

Cardiovascular System Pathology

Dr. Gzde YCEL TENEKECI

The Cardiovascular System

- A closed system of the heart and blood vessels
 - The heart pumps blood
 - Blood vessels allow blood to circulate to all parts of the body
- The function of the cardiovascular system is to deliver oxygen and nutrients and to remove carbon dioxide and other waste products

The Heart: Coverings

- Pericardium – a double serous membrane
 - Visceral pericardium - Next to heart
 - Parietal pericardium - Outside layer
- Serous fluid fills the space between the layers of pericardium

The Heart Wall: 3 layers

- Epicardium
 - Outside layer
 - This layer is the visceral pericardium
 - Connective tissue layer
- Myocardium
 - Middle layer
 - Mostly cardiac muscle
- Endocardium
 - Inner layer
 - Endothelium

The Heart: Chambers

- Right and left side act as separate pumps
- Four chambers
 - Atria
 - Receiving chambers
 - Right atrium
 - Left atrium
 - Ventricles
 - Discharging chambers
 - Right ventricle
 - Left ventricle

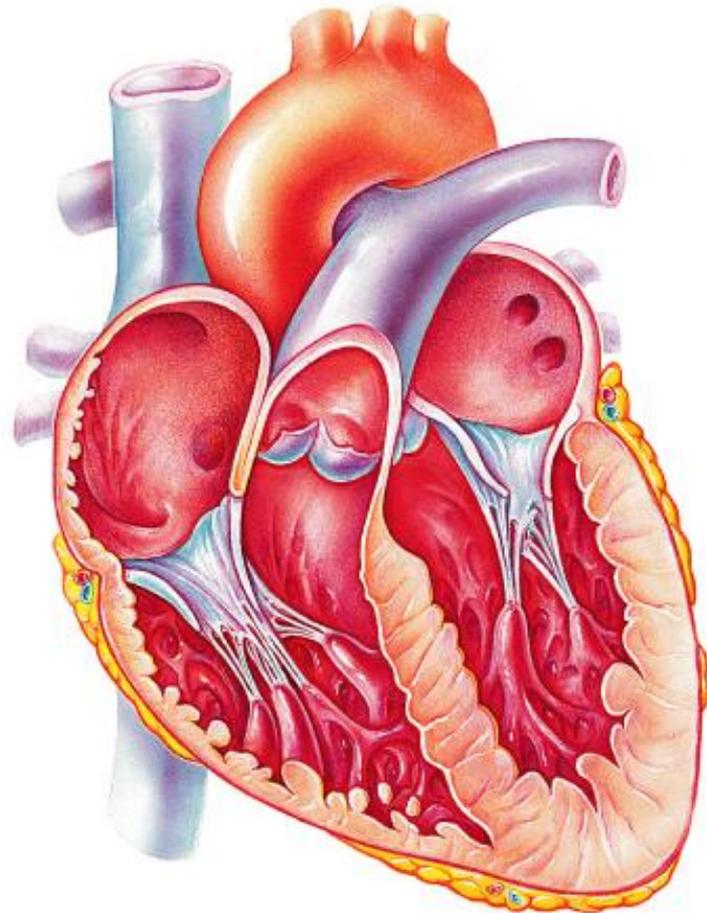
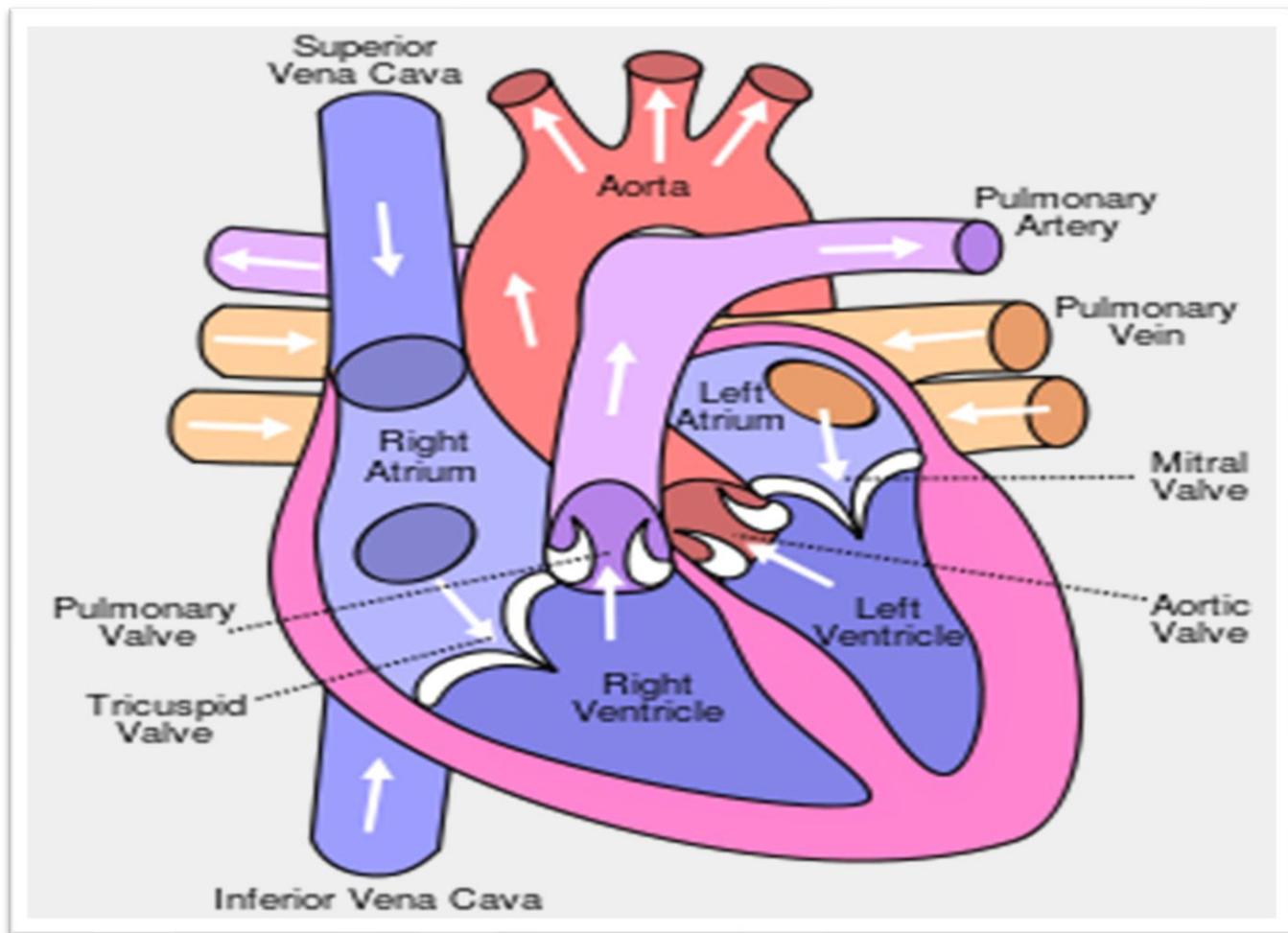
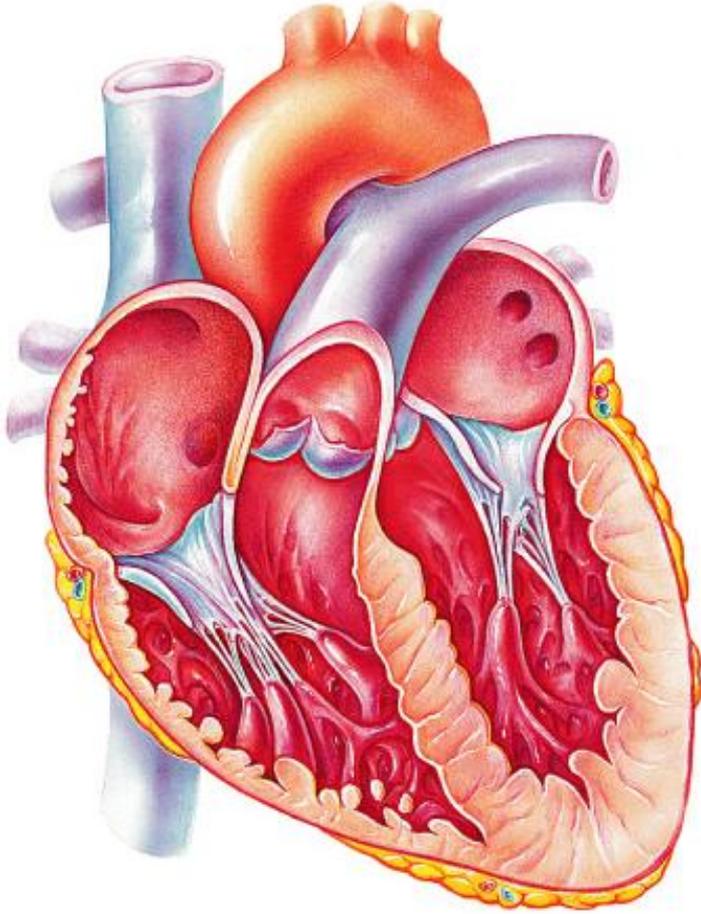


Figure 11.2c



- The pathology of the cardiovascular system is examined in the following order;
- I. Heart sac and heart pathology,
- II. Pathology of blood vessels,
- III. Pathology of lymph vessels.



HEART SAC AND HEART PATHOLOGY

Heart and heart sac anomalies

Acardia:

- This is the **absence of the heart** and the heart sac. This can be seen in one of the adjacent twins.

Hemiocardius:

- A twin fetus with a **rudimentarily formed heart**.

Acardius acephalus: The most common form, **with absence of the head, thorax and upper extremities**.

Acardius amorphus

- The least differentiated form, in which only **bones, cartilage, muscle, fat, and blood vessels are seen**.

Acardius acornus:

- This form lacks a thorax, with the umbilical cord insertion in the head. Despite the **absent heart, rudiments of thoracic structures** are often found.

Heart and heart sac anomalies

- **Ectopia cordis:** the heart may lie in *extrathoracic, presternal, or intra-abdominal positions*.
- In this case, the heart is either in the pericardium or completely naked.

Heart and heart sac anomalies

- Several locations of the aberrant heart have been described, including **cervical, abdominal** and, **pectoral**
- Ectopia cordis cervicalis
- Ectopia cordis abdominalis
- Ectopia cordis pectoralis

Heart and heart sac anomalies

Cor biloculare:

- A two-chambered heart.
Cor biloculare is due to failure of development of the walls that normally separate the two atria (interatrial septum) and the two ventricles (interventricular septum).



Cor triloculare biatriorum:

- Three-chambered heart
- Due to absence of the inter-ventricular septum.



Heart and heart sac anomalies

Persistent truncus arteriosus:

- Is a congenital heart disease that presents at birth.
- In this condition, **fails to divide into the pulmonary trunk and aorta.**
- This results in **one arterial trunk arising from the heart** and providing mixed blood to the coronary arteries, pulmonary arteries, and systemic circulation.

Heart and heart sac anomalies

Transposition of the great arteries:

- Is a congenital heart defect.
- In transposition of the great arteries,
- **The aorta** is connected to the **right ventricle**, and **the pulmonary artery** is connected to the **left ventricle**.

Failure of Closure of Fetal Cardiovascular Shunts

Interventricular Septal Defect.

- A ventricular septal defect indicates **failure of complete development of the interventricular septum** and allows the shunting of blood between the ventricles.
- The defect occurs in many species.
- Among breeds of dogs, English bulldog, English springer spaniel, and West Highland white terrier.

Failure of Closure of Fetal Cardiovascular Shunts

Atrial Septal Defect.

- **The failure of closure of the foramen ovale,**
- Faulty development of the **interatrial septum.**
- Although this defect occurs in all domestic animal species, dog breeds with greatest frequency of this defect are the boxer, Doberman pinscher, and Samoyed.

Failure of Closure of Fetal Cardiovascular Shunts

- In 1888 Fallot described in detail the four anatomical characteristics of **tetralogy of Fallot**, a congenital heart defect responsible for blue baby syndrome.

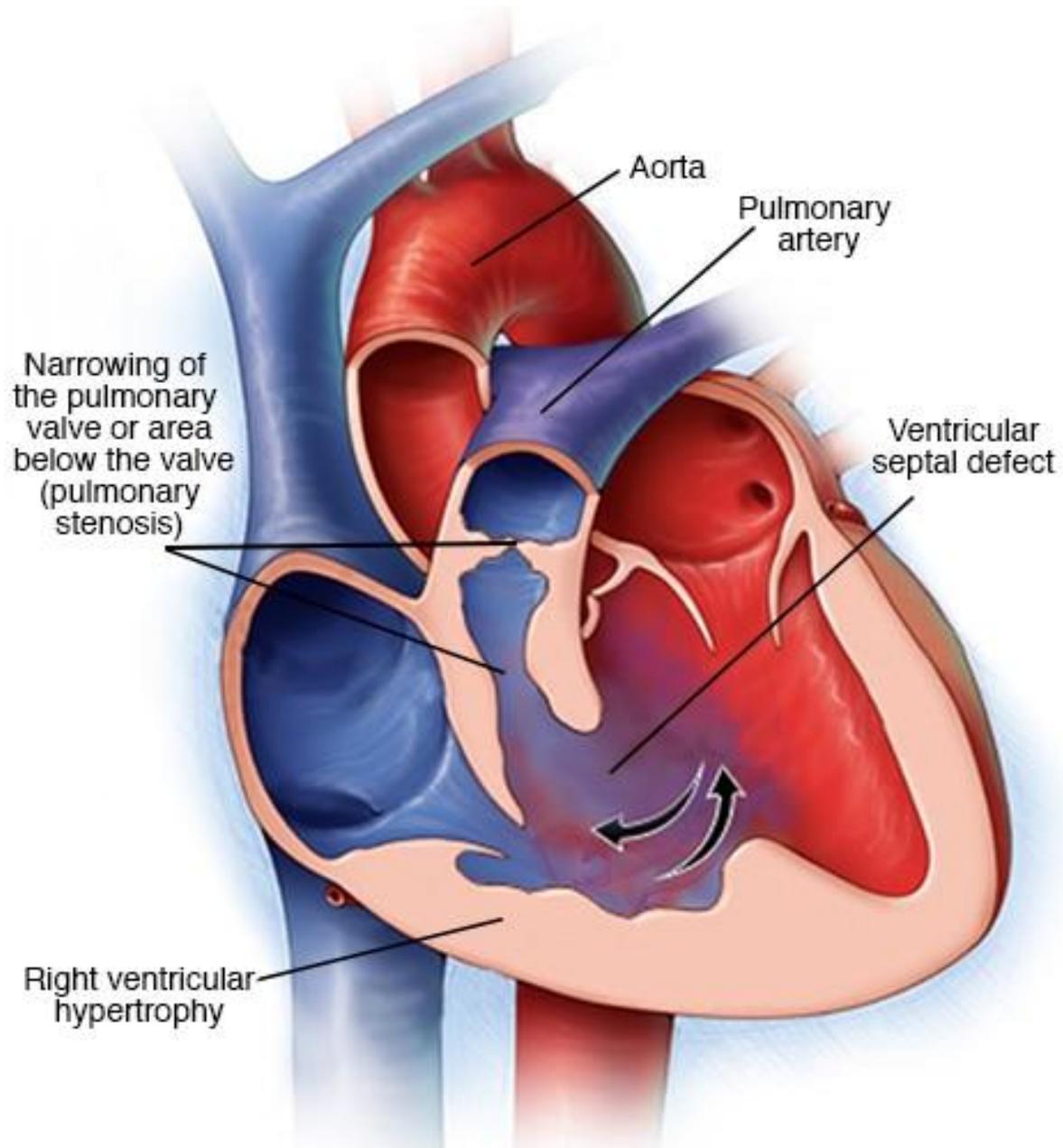
Heart and heart sac anomalies

Trilogy of Fallot:

- 1. Atrial septal defect (patent foramen ovale)** - Interatrial connection caused by an opening in the atrial septum causes right-to-left shunting of blood.
 - 2. Pulmonary valve stenosis**
 - 3. Hypertrophy of the right ventricle** - As a result of the increased effort needed to propel the blood through the stenotic pulmonary valve, hypertrophy of this cardiac chamber occurs and completes the trilogy of Fallot.
- Cyanosis is typically encountered in patients suffering from Trilogy of Fallot.

Failure of Closure of Fetal Cardiovascular Shunts

- ***Tetralogy of Fallot:***
- Is a combination of four congenital abnormalities.
- The four defects include:
 1. **A ventricular septal defect (VSD),**
 2. **Pulmonary valve stenosis,**
 3. **Overriding aorta**
 4. **Right ventricular hypertrophy** (A thickened right ventricular wall).
- Tetralogy of Fallot is a complicated cardiac anomaly seen in all animal species with four lesions



Failure of Closure of Fetal Cardiovascular Shunts

- **Eisenmenger complex:**
- The wall between the ventricles is open (*Ventricular septal defect*)
- *Displacement of the aorta and pulmonary artery*
- *Hypertrophy of the right ventricle .*
- Excessive cyanosis

Failure of Normal Valvular Development

Pulmonic Stenosis.

- Pulmonary stenosis (also called **pulmonic stenosis**) is when the **pulmonary valve** (the valve between the right ventricle and the pulmonary artery) is too small, **narrow**, or stiff.
- Pulmonic stenosis has been recognized as a **frequently occurring anomaly in dogs** and is inherited in the beagle.
- Other breeds in which this lesion is frequent are basset hound, boxer, Chihuahua, Chow Chow, cocker spaniel, English bulldog, Labrador retriever, mastiff, Newfoundland, Samoyed, schnauzer, and terrier.

PERICARDIAL DISEASE

- Non-inflammatory lesions of the pericardium
- ***Hydropericardium:*** The pericardial sac normally contains a very small quantity of clear, serous fluid.
- *Any excess in the volume of clear fluid is referred to as hydropericardium.*

Hydropericardium

- Hydropericardium is often part of *generalized anasarca* and is thus seen in many **cachectic** illnesses, perhaps as the result of **hypoalbuminemia**, and in **congestive heart failure**

Hemopericardium

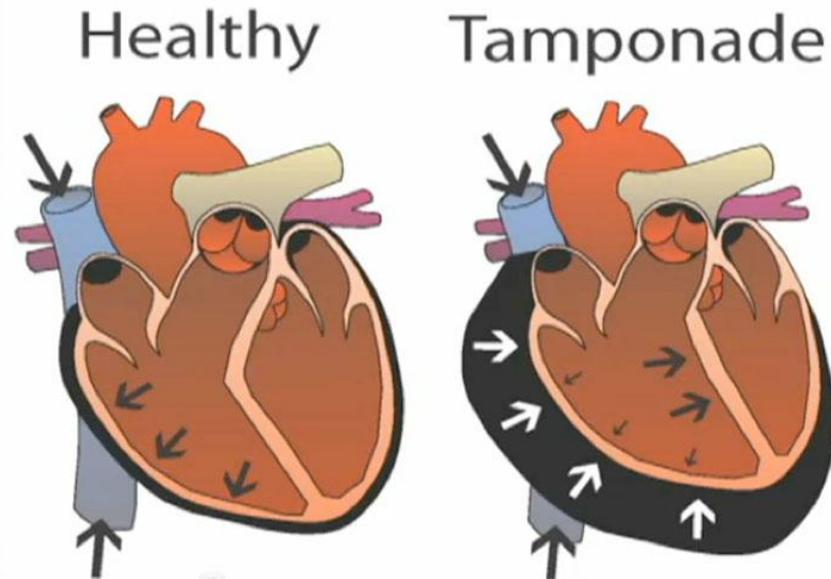
- ***Hemopericardium:*** *The term hemopericardium is limited to accumulations of pure blood in the pericardial cavity, and should not apply to mixtures of blood and serous fluid.*

Hemopericardium

- **Traumas** and **costal fractures** on the heart and pericardium.
- **Rupture** of aneurysms developed in the coronary arteries,
- **Haemorrhages** related tumors that form around the heart,
- **Blood clotting disorders** (vit K inadequacy or absence, Dikumarol poisoning)

Hemopericardium

- If the blood collected in the heart sac is too large or suddenly filled, the heart remains **under pressure** and **stops** within a short time.
- This event is called **“Heart Tamponade”**.



Hemopericardium

- A small amount of bleedings that do not affect the cardiac motions is eliminated by *lymphogen resorption* or a *resorptive inflammation*.
- In this case, adhesions due to common scarring occur.

Serous atrophy of pericardial fat

- In cachexia of any cause
- There is progressive mobilization of depot fat
- The lipid vacuoles are reduced in size, they are replaced by proteinaceous fluid
- Increase in interstitial fluid
- The depots are converted to gray, gelatinous masses that may be flecked by small white foci of fat necrosis.

Pericarditis

- The term pericarditis refers to the **inflammation** of the mesothelium layer covering the inner face **of the heart sac**.
- Pericarditis occurs during the process of many diseases such as, **pleuritis** and **peritonitis**.
- In other words, pericarditis has also infectious character.
- Except traumatic pericarditis, the disease occur in pericard **hematogenously**.
- The majority of pericarditis is exudative.

Pericarditis

- Pericarditis is divided into four groups according to the mechanism (pathogenesis);
 - A. Traumatic pericarditis,**
 - B. Infectious or secondary pericarditis,**
 - C. Sterile pericarditis,**
 - D. Special inflammation of the heart sac.**

Traumatic pericarditis

(Pericarditis traumatica)

- This type of trauma is usually a disease specific to **cattle**.
- In cattle, pericarditis is usually caused by long, **thin sharp foreign bodies** (wire, needles, nails) that **penetrate the reticulum, diaphragm and pericardial sac** resulting in traumatic pericarditis.
- These cases are rarely found in sheep and goats and other animal species.

Pericarditis traumatica

- If the foreign body gets **stuck directly into the heart**, death occurs soon.
- In this case, one of the coronary vessels or the ventricular wall of the heart is punctured.
- Regarding the developing bleeding, the heart sac is slowly filled with blood. So, the animal dies as a result of **heart tamponade**.
- If pericarditis traumatica **persists for several days**, the animal dies with severe edema or infection in 2-3 days. In this case, death is the result of asphyxia or toxemia.
- **In cases of chronic pericarditis traumatica, the animal was saved from death.** However, it is weak in terms of condition. It can never recover and its efficiency is very low. Therefore, it is referred to the **slaughter in a short time**.

Infectious (Secondary) Pericarditis

- This type of pericarditis occurs during the course of many infections in the body. The agents come in a **hematogenous** way.
- It also occurs as a retrograde lymphogen from the lymph nodes
- Such pericarditis are classified according to the **nature of the exudate**;
 1. **Serous and sero-fibrinous** pericarditis,
 2. **Fibrinous** pericarditis
 3. **Purulent** pericarditis
 4. **Gangrenous** pericarditis
 5. **Haemorrhagic** pericarditis

Serous and Sero-Fibrinous pericarditis (Pericarditis serosa et serofibrinosa)

- An acute inflammation.
- The exudate in the heart sac is **serous**. However, *sometimes fibrin* is present in the sac.
- It occurs in more slow infections and occurs in all animal species.
- **Pleuritis, peritonitis and arthritis** occur with pericarditis.
- For example; Septicemia of newborns (E. coli and Salmonella inf.), Pasteurellosis, Bovine and Horse plague, Horse Influenza, Swine swollen, Swine fever, Infantile disease, Swine disease, Dog youth disease and Leptospirosis, Also common cold and allergic factors .

Serous and Sero-Fibrinous pericarditis (Pericarditis serosa et serofibrinosa)

- Pericarditis begins with severe **hyperemia** of the vessels.
- A large amount of **exudate** exudates from the expanding vessels into the sac. This exudate is rich in **leukocyte** and **fibrin**.
- Inner side of the heart sac is **swollen** and the cells are poured into the exudate in the sac.
- **The exudate accumulated** in the sac begins to coagulate in the form of fibrin nets and pellets in a short time. In other words, ***pseudomembrane*** formation is observed.
- Most of the pericarditis remains at this stage. And it heals with the organization of pseudomembrane.

Fibrinous Pericarditis (Pericarditis fibrinosa)

- It is a subacute and chronic inflammation of the heart sac **with fibrin**. It is a type of inflammation that occurs frequently in animals and humans.

Fibrinous Pericarditis (Pericarditis fibrinosa)

- Fibrinous pericarditis is usually the result of **hematogenous microbial infections**, but it may arise by lymphatic permeation from an inflammatory process in adjacent tissue.
- In *cattle*, fibrinous pericarditis is commonly part of pasteurellosis, blackleg, contagious bovine pleuropneumonia, clostridial hemoglobinuria, and some of the neonatal coliform infections that enter via the umbilicus. Fibrinous pericarditis in adult *sheep* is usually part of pasteurellosis; in lambs, it is usually part of pasteurellosis or caused by streptococci.

Fibrinous Pericarditis (Pericarditis fibrinosa)

- The exudation of fibrin usually begins about the base of the heart and extends from there to cover both the pericardium and epicardium.
- The fibrin is **gray-white**, but it may be flecked with **blood**, or **yellow** if a large number of leukocytes are added to the exudate.
- When the leaves are drawn apart, the exudate is drawn out into **villus like projections** to give an appearance responsible for the names “**cor villosum**,” “shaggy heart,” and “bread-and-butter pericarditis”

Fibrinous Pericarditis (Pericarditis fibrinosa)

- Fibrinous exudate can be removed
- Mesothelium can be regenerated.
- *Restorative processes compete with the processes of organization.*
- *Within a week or so, there will be well-formed fibrous tissue in the deepest parts.*
- **If the course is prolonged**, organization and **scarring** will result in focal or diffuse fibrous **adhesions** between the pericardial surfaces, with partial or complete obliteration of the sac.

Purulent Pericarditis

(Pericarditis purulenta)

- Purulent pericarditis almost invariably denotes the presence of **pyogenic bacteria**, either as primary pathogens or as opportunists in *fibrinous pericarditis*.
- It occurs almost solely in cattle as a result of *traumatic perforation by a foreign body originating in the reticulum*, but it is observed in cats and horses in association with empyema (pyothorax).

Pericarditis purulenta

- **The suppurative pericardial fluid** may appear as thin, cloudy exudate; as frank, creamy pus; or as a mixture of pus and masses of fibrin. The color depends on the organisms present, but usually varies from yellow to green, being irregularly dirty gray when putrefactive bacteria are present.

Gangrenous pericarditis (Pericarditis gangrenosa)

- This type of inflammation of the heart sac is caused by *traumatic pericarditis* as it becomes *purulent and putrefactive*.
- On the inside of the sac, there is a thick colored fibrin layer. And it also contains **bad-smelling gas bubbles inside**. The exudate in the heart sac is green-yellow, dark red or brownish-brown and has a bad odor.

Haemorrhagic pericarditis (Pericarditis haemorrhagica)

- This kind of inflammation is recognized by the mixing of **blood in the exudate** with fibrin in the sac. Occasionally, the inflammation may start bleeding directly.
 - Anthrax and Pasteurellosis in cattle;
 - Bradzot disease, Infectious Necrotic Hepatitis in sheep;
 - Infectious purpura in dogs ;
 - generally, seen in pericardial tuberculosis and malignant tumor.

Sterile Pericarditis

- ✓ Infectious/granulomatous inflammation of pleura causes **sterile serous effusion** in pericard because of **irritation**. This type of **inflammation of the pleura** can gradually spread to **the pericardial sac**.
- ✓ Steril pericarditis occurs in **tumor implants** in **gout disease of poultry** and in the resorption of the fluid collected in the pericardial sac.

MYOCARDIAL DISEASE

- **Dystrophic ve Degenerative Myocardiopathie**
 - Disorders and degenerative changes in the heart muscle that are not related to inflammation and developmental disorders.
- A. Dystrophic myocardiopathie
 - B. Degenerative myocardiopathie

A. Dystrophic myocardiopathie

- **Atrophia cordis**
- **Hypertrophia cordis**
- **Dilatatio cordis**
- **Brisket disaese**
- **Round heart disease**
- Cardiac aneurysm
- Pigmentation

Hypertrophia cordis

- **a. Concentric hypertrophy in the heart**

(The heart muscle thickens at the same rate on all sides.)

- **b. Eccentric hypertrophy in the heart**

(It is the enlargement of the ventricles with expansion and is mostly observed in the right ventricle.)

- **c. Pseudo-hypertrophy in the heart**

(It involves the collection of excess fat in the interstitium of the heart muscle.)

Hypertrophica cordis

- Causes of hypertrophy in heart muscle can be grouped into two groups;

I. Causes of physiological hypertrophy,

a. Overwork

(running horses, hunting dogs, etc.)

b. Overload towing

(forcing animals to be transported with heavy loads)

c. Severe pregnancy states

(twin in the uterus, triplets, and more normal than normal puppies)

Hypertrophia cordis

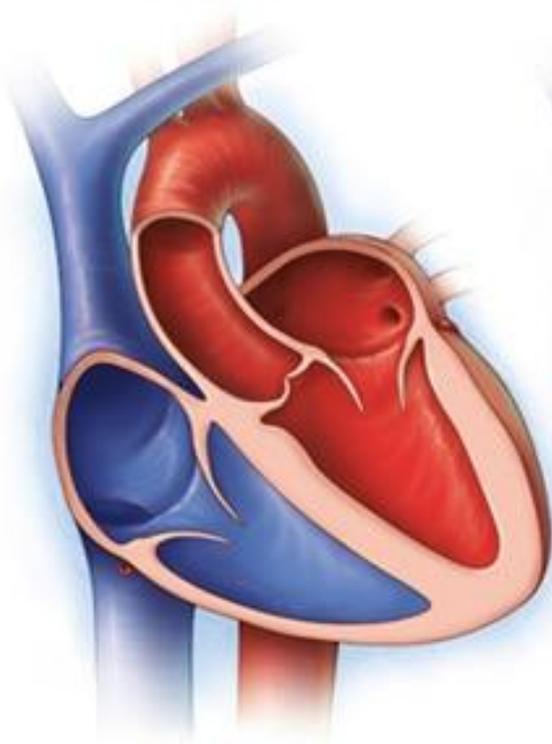
II. Causes of pathological hypertrophy

- **The thickening** of the semilunar **valvule** of the aorta or narrowing of the **ostiums**, **prevention of blood flow** in the arteries, common **chronic interstitial nephritis** in the kidneys
- **Stenosis** of arteria pulmonalis, **the thickening** of the semilunar valvule of the arteria pulmonalis, insufficiency or stenosis in mitral valves, **parasitic infestations** (*D.immitis*) in dogs, chronic emphysema or chronic interstitial pneumonia in lungs, large amounts of exudate or transudate in chest cavity, pleuritis chronica adhesive
- **Disorders involving all the heart valves**, increased blood viscosity, chronic anemia (especially in humans)

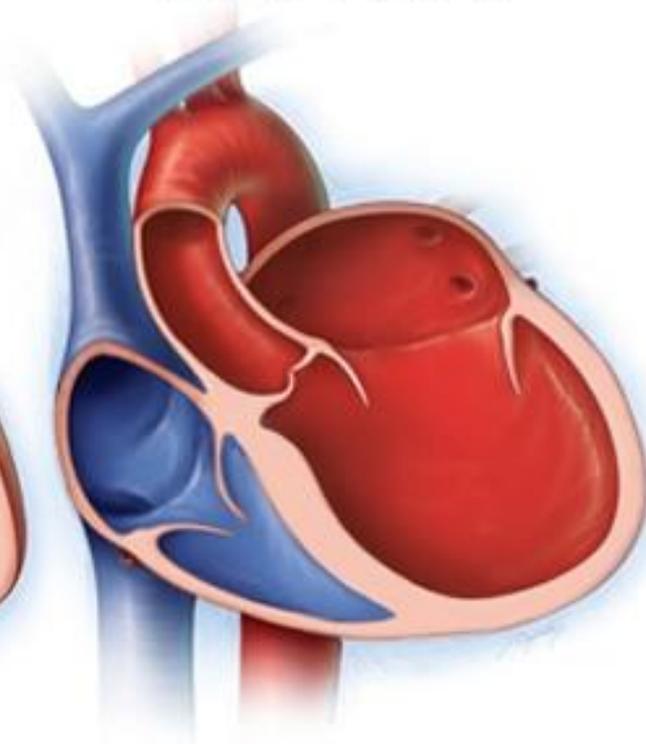
Dilatatio cordis

- Pathological **expansion** of one or both **ventricles**.
- Dilatation is seen usually in **right ventricle**.
- Because, in the left ventricle there is a resistance to enlargement and the wall is already thick.
- Dilatation in the heart can occur **acute and chronic**.
- In acute form it develops within a few hours or several days.
- In chronic form it occurs within months. There is also thickening of the ventricle wall in chronic dilatation. **In other words, there is always a hypertrophy in the heart in chronic heart dilatation.**

Normal heart



Dilated cardiomyopathy



Brisket Disease

“High-altitude disease” of cattle

- *This disease is caused by pulmonary hypertension that results in **dilation and hypertrophy of the right ventricle** with the ultimate development of cardiac decompensation and right-sided congestive cardiac failure.*
- Edematous swelling of the venter, as is typical of congestive heart failure in cattle, is responsible for the synonym *“brisket disease.”*
- It is also known as '**Mountain Disease**' since it is seen in mountainous areas up to 3000-4000 meters. It is also known as '**High Altitude Disease**' since it can be seen in high plateaus up to 2500 meters.

Brisket Disease

- There are species differences in the hypertensive response to hypoxia
- Sheep and dogs are hyporesponders, humans are intermediate, **whereas cattle and pigs are hyperresponders.**
- **Young cattle are more susceptible** than adults, and the morbidity rate is highest in animals exposed to high altitudes for the first time.

Brisket Disease

- Cause of **atmospheric density** and **pressure** are **low** at high altitude.
- **Hypoxia and anoxia** formation cause the disease to develop.
- **Dystrophy, anemia, pneumonia** and other lung diseases are among the **preparatory reasons**.
- It is also reported that hypoproteinemia related to the lack of pastures in mountainous regions and some poisonous plants that grow in such regions are among the preparatory reasons.

Brisket Disease

Macroscopically;

- **The hairs** covering the body are **dull and rough**.
- There is excessive **swelling in V. jugularis**.
- There is a prominent edema especially in the chest area, under the skin(neck and abdomen).
- Mucous membranes are **cyanotic**.
- **The heart was enlarged and the right ventricular wall was thickened.**
- There is severe passive hyperemia in liver and spleen.
- Transudate accumulation may be observed in pericardium, pleura and peritoneum.

Brisket Disease

Microscopically;

- **Intramuscular edema** in the heart,
- **Muscular fibers degenerative** appearance, intracellular edema,
- Edema in the lung,

❖ Polycythemia in clinical blood examination

B. Degenerative myocardiopathies

- Parenchymal degeneration in cardiac muscle,
- Fat degeneration in the cardiac muscle,
- Hyaline degeneration in cardiac muscle.

Special Myocardiopathies

- White Muscle Disease (Enzootic Muscular Dystrophie)
- Foot and Mouth Disease,
- Sudden Heart Failure
(Sudden Cardiac Arrest, Sudden Cardiac Death)
- Mulberry Heart Disease,
- Transport Necrosis,
- Myocardiopathie related to alcoholism in humans

White Muscle Disease

- Nutritional myopathies are *principally diseases of calves, lambs, swine, and foals.*
- *It is a* nutritional myopathy caused by a selenium or (less commonly) vitamin E deficiency.
- Young animals aged 1 to 8 weeks are the most sensitive to the disease.

White Muscle Disease

- In many cells, *vitamin E- and selenium-containing enzymes are required* as physiologic antagonists to a group of chemically varied substances known as *free radicals*.
- *Free radicals may initiate cellular injury by causing peroxidation of membrane lipids.*
- So, In the **absence of** sufficient protection by **selenium and/or vitamin E**, cellular membranes are damaged by lipid peroxide.

White Muscle Disease

- Macroscopic lesions are quite characteristic.
- Gross changes **included chalky-white appearance** of entire endocardium of **right ventricle** and subendocardial plaques in the **interventricular septum** and **left ventricular Wall** and **Papillary muscle**.
- Degenerations in skeletal muscles are mostly in the back, rump, front and hind leg muscles and locate symmetrically.

White Muscle Disease

- The lesions in the **diaphragm** are in lines and stripes.
- The affected muscle groups are **edematous, swollen and crispy**. **Calcification** is also seen.

White Muscle Disease

- Microscopically;
- Microscopic changes include loss of striations followed by swelling, hypereosinophilia, glassy or hyaline appearance (**Zenker's degeneration**).
- The calcification of degenerated muscle can be seen.
- Macrophage and lymphocyte infiltrations are seen in degeneration regions.

White Muscle Disease

- In some areas, there are **multinucleated myogen giant cells** that are related to regeneration.
- It is also noteworthy that the areas where muscle tissue is **necrosed** are filled by young **connective tissue cells**.

Foot and Mouth Disease

- Especially, lesions occurring in young cattle begin with hyaline degeneration in the form of stains or lines in heart muscle fibers and rapidly progress towards necrosis.

Foot and Mouth Disease

- Most of the time there is healing with scars, and these scars (“tiger stripes” or “tiger heart”) can be found after slaughter.
- Histological examination of the acute and subacute resorption periods, muscle degenerations and lymphohistiocytes and plasma cell infiltrations are coexisting.

Mulberry Heart Disease of swine (Diatetic Microangiopathie)

- The name “mulberry heart” is vaguely suggested by the *extensive hemorrhages on the surface of the heart.*
- The disease occurs in pigs only, chiefly those 2-4 months of age and in excellent condition, but it has been observed in animals from 3 weeks to 4 years of age.
- Although the reason is not completely clear; *vit. E and selenium deficiency are emphasized.*

Mulberry Heart Disease

- Large amounts of fluid around the heart and lungs.
- **Haemorrhagic** and **pale areas** in heart muscle.
- Fluid in the abdomen with pieces of fibrin.
- Pale muscle areas (necrosis) particularly in the lumber muscles and hind muscles of the leg which contain excesses amounts of fluid.

Mulberry Heart Disease

- *Hemorrhages, linear and ecchymotic, are present beneath the **epicardium**. They may be few, or they may be extensive and involve the epicardium of all chambers, the **myocardium**, and beneath the **endocardium** of the papillary muscles and septum.*

Myocardial hemorrhage with necrosis and mineralization of myocytes in mulberry heart disease.

MYOCARDITIS

- *Myocarditis, or inflammation of the myocardium, is a common lesion found in a wide variety of systemic diseases.*
- It is rarely primary
- It occurs hematogenously in many infectious diseases, and also by direct extension from inflammatory lesions of the endocardium and pericardium.

MYOCARDITIS

- The inflammation in the heart muscle, primarily related to the hematogenous origin of the disease agents, starts and settles in the interstitial area.
- The heart muscle is subsequently and secondary affected.
- In the meantime, some intoxications and nutrition disorders, first heart muscle is affected and dystrophic disorders occur.

MYOCARDITIS

- A. Myocarditis nonpurulenta (Lymphocytaria - Paranchymatosa)
- B. Myocarditis purulenta (Apostomatosa)
- C. Myocarditis necroticans
- D. Myocarditis allergica
- E. Special myocarditis

MYOCARDITIS

- **A. Nonpurulent myocarditis** occurs generalized and acute infectious and toxic diseases.
- Therefore, it is often not possible to conclude serous myocarditis to specific causes.

MYOCARDITIS

- However, the diseases can be listed as follows.
- **In cattle:** Foot and Mouth Disease, C.G.B., Blackleg, Rinderpest, etc.
- **In horses:** Infectious viral anemia, parasitic invasions, etc.
- **In dogs:** Acute form of Distemper, Leptospirosis, H.C.C. etc.
- **In cats:** Agranulocytosis etc.
- **Chickens:** Salmonellosis, Pullorum etc.
- **In pigs:** Aujeszky's disease (pseudorabies), Teschen disease, Swine erysipelas etc.

MYOCARDITIS

- Microscopical lesions** in nonpurulent myocarditis;
- In some cases; hyperemia and interstitial edema (**serous myocarditis**), myofibrillary **degeneration**, separation of myofibrils and small amounts of **mononuclear cell infiltration**.
 - In some cases; Hyaline degeneration in muscles, fibrocyte proliferations in chronic cases and multiple lymphohistiocytic cells infiltrations

MYOCARDITIS

- **B. Purulent myocarditis**
- These factors are either **hematogenous** (metastatic) or **directly located in the heart muscle**.
- *Pyogenic bacteria, which may originate from any other suppurative focus in the body, reach the heart as bacteria embolism.*

MYOCARDITIS

- In cattle; myocarditis may be caused **by a pus** and inflammation in the organs such as lung, uterus, endocardium, intestine, umbilical cord, etc.
- **Directly, traumatic pericarditis usually plays a role as a precursor.**

MYOCARDITIS

In purulent myocarditis, macroscopically,

- **Yellowish gray small puss** foci are occasionally seen in the heart muscle. They have varying diameters ranging from the needle to the lentil size. And this condition is called “Myocarditis purulenta disseminata”.
- **Microscopy** of such case pus focus and abscess formed by **neutrophil leukocytes** are seen.

MYOCARDITIS

- **C. Necrotic myocarditis:**
- It is always secondary.
- Inflammation is caused by "*necrosis bacillus (Spherophorus necrophorus)*".
- Necrobacillosis reach **hematogenously** from mammary, uterus, vagina and liver in cattles; umbilical cord in calves.

MYOCARDITIS

- **Macroscopically**; the lesion in myocardium is *dirty-yellow-brown* in color, the environment is surrounded by an irregular and hyperemic halo. This necrosis tissue that can be crushed between the fingers can sometimes be a perforated view due to gas formation.
- **Microscopically**; There is a demarcation site with *coagulation necrosis and neutrophil leukocyte infiltration* around it.

MYOCARDITIS

D. Myocarditis allergica

- In animals, the presence of lesions similar to those of human heart and related to *rheumatism* has not been established yet.
- However, periarteritis nodosa lesions are seen. Accordingly, it is thought that there may be *allergic inflammations* caused by antigen-antibody reactions in animals.

MYOCARDITIS

- **E. Tuberculosis** is rarely seen in diseases of the special infectious granuloma group located in the heart muscle. It has also been reported in some reports that there may be diseases such as **glanders, actinomycosis, aspergillosis.**

Parasites in Heart muscle

- Parasites are common in heart.
- **Sarcosporidiosis** (cattle, sheep, goats, pigs and infrequently carnivores),
- **Cysticercosis** (C.cellulosa / pig; C.inermus / cattle; C.ovis / sheep),
- **Fasciola hepatica** (sheep - in some cases hematogenous to the heart.),
- **Filaria and Strongylus**,
- **Diroflaria immitis** (In the heart, especially in the right ventricle, lives in the pulmonary arteries and causes obturation.)
- **Echinococcus** cysts (cattle, sheep, goats),
- **Toxoplasmosis** (It usually develops in relation to the generalization of lung, liver and brain lesions in dogs.).

ENDOCARDIUM

Calcification in Endocardium

- Calcification of the elastic fibers of the endocardium. It is mostly seen in cattle, horses and dogs. It is more common in older animals.
- **Metastatic calcification** occurs in cases of **deficiency in calcium metabolism**. This is seen in hypervitaminosis D, uremia and nutrition disorders.
- Calcification, which is related to the causes affecting the endocardium, also develops **dystrophic calcification**.
- Therefore, it is observed in diseases such as **White Muscle Disease**, Leptospirosis, Tuberculosis etc.

ENDOCARDITIS

- **Inflammation** of the inner layer of the heart (**endocardium**).
- It can occur for a variety of reasons.
- It is *usually bacterial in cause, the exceptions being occasional parasitic or mycotic lesions.*
- Dystrophic disorders of the endocardium can also be converted to endocarditis

ENDOCARDITIS

According to the **the location** of the inflammation;

- Endocarditis valvularis
- Endocarditis parietalis
- Endocarditis chordalis

ENDOCARDITIS

According to the **pathological - anatomical features** of the inflammation;

- Endocarditis (Thromboendocarditis) simplex superficialis,
- Endocarditis polyposa et ulcerosa
- Endocarditis verrucosa
- Endocarditis chronica fibrosa
- Endocarditis calcificans

ENDOCARDITIS

- In spite of all these classifications, endocardium inflammations can easily infiltrate to many areas and transform from one form to another.
- For these reasons, endocarditis is usually examined under two headings.
- **a. Endocarditis valvularis**
- **b. Endocarditis parietalis**

Endocarditis valvularis

- Endocardial inflammation is located in the heart valves.
- Endocarditis in animals are mostly located in the **mitral valve**
- Less is seen in the **aortic valves** and the **tricuspidal valve**.
- Endocarditis valvularis events are almost bacterial origin.
- Generally, they are seen in septicemia and pyemia.

Endocarditis valvularis

- **In cattles;** *Corynebacterium pyogenes*, *Streptococcus* species, *Staphylococcus aureus*, *E. coli* serotypes
- **In horses;** *Streptococcus equi*, *Bact. viscosum equi*, umbilical cord infections, *Meningococci*, Infectious Viral Anemia
- **In dogs;** Non-hemolytic *Streptococ* species, *E. coli* serotypes, *Staphylococcus* species, *Pseudomonas aeruginosa*
- **In Sheep and Goats;** *Pseudotuberculosis ovis*, *Streptococcus fecalis*.
- **In pigs;** *Erysipel*, *Corynebacterium pyogenes*
- ✓ Common in all animal species; *Salmonella*, *Pasteurella*, *E. coli* infections, and all pyogenic disease

Endocarditis valvularis

- Valvular endocarditis is classified according to the lesions seen in necropsy;
 - 1. Endocarditis valvularis simplex**
 - 2. Endocarditis valvularis polyposa et ulcerosa**
 - 3. Endocarditis valvularis verrucosa**
 - 4. Endocarditis valvularis fibrosa**

1. Endocarditis valvularis simplex

- Valves are slightly **edematous** and **petechial hemorrhages** are seen.
- **Thrombotic masses** appear on the valvules.

2. Endocarditis valvularis polyposa et ulcerosa:

- It is an advanced form of endocarditis.
- There are red-brownish irregular **polyps** or **villus** formations on the valves.
- In addition, **necrosis** may form. If the necrosis is removed, **ulcers** are formed.

3. Endocarditis valvularis verrucosa

- On the edge of the valvulus, there are **gray-white colored nodes**.
- They may also **have fibrin masses**.
- **Petechial hemorrhages** may be encountered in the valves.
- These hard nodes cannot be removed easily.
- Sometimes these nodes can be calcified.

4. Endocarditis valvularis fibrosa

- Valves are thickened **into hard nodes**, and **thrombotic masses** may be present on them.
- The valves themselves **are thickened by** the formation of **scar**, and such valves can no longer function properly.

Endocarditis valvularis

○ Microscopically;

1. Endocarditis valvularis simplex:

- Valvular vessels are hyperemic
- Edema in the subendocardial connective tissue.
- Dissociation of endothelial cells
- Fibrin fibers and neutrophil leukocytes are common

Endocarditis valvularis

○ Microscopically;

2. Endocarditis valvularis polyposa et ulcerosa:

- There are large and/or small thrombosis on the valve.
- In these thromboses, bacterial clusters and necrosis are observed.
- There are neutrophil leukocytes around the necrosis.
- Calcification can be seen.
- This area is surrounded by granulation tissue.

Endocarditis valvularis

○ Microscopically;

3. Endocarditis valvularis verrucosa:

- Thrombosis of the endocardium begins to be organized by the granulation tissue that develops from the subendocardial connective tissue. Later, it becomes a scar tissue.

Endocarditis valvularis

○ Microscopically;

4. Endocarditis valvularis fibrosa:

- It is seen that the nodes on the endocard become scatrix.
- In the same way, the valve can be transformed into thick scar tissue.

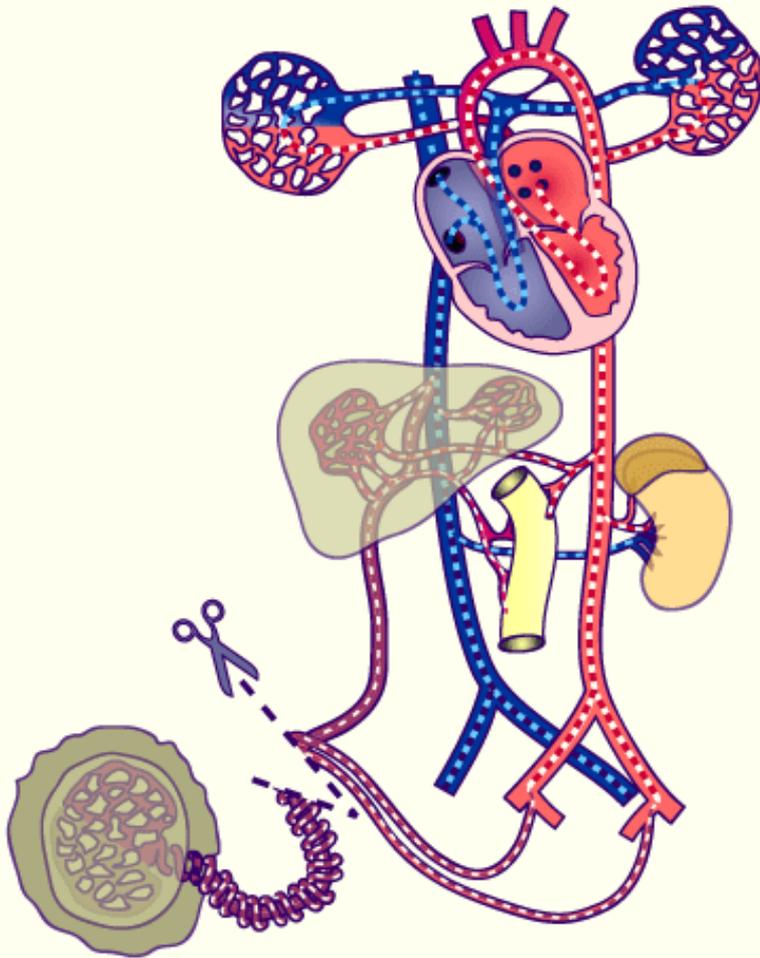
Endocarditis parietalis

- It is a state of involvement of endocardium inflammations in the parietal section and is much less observed than valvular endocarditis.
- In most of the cases, the lesions are seen in the right and left ventricle walls.
- Parietal endocardial inflammation occurs mostly in cattle.
- The most important causes are the occurrence of septicemia related to Erisipel in pigs and the uremia events related to leptospirosis in dogs.
- In addition, pyemia related to pyogenic infections often cause an endocarditis.

○ Special Inflammations of the Endocardium:

- Lesions related to special infectious granulomatous diseases are rare.
- However, it is reported that endocardial tuberculosis can be found in cattle, pigs and dogs.

after birth



DISEASES OF THE VASCULAR SYSTEM

- A. Arterial Diseases
- B. Venous Diseases

Congenital anomalies

- **Arteriovenous fistula**, the communication of an artery and vein that bypasses the capillary system, is a defect that may arise developmentally or be acquired subsequent to physical trauma, inflammatory necrosis involving adjacent vessels, neoplastic infiltration, or rupture of an arterial aneurysm into a vein.
- Fistulas occur in dogs, cats, horses, and cattle, but are uncommon.
- In general, arteriovenous fistulas cause decreased peripheral resistance and increased venous return to the right heart.

Congenital anomalies

- **Thebesian veins** connect the cardiac chambers, between the trabeculae carneae, with the myocardial sinusoids and capillaries.
- They may also connect with coronary arterioles to form direct connections between the coronary arterioles and the cardiac chambers.
- Areas of myocardium in which the Thebesian vein system is richly persistent are randomly distributed, and the true nature of the defect may be overlooked because of the tortuous aneurysmal dilation of the affected coronary artery.
- Injection studies are necessary to demonstrate the extent of the anastomosis.

Metabolic arteriopathies

- These are arterial diseases related to disorders of arteries that are not related to inflammation.

Metabolic arteriopathies

- Metabolic arteriopathies are divided into two main groups;
- a. Dystrophic arteriopathies,
- b. Degenerative arteriopathies

Dystrophic arteriopathies

- Dystrophic arterial diseases are relating to or caused by faulty nutrition of arterial wall.

Dystrophic arteriopathies

❑ Changes related to **aging** in arteries:

Some changes occur in the walls of the artery as a result of events that are related to the progression of age

- **Fibrosis** in the aorta and A. pulmonalis
- **Thickening** of the artery wall
- **Fat accumulation** in the artery wall
- **Folding** of tunica intima in artery
- **Pregnancy sclerosis** in uterine vessels

Dystrophic arteriopathies

□ Intima Sclerosis in Arteries :

- Morphologically, it is divided into sub-sections as **focal and diffuse - central and peripheral**.
- It can be divided into subgroups about pathogenesis as **reparative - degenerative - inflammatory and compensative** -.
- Intima sclerosis usually occurs in the heart, kidney and thyroid arteries.
- Among animal species, it is most common in dogs with chronic kidney disease.

Dystrophic arteriopathies

☐ Intima Sclerosis in Arteries :

The reasons for this can be listed as follows;

❖ Degenerative and inflammatory arteriopathies

❖ Traumas in the arter wall

- **Damage** to the intima layer - rupture - injury - lesions caused by parasites and thrombosis are **tried to be repaired by** proliferation of a **wide range of fibrils**. Hence, the tissues of these lesions occur in the **scar**. Thus, **regional intima thickening occurs**.
- In the case of **fibrinoid degeneration** in the arteries, the **degenerated parts are resorbed**, and then they recover in the remaining damaged areas.
- Thus, **intima sclerosis is formed**.

- Intima sclerosis is **permanent**.
- The lumens of such arteries **narrow**.
- Therefore, **nutritional disorders** are **seen in the organs** fed by arteries in which sclerosis occurs.
- Sometimes **calcifications** may occur in such lesions.

Dystrophic arteriopathies

❑ Medial Hyperplasia in the Artery Wall:

- An artery thickening is associated with an increase in muscle fibers in the media layer.
- It is mostly seen *in humans.*
- In animals, *the cats, rabbits and guinea pigs* are found only in the pulmonary arteries.

Dystrophic arteriopathies

□ Medial hypertrophy of the pulmonary arteries of cats:

- Displays no age, sex, or breed predilection
- Appears to be normal anatomic variation in cats
- Similar pulmonary arterial lesions have been described in cats infected with the **parasites** *Aelurostrongylus abstrusus* and *Dirofilaria immitis*.

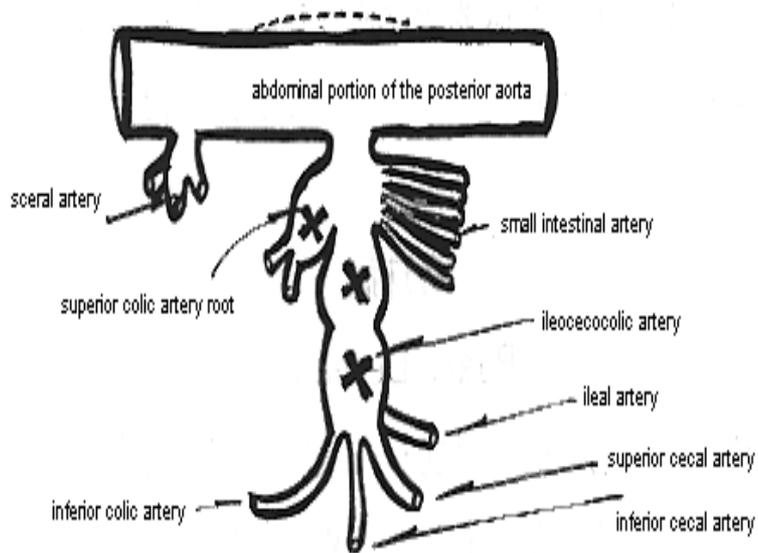
- Pulmonary hypertension does not result from the vascular change, right ventricular pressure does not increase, nor does the right ventricle hypertrophy.
- ✓ **The most severely affected vessels** may be grossly visible **on the cut surface of the lung** and through the pleura when the lungs are collapsed, and are even palpable in some cases.
- ✓ The histologic spectrum of arterial changes ranges from mild *sporadic or generalized hypertrophy* of the **tunica media.**

Dystrophic arteriopathies

□ Arterial aneurysm:

- It is a limited and transverse **extension** of the artery wall.
- It is not a direct disease but a lesion that occurs as a result of many vascular diseases.
- It occurs more often **in horses** and often occurs in relation to **parasites**.
- Most of these events in horses occur in **Arteria ileocaecalis** and the less in branches of Arteria mesenterica cranialis.
- **Strongylus vulgaris** larvae form **verminous aneurysm** in horses.

Arterial aneurysm



frequent sites of parasitic
aneurysms in horse

Dystrophic arteriopathies

❑ **Calcification in the artery wall (intima and media):**

- **Calcification (mineralization)** occurs quite frequently in the arteries of animals, either as a dystrophic or metastatic process.

- **Dystrophic mineralization**, or *mineralization of dying or dead tissue*, occurs in areas of inflammation, degeneration, and thrombosis, and not necessarily in association with pre-existing arteriosclerosis.
- **Metastatic mineralization** occurs as the *result of hypercalcemia and/or hyperphosphatemia*.

- **Causes of dystrophic calcification,**
 - Dystrophic, degenerative and inflammatory arteriopathies,
 - Verminous arteriopathies,
 - Tromboendoarteritis
 - Endoarteritis due to leptospirosis in the lung arteries,
 - Infarction and media sclerosis,
 - Lesions associated with diseases like Tuberculosis e.g.
- **Reasons for metastatic calcification,**
 - Hipovitaminosis D,
 - Common chronic interstitial nephritis of dogs,
 - Renal osteopathies,
 - Parathyroid hyperfunction and tumors.

□ Arterial thrombosis and embolism:

The 3 major predispositions to thrombosis are

- 1. *Injury to endothelium***, for instance, via infectious, toxic, or immunologic mechanisms
- 2. *Altered blood flow***, as occurs with stasis or turbulence
- 3. *Hypercoagulability of the blood***, which may result from increased concentrations of activated procoagulants, increased numbers or stickiness of platelets, or decreased concentrations of inhibitors such as antithrombin in the nephrotic syndrome.

- **Thrombi** are of importance because they **occlude** the vessel.
- Arterial occlusion is of significance in organs with an **end-arterial blood supply**, such as the kidney, because of the absence of collateral circulation and the development of infarction.
- Simultaneous occlusion of a large number of pulmonary arterioles or arteries by thrombi can lead to **right heart failure (cor pulmonale)** and **death**.

□ Disseminated intravascular coagulation (DIC):

- *Disseminated intravascular coagulation (DIC) is a common and important intermediary mechanism of disease, but is not a disease in itself.*

DIC

- It may be defined as a pathologic activation of the **coagulation system** that leads to **generalized intravascular clotting** involving, in particular, arterioles and capillaries.
- The process may be *acute, subacute, or chronic*, and may be *localized or generalized*.
- The terms *“consumption coagulopathy,” “defibrination syndrome,”* and *“consumptive thrombohemorrhagic disorder”* are also used because of the massive consumption of coagulation factors that occurs and that may be sufficiently severe for hemorrhagic diathesis to result.

DIC

- *A wide array of agents and conditions will initiate coagulation either by*
 - ❖ Causing *widespread **endothelial damage*** and thus exposing thrombogenic subendothelial collagen, or
 - ❖ Directly ***activating the coagulation cascade*** via the intrinsic or extrinsic pathway.

DIC

- Exposure of monocytes, macrophages, and endothelial cells to disease agents or mediators will cause ***expression of tissue factor (tissue thromboplastin)*** on the cell surfaces and activation of the extrinsic pathway of coagulation, and is likely a predominant pathway of DIC.

Agents or conditions known to induce disseminated intravascular coagulation (DIC) in animals:

❖ Bacteria

- Gram-negative (endotoxin)
- Gram-positive
- *Rickettsia rickettsii* (Rocky Mountain spotted fever)

❖ Helminths

- *Angiostrongylus vasorum*
- *Dirofilaria immitis*

❖ Protozoa

- *Babesia* spp.
- *Sarcocystis* spp.
- *Theileria* spp.
- *Trypanosoma* spp.

❖ Viruses

- African swine fever virus
- Aleutian mink disease virus
- Bluetongue virus
- Canine adenovirus 1 (infectious canine hepatitis)
- Classical swine fever virus
- Epizootic hemorrhagic disease virus of deer
- Feline infectious peritonitis virus

❖ Neoplasia

- Carcinoma
- Hemangiosarcoma
- Leukemia

❖ Other

- Aflatoxicosis
- Antigen-antibody complexes— incompatible blood
- transfusion
- Gastric dilation-volvulus
- Heat stroke
- Hyperlipemia in ponies
- Hyperosmolality
- Immunologic endothelial injury
- Ingestion of red maple leaves
- Nephrotic syndrome
- Proteolytic enzymes—pancreatitis, snakebite
- Shock, vascular stasis, prolonged anesthesia, acidosis
- Tissue necrosis—hepatic, pneumonia, postsurgery, burns

Degenerative arteriopathies

- The degenerative events forming in tissues of the artery wall can be listed as follows:
 - ❖ **Hyaline degeneration** of arterial wall (Hyalinose),
 - ❖ **Amyloid degeneration** of the artery Wall (Amyloidose),
 - ❖ **Fat degeneration** of the artery wall (Lipoidose).

ARTERIOSCLEROSIS

Arteriosclerosis literally means “**hardening of the arteries,**” and is more fully defined as: *chronic arterial change consisting of hardening*

- *loss of elasticity*
- *luminal narrowing* (resulting usually from proliferative and degenerative, rather than inflammatory)
- *changes of the media and intima*

ARTERIOSCLEROSIS

- Arteriosclerosis includes three main types of lesions.
 - a) Atherosclerosis,
 - b) Monckeberg's medial sclerosis,
 - c) Arteriolosclerosis

a) Atherosclerosis

- It is characterized by the formation of
 - ✓ yellowish atheromatous plaques
 - ✓ on the *inside of intima and medial layers*
 - ✓ in large and medium size arteries and
 - ✓ excessive fibrosis of these layers.

a) Atherosclerosis

- The term **atherosclerosis** is applied to lesions of arteriosclerosis in which *degenerative fatty changes* also occur.
- **Atherosclerosis** is the most common and important type of arteriosclerosis in humans, and the terms can thus be used interchangeably with little loss of meaning when discussing this species.
- In domestic animals, **arteriosclerosis is common, but of little clinical importance, and atherosclerosis is rare.**

a) Atherosclerosis

- The structure of atheromas consists of **cholesterol**, **lipoid substances**, **destroyed cell** and **tissue residues** and **lipophages**.
- Atheromas are softy masses.
- These masses surround the fibrous tissue that develops from the intima and form focal protrusions above the intima.
- Therefore, these masses are called 'atheromatous plaque' "or fibrolipoid plaque'.

a) Atherosclerosis

- **Atherosclerosis** is the most common form of arteriosclerosis **in humans**.
- Atheromatous plaques occur not only in the aorta and its branches, but also in the coronary and cerebral arteries.
- **Parrots and home pigeons** are the most common species of atherosclerose.
- In mammalian species, only **pigs** have genuine atherosclerose.
- Meanwhile, atherosclerose events have been reported in **cattle, horses and dogs**.
- It has been reported that atherosclerosis of endocardial, aorta and renal arteries in Guernsey breed with **paratuberculosis** is similar to atherosclerosis in humans.

b) Monckeberg's medial sclerosis

Monckeberg's arteriosclerosis (medial calcific sclerosis),

- This form of sclerosis occurs in muscular medium-diameter arteries.
- It is characterized by excessive thickening/ hardening and calcification of the **medial layer of arteries.**
- The disease is also called 'Monckeberg degeneration'. It is mostly seen in older people.
- In animals, this form of disease is quite common. This type of calcification is often seen in the thoracic arteries of butchery cattle.

c) Arteriolosclerosis

- In peripheral small-diameter arteries (arterioles) is recognized by the thickening of the **intimal layer**.
- The disease is divided into two subgroups as 'hyaline arteriolosclerosis' and 'hyperplastic arteriolosclerosis'.

ARTERIOSCLEROSIS

REASONS:

- Factors considered as the cause of all forms of arteriosclerosis;
- **Senility**
- **Hypertension** (in humans)
- **Genetic factors** affecting the structure of the vessel wall
- Increased endothelial permeability and injury of endothelial layers of the arteries in infectious - toxic diseases.

ARTERIOSCLEROSIS

REASONS:

a) Atherosclerosis;

- General disorders of lipid metabolism,
- Excessive amounts of fatty foods through the digestive system, hence increasing **plasma cholesterol levels**,
- All factors leading to **hyperlipidemia**
- **Hyperthyroidism**
- Animals, especially poultry, due to **not moving freely**
- Excessive **lubrication**,
- **Paratuberculosis** events in Guernsey breed cattle.

ARTERIOSCLEROSIS

REASONS:

b) Monckeberg's arteriosclerosis ,

- Factors that are particularly vasotonic in this type of arteriosclerosis are considered to be responsible.
 - For example, excessive stimulation of smooth muscle tissue in the media layer of arteries through vasomotor.
 - Monckeberg's arteriosclerosis was created experimentally in dogs and rabbits by epinephrine (adrenaline) and nicotine injection.
- Hypervitaminosis D

ARTERIOSCLEROSIS

REASONS:

c) Arteriolosclerosis

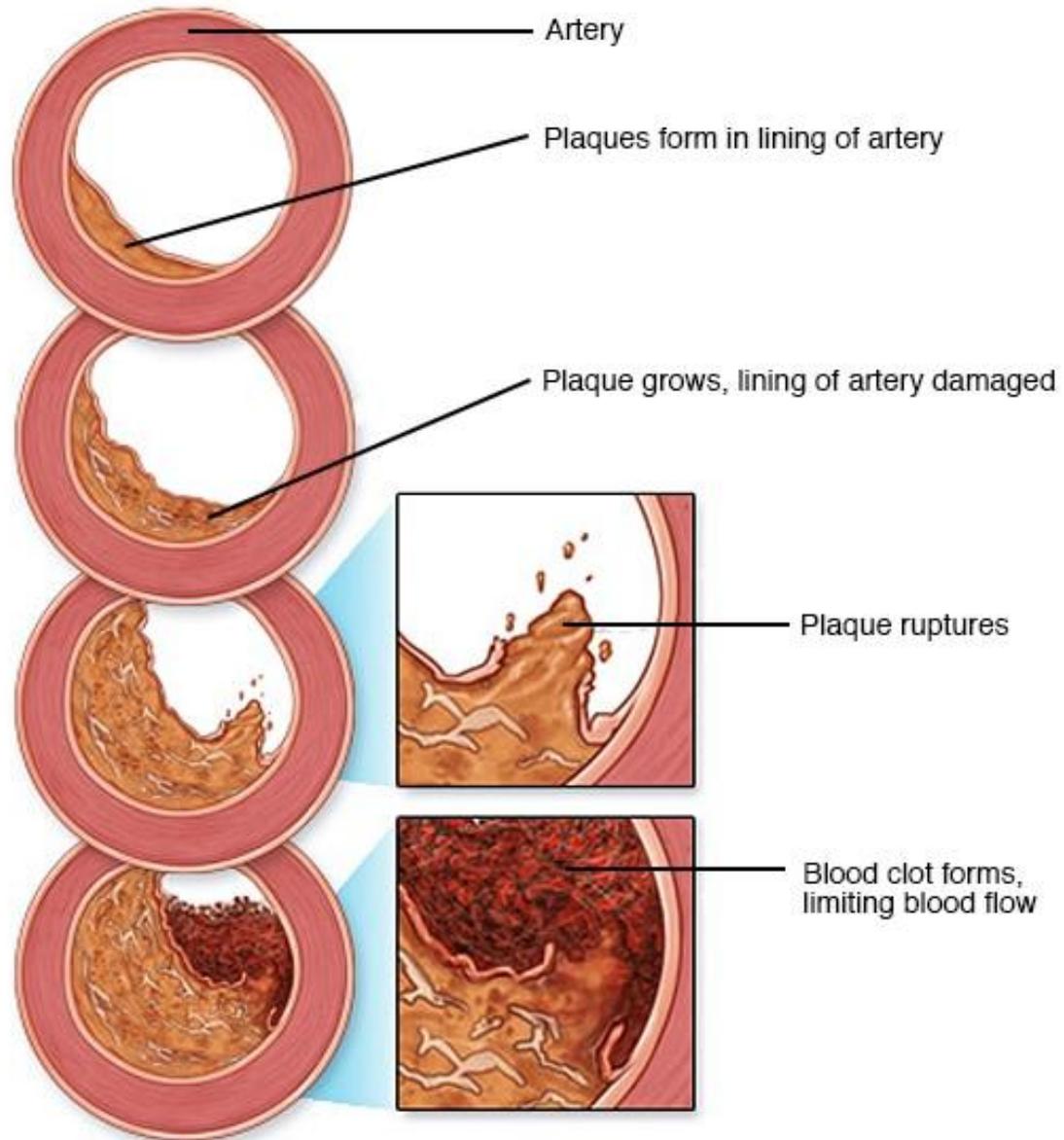
- Hypertension,
- Nicotine (1-2 pack of cigarettes per person for a long time),
- Damage of the endothelial layer by endotoxins,
- Damage of the endothelial layer by various drugs,
- Diabetes mellitus (in humans).

ARTERIOSCLEROSIS

MACROSCOPIC FINDINGS:

a) Atherosclerosis,

- The vein wall is thickened and hardened especially in the big arteries. Typically **sclerotic atheromatous plaques** are seen on the inner face (intima layer) of these arteries.
- The new ones are in *yellowish spots and blisters*.
- In advanced cases, these plaques are enlarged and thickened.
- **In paratuberculosis of the cattle**, it has been reported that *petechial hemorrhages, necroses* and *ulcers* are also found in the aorta thoracica with arteriosclerosis.



ARTERIOSCLEROSIS

MACROSCOPIC FINDINGS:

b) Monckeberg's arteriosclerosis ,

- Since the calcification occurs in the form of rings in the vessel wall or less as plaques, the affected vessel becomes a stiff pipe.

MACROSCOPIC FINDINGS:

c) Arteriolosclerosis,

- In hyaline arteriolosclerosis, altered arterioles are seen as gray-white transparent foci (eg, nephrosclerosis).
- In hyperplastic arteriolosclerosis, the affected arterioles are in appearance resembling the onion (the walls of such vessels are thickened concentrically).

ARTERIOSCLEROSIS

MICROSCOPICAL FINDINGS:

a) Atherosclerosis,

- At the beginning, the intimal layer becomes edematous, follows it fibrous connective tissue proliferation, follows it the accumulation of **cholesterine crystals** or accumulation of **neutral fats** inside **cholesterol clefts**.
- Most of the lipid substances were phagocytosed by foam cells - fat macrophages. This event is identified as '**atherosclerosis plaque**'.
- When the event progresses, becomes old and widespread, it is observed that the tissues forming the artery wall are destroyed.
- It is observed that a giant cell granuloma tissue is formed in the environment. *In some cases (necrotic events), calcifications are also present. Metaplastic cartilage tissue may also be encountered in old events.*

Atherosclerosis



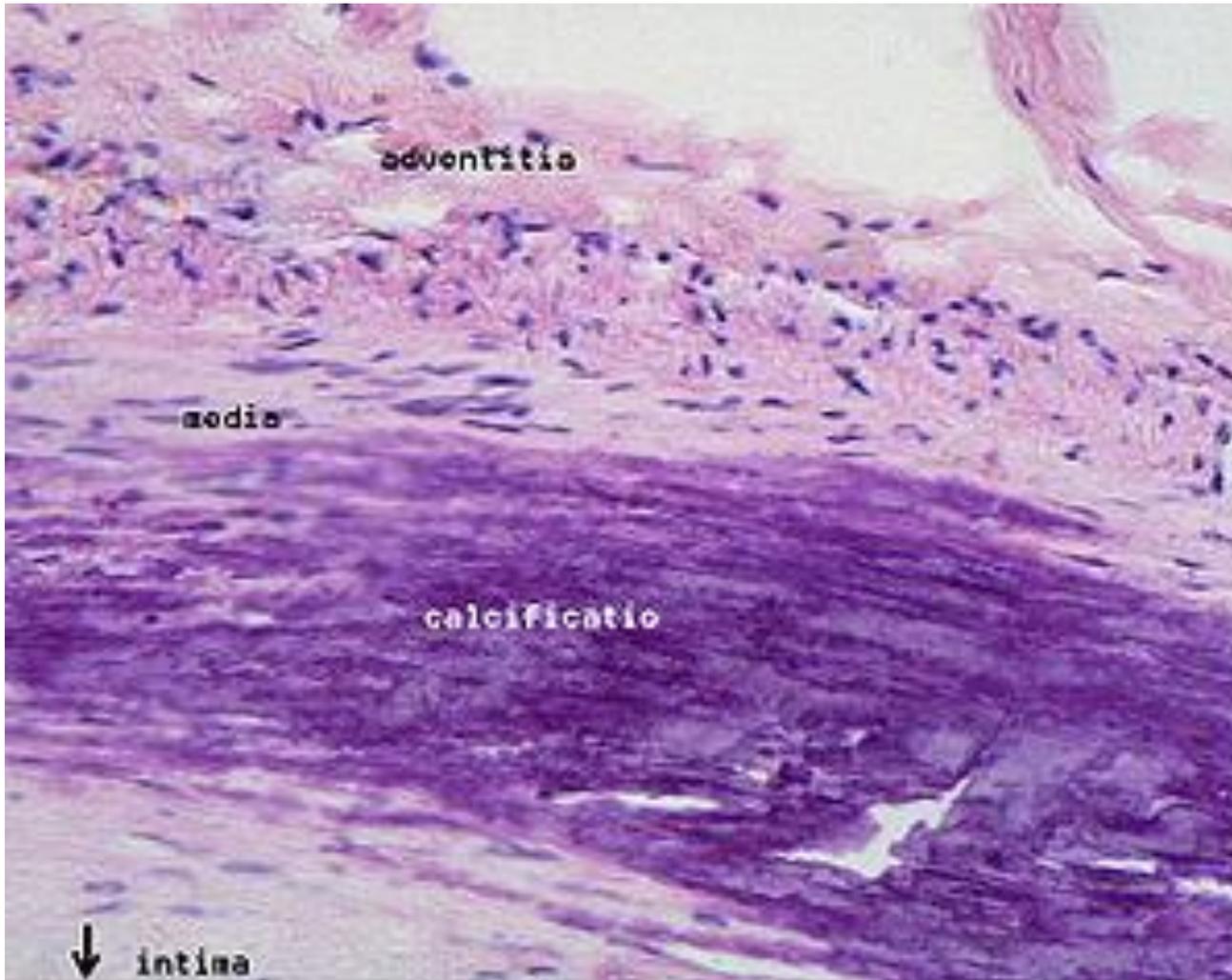
ARTERIOSCLEROSIS

MICROSCOPICAL FINDINGS:

b) Monckeberg's arteriosclerosis,

In affected arterial walls, in the medial layer of the areas where fibrous connective tissue growth, it is observed that it becomes thick due to the circle and ring-shaped **calcification zones**.

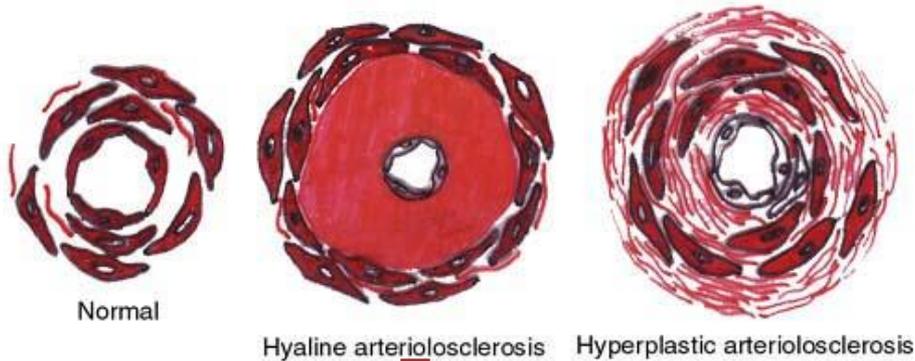
Monckeberg's arteriosclerosis



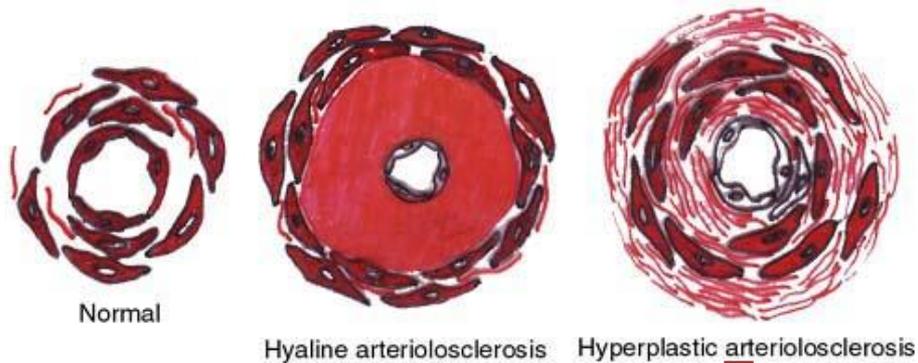
ARTERIOSCLEROSIS

MICROSCOPICAL FINDINGS:

c) Arteriolosclerosis,



- **In hyaline arteriolosclerosis**, a homogenous hyaline accumulation is initially observed in the subendothelial tissue.



- In the hyperplastic arteriolosclerosis, it is observed that the smooth muscle cells initially progress towards the intimal layer. In advanced stages, these cells overgrow and collagen accumulation is seen.

- **In old lesions**, such arteriolar walls (with respect to smooth muscle and fibrous connective tissue proliferation) appear to be a **concentric layer resembling onion section**.



Hyperplastic arteriosclerosis

VASCULITIS

- *Vasculitis*, or inflammation of a vessel, is characterized by the presence of *inflammatory cells* within and around the blood vessel wall *with* concomitant vessel wall damage as indicated by fibrin deposition, collagen degeneration, and necrosis of endothelial and smooth muscle cells.
- The term 'angitis' is also used instead.
- Arteritis
- Phlebitis

VASCULITIS

- In vasculitis;
- Vascular endothelium may be damaged and thrombosis may occur.
- Lymphohistiocytes and neutrophil leukocyte infiltrations may occur around the vein.
- Fibrinoid degeneration, necrosis, fibrosis, proliferation are observed in the vessel wall and calcification may occur.

VASCULITIS

Causes of vasculitis in domestic animals

- VIRAL;
 - Equine viral arteritis
 - Equine infectious anemia
 - African horse sickness
 - Hog cholera
 - *Corysa Gangrenosa Bovum* (Malignant Catarrhal Fever)
 - Bluetongue
 - Equine viral rhinopneumonitis (in the form of encephalomalacie)
 - Feline Infectious Peritonitis (FIP)
 - Aleutian disease (mink)

VASCULITIS

- **CHLAMYDIAL;**
 - Sporadic bovine encephalomyelitis (*Chlamydia psittaci*)
- **RICKETTSIAL;**
 - *Rickettsia rickettsii*
 - *Ehrlichia canis*, *Ehrlichia equi*
- **BACTERIAL;**
 - *Salmonella* spp.
 - *Erysipelothrix rhusiopathiae*
 - *Hemophilus somnus*, *H. suis*, *H. parasuis*, *H. pleuropneumoniae*
 - *Corynebacterium pseudotuberculosis*

VASCULITIS

- MYCOTIC;
 - mucormycosis
 - *Aspergillus fumigatus*
 - *Histoplasma farciminosum*
 - *Sporothrix schenckii*
- PROTOZOAL;
 - *Encephalitozoon caniculi*
 - *Besnoitia besnoiti*
- HELMINTHS;
 - *Strongylus vulgaris*, *Dirofilaria immitis*, *Spirocera lupi*, *Onchocerca* spp.,
 - *Elaeophora* spp., *Aelurostrongylus abstrusus*, *Angiostrongylus vasorum*,
 - *Schistosoma* spp. (Phlebitis), *Brugia* spp. (Lymphangitis).

VASCULITIS

Non-infectious;

- Immune mediated,
 - Systemic Lupus erythematosus
 - Rheumatoid arthritis
 - Polyarthriti nodosa
 - Anaphylactoid purpura
 - Staphylococcal hypersensitivity
 - Serum disease.
 - Some drugs
- Immune mediated;
 - Uremia

ARTERITIS

Is the inflammation of the tissues forming the artery wall.

- Cause by infectious - toxic - parasitic - immunological effects.

According to location:

- Endarteritis (when located in the endothelial layer),
- Mesarteritis (when located in intimal and medial layer),
- Periarteritis (when located in adventitia),
- Panarteritis (if inflammation spread to all layers).

ARTERITIS

- This classification of arteritis is not possible in practice. Because inflammation often involves several layers.
- Therefore, the classification of arterial inflammation can be done due to the nature of the exudate and the pathogenesis of the case.

ARTERITIS

- According to this;
 1. Arteritis serosa
 2. Arteritis purulenta
 3. Arteritis trombotica
 4. Arteritis necroticans
 5. Arteritis productiva

ARTERITIS

- ✓ **Arteritis serosa:** Serous exudate is present in the artery wall and the beginning of other inflammations.
- ✓ **Arteritis purulenta:** Purulent exudate penetrates the artery wall. Causes of different types of pus are responsible. It is a common type of arteritis in animals. It mostly occurs in the umbilical arteries in relation to the umbilical infections. Macroscopically, the vein is dirty-red-red and bulging.
 - Microscopically, neutrophil leukocyte-rich inflammatory edema, degeneration of the vascular wall, necrosis, desquamation of endothelial cells and lumen thrombosis can be seen.

ARTERITIS

- ✓ **Arteritis thrombotica:** is a purulent-trombotic inflammation that is about parasitic larvae occurs in all layers of artery wall.
- This type of arterial inflammation is seen with the invasion of Strongylus vulgaris larvae in horses.
- Parasitic larvae form embolus in arteries with blood. It causes injury to the endothelial layer. Therefore, local traumatic lesions develop. As a result of the accumulation of leukocytes and fibrin in intima and media, thromboses occur at the sites of injury and such inflammation occurs. If the thrombosis is organized over time, the vessel wall becomes thicker.
- Scar and calcification are formed.

ARTERITIS

- ✓ **Arteritis necroticans:** It is characterized by necrosis in the vein wall. Intima is rough-brown-greenish in color. Fibrin masses are noticeable. Thrombosis usually occurs in the lumen.
 - Microscopically, necrosis areas and cell debris are seen in pink homogenous appearance in the affected vessel. There are neutrophil leukocytes, lymphocytes and macrophage infiltrations around them. In chronic events, fibrous connective tissue proliferation is observed. In addition, aneurysms may be seen in place of these lesions.
- ✓ **Arteritis productiva:** Arterial walls are thick-hard-yellowish-brown. Excessive connective tissue proliferation, lymphohistiocytic cell infiltrations are seen. In the intima, connective tissue increase and thrombosis are formed. This type of features are seen in large vessels.

ARTERITIS

Important diseases with arteritis

- Equine Viral Arteritis (Pink Eye),
- African horse sickness
- Gangrenous Coryza (Malignant Catarrhal Fever),
- Hog Cholera (Pestis Suis, Schweinepest),
- Rocky Mountain Spotted Fever
- Uremic Arteritis,
- Panarteritis Nodosa.

Equine viral arteritis

- **Equine viral arteritis.** This disease is caused by species *Equine arteritis virus* (EAV), an RNA virus of the family *Arteriviridae*, genus *Arterivirus*, which is pathogenic only for horses and is cytopathic in equine kidney culture.

Equine viral arteritis

- Transmission of virus occurs primarily by *respiratory and venereal routes* during the acute phase of infection.

Equine viral arteritis

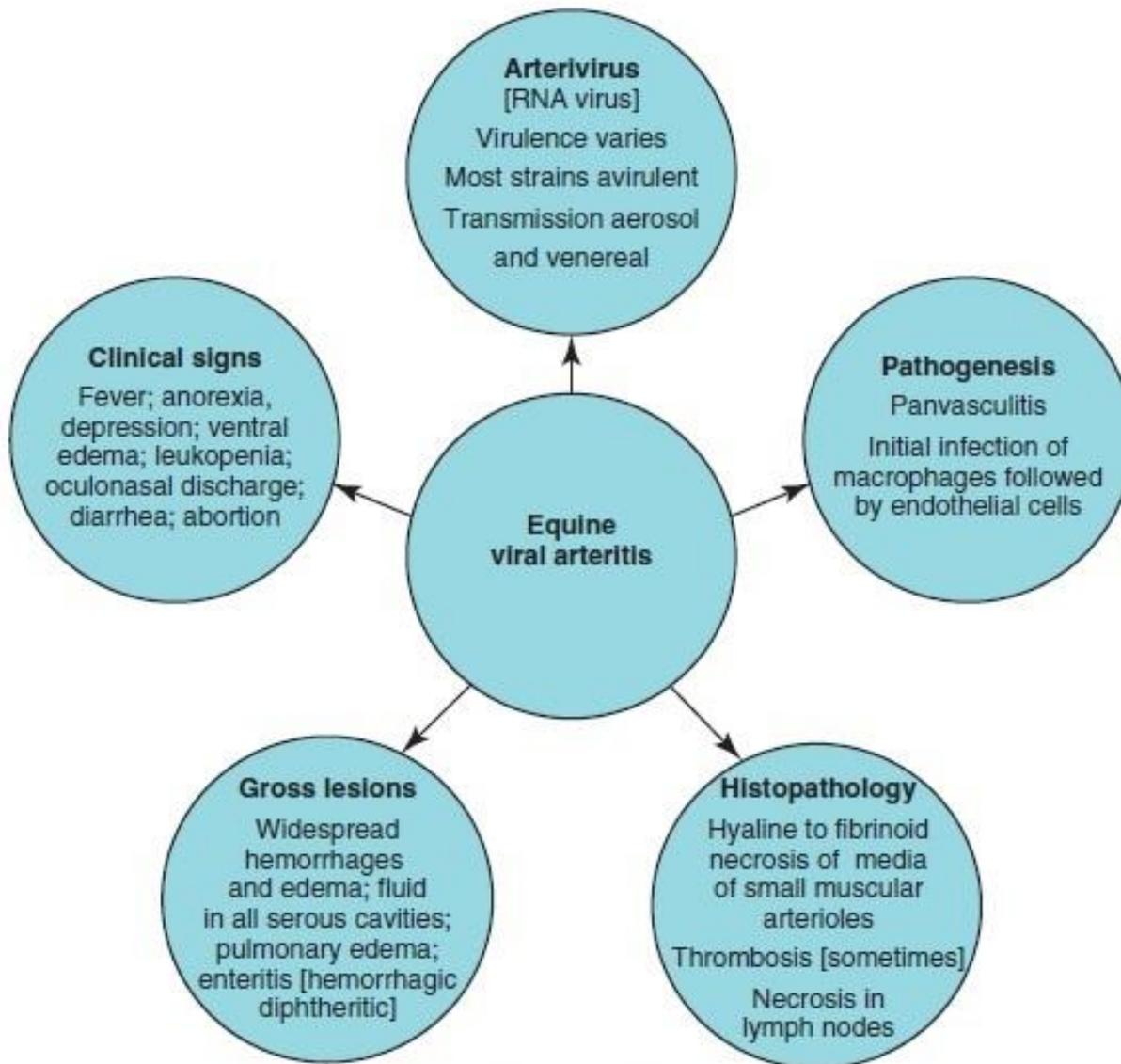
Macroscopical findings

- Transudate with **fibrin** in pericardium, thorax and abdominal cavity,
- Subcutaneous **edema**,
- **Hemorrhage** in various organs (pleura, endo - epicard, larynx, pharynx, muscles, etc.),
- Lymph nodes are bulging due to **edema and hyperemia**,
- Edema and catarrhal inflammation in the lung,
- Edematous enteric wall and catarrhal hemorrhagic enteritis,
- Edema around the aorta.

Equine viral arteritis

Microscopical findings

- **The presence of fibrinoid degeneration and necrosis** in the small arteries (0.5 mm diameter) is the characteristic finding.
- Since there are *no degenerative changes in endothelial cells*, thrombosis is not formed.
- However, in some cases, **thrombosis** may occur in the colon and caecum, and Hemorrhage and necrosis may be formed.



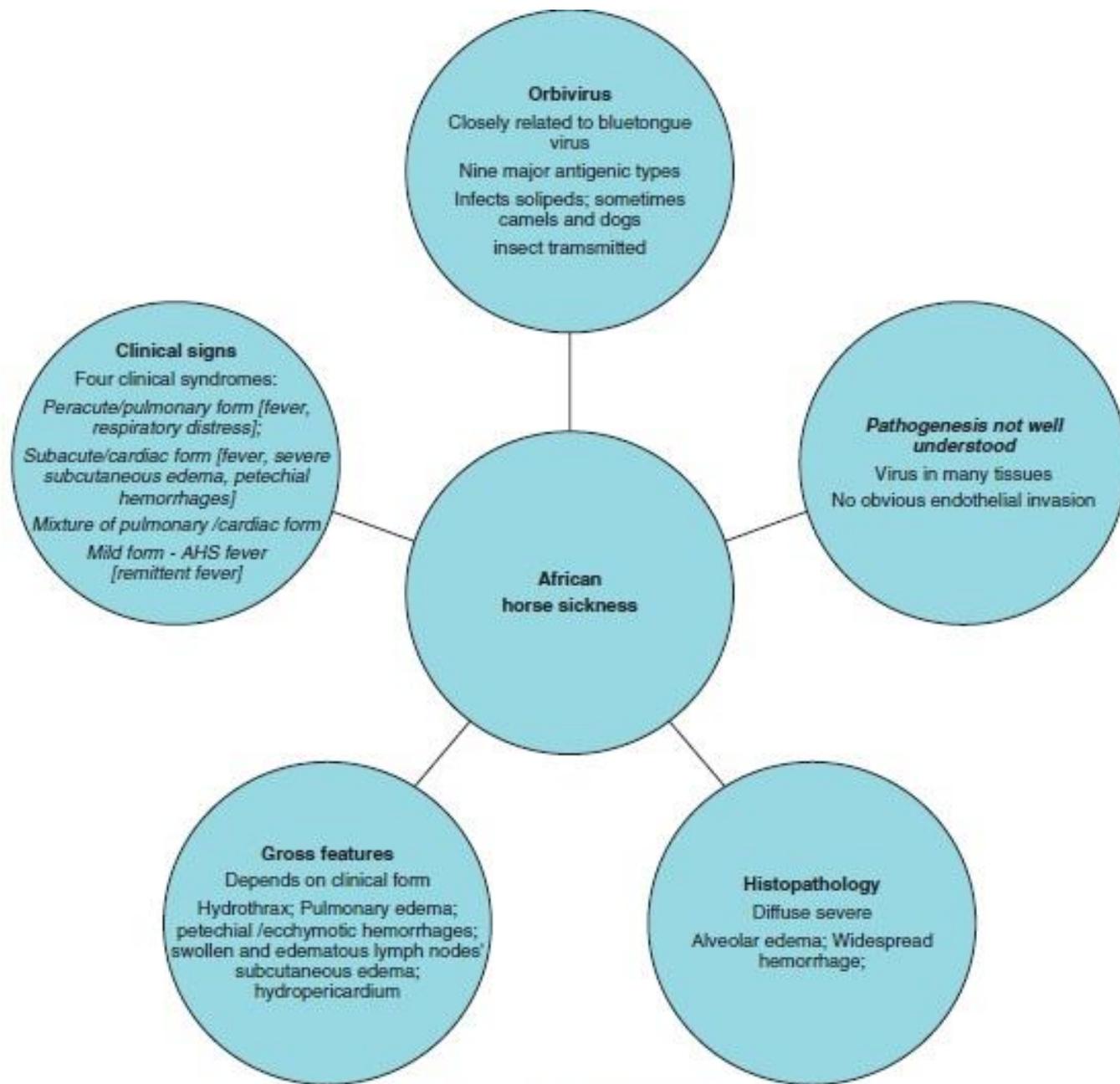
eFigure 1-20 Major features of equine viral arteritis.

African horse sickness

- The agent is orbivirus.
- It is transmitted by insects (culicoides).
- Dogs eating the flesh of infected horses can be infected even if they have disease.

African horse sickness

- **Clinically**; four forms were defined as lung, heart, mixed and horse fever.
- **Macroscopically**;
 - ✓ **In the lung form**, edema in the lungs, hydrothorax is seen.
 - ✓ **In the heart form**, **hydropericardium**, **ecchymotic hemorrhages** in epicard and endocard, these hemorrhages are concentrated along the coronary vessels, especially under the bicuspidal and tricuspidal valves and at the connection sites of the chordae tendineae in the papillary muscles.
 - ✓ **In mixed form**, lesions observed in lung and heart forms are observed.
- **Microscopically**; hemorrhages, edema, focal necrosis, inflammatory cell infiltrations and increase in connective tissue.



eFigure 1-21 Major features of African horse sickness (AHS).

Corysa Gangrenosa Bovum (Malignant Catarrhal Fever)

- Microscopic lesions that are pathognomonic are necrosis in lymphocytes in lymphoid tissue and **fibrinoid - necrotic vasculitis** especially in the wall and adventitia of small arteries and mononuclear cell infiltration.

Hog Cholera (Classical swine fever)

- *Classical swine fever (CSF) is a highly contagious viral disease of swine; it may occur as acute, subacute, chronic, or inapparent syndromes.*
- CSF is caused by a *Pestivirus*, a member of the family *Flaviviridae*.
- *Transmission* of the disease is usually by **direct contact** of infected pigs.
- The virus is found in the **urine** and **faeces** of the infected animals and **the eye and nasal discharge** and infects the environment.

Hog Cholera (Classical swine fever)

- *Clinically, classical swine fever is characteristically an **acute disease** of high morbidity and mortality, most animals surviving only to 14 days after showing the first signs of illness.*

Hog Cholera (Classical swine fever)

Macroscopically:

- The cadaver is dehydrated, the eyes are submerged.
- In the non-pigmented parts of the skin **bleeding** may occur.
- **Petechia** are observed in the periphery of the lymph nodes and **in the kidneys**.
- **Infarction of spleen** in acute cases is pathognomic.
- In the epicardium, **petechiae** are common. The pericardium contains a small amount of exudate in the color of straw.

Hog Cholera (Classical swine fever)

Macroscopically:

- With transplacental infection nervous system findings such as:
 - ✓ microencephalus,
 - ✓ hydrocephalus,
 - ✓ cerebellar hypoplasia,
 - ✓ Hypomyelinogenesis
 - ✓ pulmonary hypoplasia

Macroscopically:

- Changes in veins are in the form of primary degenerative changes in the endothelium and sometimes in the form of proliferative changes.
- In the vein, **fibrinoid vasculitis and degenerative changes and perivascular lymphocyte - histiocyte infiltrations** occur.
- Infarcts with diagnostic significance occur in the spleen.

Rocky Mountain spotted fever

- **Rocky Mountain spotted fever.** Rocky Mountain spotted fever (RMSF), a febrile exanthema caused by *Rickettsia rickettsii*,
- is an important rickettsiosis of dogs and humans
- **The ticks** most commonly responsible for the **transmission.**
- The factors proliferate in endothelial cells of small blood vessels.
- Is seen in dogs especially under 2 years of age, it is reported that **people are affected.**

Rocky Mountain spotted fever

Clinically:

- Stagnation, fever, lymphadenomegaly, dyspnea, conjunctivitis, paralumbar hyperesthesia, edema in the face and extremities, petechiae or hemorrhagic diathesis, vomiting and diarrhea are seen.

Rocky Mountain spotted fever

Macroscopically:

- Nasal and ear **edema**, ulcerative glossitis, scrotal dermatitis and **petechial hemorrhages** in mucosa, abdominal skin, pleural and gastric wall are observed.
- Hemorrhagic colitis and hemorrhagic lymphadenopathy may also occur.

Microscopically:

- **Necrotic vasculitis in capillaries and arterioles** in small veins; **perivascular lymphocytes and macrophage infiltrations** are observed.
- Acute splenitis and interstitial pneumonia; multifocal necrosis occurs in the myocardium, adrenal glands and liver.

Uremic Arteritis

- In uremia, changes occur especially in *muscular arteries and arterioles*.
- The changes are localized mostly in vessels of the *gastric mucosa, tongue, colon, gallbladder, urinary bladder, kidney*, and rarely in the *small intestine, myocardium and other organs*.
- The disease is observed in dogs.

Uremic Arteritis

- Macroscopically, the small arteries in the organs are gray-yellowish, showing thickening and bulging.
- Microscopically; **fibrinoid necrotic vasculitis** in media and adventitia, **neutrophil leukocyte** infiltrations in the intima.
- As a result of the lesions, uremic gastritis and **calcification** are seen in the stomach in relation to ischaemia.

Panarteritis nodosa (polyarteritis, or periarteritis nodosa)

- The term “polyarteritis nodosa” has been applied to a *heterogeneous group of arteritides*, which occur sporadically in all species of domestic animals.
- It develops as a result of immune reactions (antigen-antibodies).
- ***Small and medium-sized arteries*** undergo severe *necrotizing inflammation, often in a sharply segmental (nodose) pattern*, and with a predilection for branching points.
- ***As all layers of the arterial*** Wall are involved, the lesion is also referred to as “***panarteritis*”**.
- Arterioles, capillaries, and venules are not involved, and glomerulonephritis is not present.

Panarteritis nodosa

- It's a chronic event.
- After streptococcal infections in humans,,
- Aleutian disease in pox,
- Erysipelas in pork,
- Rarely seen in cattle, cats and dogs.

Panarteritis nodosa

It develops in three phases;

- **Fibrinoid deg. and necrosis phase:** Degeneration of muscle cells and multiple leukocytes.
- **Reperative phase:** First, leukocytes, lymphocytes, histiocyte infiltrations are seen, then develops proliferative events in adventitia (periarteritis nodosa).
- **Recovery phase:** Connective tissue proliferation and peri-arterial cell infiltration are observed in the media.

Panarteritis nodosa

Depending on the vascular changes,

- aneurysm in vessels,
- ulcer can develop.
- and vessels.
- It is more common in organs such as heart (coronary vessels), kidney, gastrointestinal system.

Venous Disease

Dilation of a vein

Enlargement of inner diameter of the veins and is divided into two main groups;

Phlebectasia: It is a widespread extension of the vein, along all vessel.

Varix-Varicose: A dilation or swelling of a vein. Dilatation locally and like a *pouch*.

- The most common expansion is observed in varix.
- For example;
 - *Varicocele* in the vena plexus of the funiculus spermaticus
 - *Haemorrhoids* of haemorrhoidal plexus
 - *Telangiectasia* in liver or in the small veins in the skin are called.

The reasons;

- Stagnation of blood flow,
- Hereditary or acquired weakness of the vena wall tissues,
- Vena porta thrombosis,
- Cirrhosis of the liver

- **In the case of phlebectasia,** by increasing the functional needs, the veins **are expanding totally.** In this case, some areas of the vena wall are thinned, and some areas become thicker as a result of tissue hyperplasia.

Phlebectasia cases are seen *in vena mammaria* in cows, and *vena thoracica externa, vena caphena* and *vena spermatica* in horses. Enlarged veins may reach the thumb thickness.

In the case of varix, because of **the local disorder** in the veins wall and the blood flow, veins form varix by *expanding into bags or pouches.*

- **Thrombosis** or **sclerosis** may be sequelae.
- The acquired portosystemic anastomoses noted above usually result from dilation of pre-existing microscopic venous anastomoses to produce collateral venous drainage.

- In humans, varix is very suitable for **rupturing**.

Complete or incomplete rupturs of varixs can result in fatal **bleeding**.

Varix in the legs of people, *chronic subcutaneous edema*, the skin under the *fibrous connective tissue* reproduction leads to the formation of **elephant leg (elephantiasis)**.

- Varix and phlebectasie lesions are permanent for life.
- Varixs cause stasis and circulatory disorders in the region where they occur.
- Since the animal body is covered with hairs, varix and phlebectasies are hardly noticeable.

Phlebitis

- **A) Periphelebitis,**
- **B) Endophlebitis**

A) Periphlebitis

The beginning of the inflammation in the adventitia layer.

- ❖ **Periphlebitis serosa,**
- ❖ **Periphlebitis purulenta,**
- ❖ **Periphlebitis gangrenosa,**
- ❖ **Periphlebitis productiva.**

- B) Endophlebitis
- In the intima and media layers of the inflammation.

- ❖ **Endophlebitis purulenta,**
- ❖ **Thromboendophlebitis,**
- ❖ **Endophlebitis productiva.**

Pathology of lymph vessels

Lymphangiectasie

- The expansion of the Lymph vessels lumen.
- The enlargement is mostly seen in the serous and subserous lymph vessels such as mesenterium, peritoneum, pleura and epicart.
- The main reasons are the common inflammation in the tissues and tumor formation.

Lymphangitis

- Lymphangitis serosa(simplex)
- Lymphangitis purulenta.
- Lymphangitis productiva.