relaxation follows the rigor and the right ventricle usually seems slightly flaccid compared to the left ventricle.

Dilation and hypertrophy - Within certain limits, both dilation and hypertrophy are normal physiologic or compensatory responses of the heart to increased workload elicited by such factors as exercise, chronic hypoxia, excess thyroid hormone, and excess catecholamines. Physiologic dilation and hypertrophy can enhance cardiac function, up to a point.

Hypertrophy initially improves cardiac output, but continued hypertrophy eventually reduces myofiber contractility and diminishes cardiac function. This can be attributed, in part, to reduction in the mitochondrial to myofibrillar volume ratio and to failure of coronary vascular development to keep pace with the increased myocardial mass. Further, hypertrophied myocardium is more susceptible to ischemia than normal myocardium.

Patterns and causes of cardiac hypertrophy:

Central eccentric hypertrophy

also known as volume overload hypertrophy, as previously discussed, is recognized by increased chamber size and walls that are thin or normal in thickness. The cardiac silhouette is rounder than normal. The severe end-stage change is a markedly rounded and thin-walled or “globoid” heart.

Eccentric hypertrophy is associated with the following causes:
- a severe valvular insufficiency
- an atrial or ventricular septal defect
also known as *pressure overload hypertrophy*, is recognized by decreased chamber size and thickened walls. The heart is usually elongated and somewhat valentine-shaped.

Concentric hypertrophy is associated with the following causes:
- pulmonic and/or aortic stenosis
- pulmonary and/or systemic hypertension
- hyperthyroidism in an old cat

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**Primary Cardiomyopathies**

There is a whole category of “poorly functioning myocardiums WITHOUT a known cause” – these are called cardiomyopathies. They occur mostly in dogs and cats and we won’t focus on them in our distance pathology course because we are more concerned with diseases of food producing animals.

**Lesions associated with myocardial degeneration and/or cellular infiltrates**

Lesions may be large or very small, as well as focal, multifocal, disseminated, or diffuse. Lesions causing significant vascular damage will be red (hemorrhagic) and could be primarily necrotic (e.g., white muscle disease in young ruminants), inflammatory (e.g., blackleg in cattle), or neoplastic (e.g., hemangiosarcoma in dogs). Lesions of myocardial degeneration or non-vascular neoplasia are usually pale (white, tan, or gray). It is often not possible to distinguish gross lesions of myocardial degeneration/necrosis from those caused by inflammatory or neoplastic infiltrates. If a lesion is clearly raised or nodular, it probably involves an infiltrate of some kind.
Causes of myocardial degeneration/necrosis are varied:

- **Metabolic changes** include nutritional deficiencies such as vitamin E/selenium-responsive syndromes (white muscle disease and mulberry heart disease), exertional myopathies, monensin in horses, and coffeeweed (*Cassia occidentalis*) in ruminants.

  In white muscle disease, there is not enough Vitamin E or selenium to stabilize membranes and the membranes of muscles (especially heart) undergo rapid degeneration. They also mineralize quickly with this disease.

- **Circulatory disturbances**, i.e., ischemia and infarction, occur in a variety of ways.

  *Hypertrophied myocardium* has increased susceptibility to ischemia and infarction just because the heart myofibers can’t multiply, they can only get bigger. So oxygen has to find its way further into the cell. Infarction may occur as the result of *emboli* from vegetative endocarditis. *Arteriosclerosis*, including atherosclerosis, is common in animals but seldom severe enough to lead to myocardial infarction, as occurs frequently in humans.

- Some *infectious agents* may cause significant myocardial necrosis without a lot of cellular inflammation. These are usually very acute infections - foot-and-mouth disease in young ruminants, canine parvovirus in neonatal puppies, and blackleg in cattle. In the first two examples, necrosis is due to overwhelming and acute infection of cardiac myofibers by the virus. In the latter example, the necrosis of the myocardium is caused by bacterial exotoxins.

- Some *systemic conditions* such as septicemia, toxemia and anemia can lead to myocardial degeneration. In these cases, much of the degeneration is due to hypoxia or vascular effects.
Inflammatory infiltrates (myocarditis)

★ Myocarditis occurs most commonly as a manifestation of systemic disease rather than as an isolated lesion. Etiologic agents are numerous and include bacteria, viruses, fungi, protozoa, algae, and parasites. Myocardial involvement may occur via direct extension from a pericarditis/endocarditis or by hematogenous spread of infectious agents.

★ Microscopically, inflammatory lesions are classified in the usual manner (purulent, granulomatous, etc.) and the character of the infiltrate is often a reflection of the type of etiologic agent. For instance, purulent myocarditis is associated with bacteria, lymphocytic myocarditis with viruses, and granulomatous myocarditis with higher bacteria and fungi.
Unit 5 - Vascular Lesions

There is a web lesson, “Vascular lesions,” that accompanies these notes.

Congenital malformations of the vascular system

Tetralogy of Fallot is consists of FOUR heart defects, all arising from the same underlying problem, which is embryonic failure of development of the interventricular septum and displacement of the conal septum, leading to an overriding aorta and obstruction of the right ventricular outflow. Sounds complicated.

Here are the four problems you will end up seeing in the affected heart:

1. ventricular septal defect
2. aorta that overrides the ventricular septal defect (biventricular origin of the aorta, dextroposition of the aortic valve)
3. pulmonary or subpulmonic stenosis causing obstruction of right ventricular outflow
   (1, 2, and 3 are all basically the same problem – the great vessels don’t get situated properly on the septum.)
4. compensatory hypertrophy of the right ventricle
With T of F, animals are usually cyanotic. As the right ventricle hypertrophies, there is eventually right to left shunting and unoxygenated blood going to the systemic circulation. Not good.

**Morphologic reactions of injured vessels**

*Mild injury* results in endothelial cell swelling and hypertrophy.

*Vascular hyalinosis* is a term that refers to an increased, homogeneous eosinophilia of the vessel wall. When severe, it is called fibrinoid necrosis and is seen in uremia and edema disease of baby pigs.

*Mineralization of vessel walls* may be either “dystrophic”, in vessels damaged by degenerative or inflammatory processes, or “metastatic” during conditions of hypercalcemia and/or hyperphosphatemia.

*Thrombosis* occurs when hemostasis is disturbed in favor of coagulation. Predisposing factors for thrombosis are: endothelial injury, stasis or turbulence of blood flow, and hypercoagulability of blood. Remember all this from the pink pages in General Pathology? …Life is cumulative….

**Examples:**

- “saddle thrombus” in cats with cardiomyopathy. A thrombus forms in the left atrium due to turbulence or stasis of blood, and embolizes to the terminal aorta and iliac arteries.
- disseminated intravascular coagulation (DIC)
- jugular vein thrombosis due to venipuncture
- cranial mesenteric arteritis in horses infested with *Strongylus vulgaris*

- renal amyloidosis in dogs causes hypercoagulation due to depletion of antithrombin III
- thrombosis of femoral tributaries in “downer” cows
Do you remember the difference between antemortem thrombi and postmortem clots?

Antemortem thrombi are attached to the vessel wall at some point and are usually friable and dull red. Postmortem clots are not attached, are soft (not friable), and glistening (not dull).

Vasculitis

Vasculitis is defined as an inflammation of vessels characterized by presence of inflammatory cells within and around vessel walls with concomitant damage to the vessel wall. Lesions can be more specifically designated as arteritis (arteries), phlebitis (veins), or omphalophlebitis (umbilical veins).

Vasculitis is commonly associated with septicemic, viremic, and toxemic diseases and may have a major role in lesion development of some diseases. Endothelial cell damage is a primary factor in the pathogenesis of many diseases including, among others: canine herpes, infectious canine hepatitis, classical swine fever, heartwater, African horse sickness, epizootic hemorrhagic disease, and bluetongue. Also, vessel damage may be the most critical lesion in endotoxemia. The vessel damage in feline infectious peritonitis and systemic lupus erythematosus is indirect, and the result of immune complex deposition. Lesions associated with vasculitis vary and, depending on the damage, can be seen as serous, fibrinous, or hemorrhagic foci around the vessels. If damage is very prolonged, the vessel may respond by intimal proliferation. This is seen in pulmonary villous endarteritis in dogs with heartworm disease.

SEE how VASCULITIS can lead to THROMBOSIS?????

The wall of the vessel is disrupted, so cells stick there, and before you know it, there is a thrombus…. BAD NEWS.
Arteriosclerosis is an umbrella term for several types of changes that cause hardening of arteries. Arteriosclerosis without lipid deposition can occur due to increased connective tissue, vascular mineralization (Johne’s disease), or hyaline degeneration (coronary arteries of old dogs). Arteriosclerosis with lipid deposition, also known as atherosclerosis, is common in humans but infrequent in animals. An exception is atherosclerosis of myocardial, cerebral and renal vessels in dogs that have hypercholesterolemia due to hypothyroidism. In humans, these atherosclerotic plaques occur in the intima and are easily dislodged, causing an infarctive episode downstream. As serious as a heart attack. However, in dogs, when it occurs, the atherosclerotic plaque is in the tunica media or adventitia and so is much less likely to dislodge.
The diagram above represents how LDL’s facilitate lipid accumulation in plaque and HDL’s help to purge cholesterol from the plaque. “HDL’s good, LDL’s bad.” To help you remember, think of Napoleon the pig in George Orwell’s *Animal Farm*, “Four legs good, two legs bad.”

**Aneurysms**

An aneurysm is a focal abnormal dilation of a blood vessel (usually one under high pressure like an artery). A true aneurysm results from thinning of the tunica media and occurs rarely in animals. In dissecting aneurysms, blood under high pressure enters the vessel wall through an intimal tear and creates a separation in the layers of the tunica media. The significance is that aneurysms are susceptible to rupture, resulting in significant hemorrhage since the blood is under high pressure.