MJF COLLEGE OF VETERINARY AND ANIMAL SCIENCE, CHOMU, JAIPUR

DEPARTMENT OF VETERINARY PATHOLOGY

EDEMA

HYPEREMIA AND CONGESTION

HEMORRHAGE

HEMOSTASIS & THROMBOSIS

EMBOLISM

INFRACTION

SHOCK

Embolis

- An embolus (plural emboli) is any foreign body floating in the blood.

- **An embolus is an intravascular solid, liquid, or gaseous mass that is carried by the blood to a site away from its point of origin**.

- The vast majority of emboli derive from a dislodged thrombus—hence the term thromboembolism
- However other types can occur
- In animals,more common in arteries and capillary
- In human, more common in veins

Type or Composition of emboli

- Thromboembolism
- Bacteria
- Parasites
- Fat emboli
- Air or gas emboli
- Other types of emboli

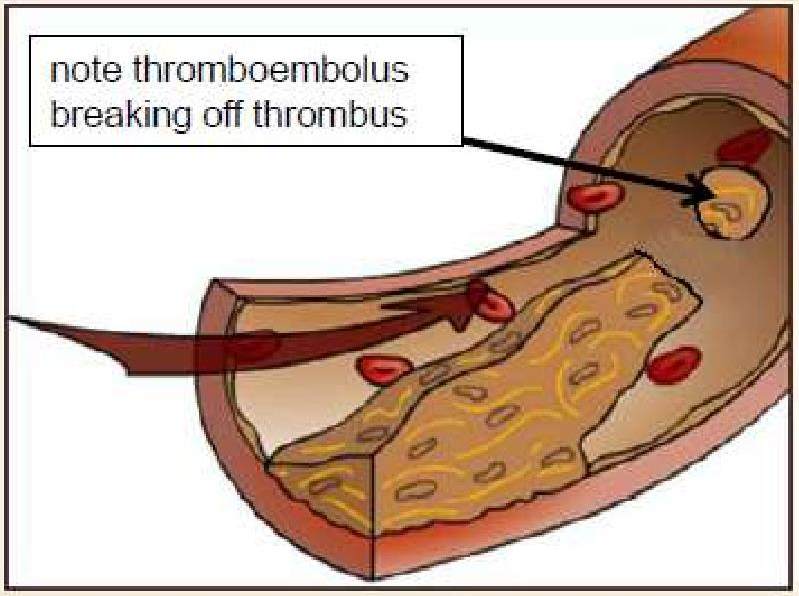
Thromboembolism

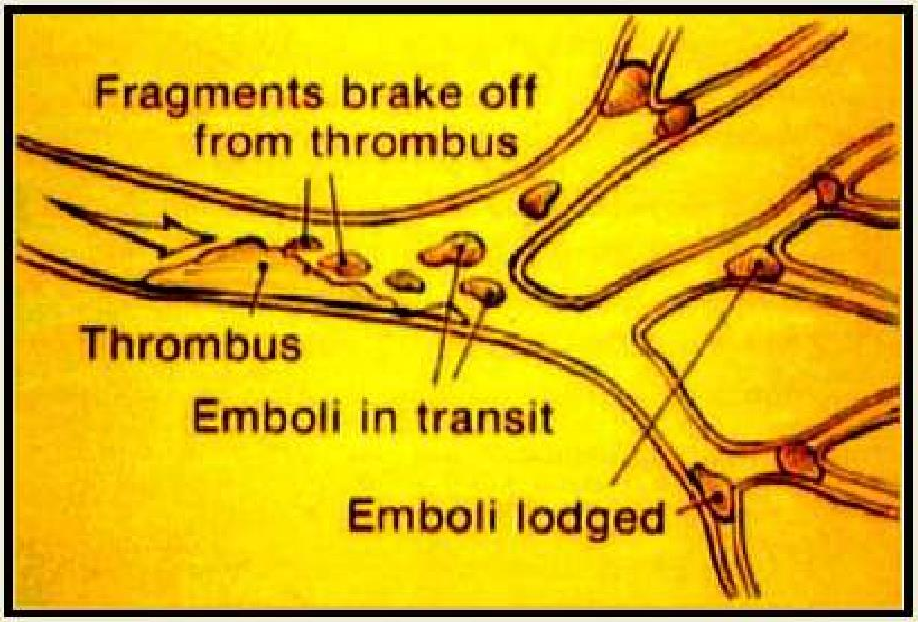
Thromboembolism

- occlusion of a blood vessel by an embolus that has broken away from a thrombus
- localizes at point where it can not longer "fit" through

Thromboembolus / Thromboemboli *(pl.)*

- the piece(s) of thrombotic material transported in the bloodstream to another site





Thromboembolism

Thrombosis at a vessel bifurcation, gives a saddle shape to the thrombus, ie "saddle thrombus"

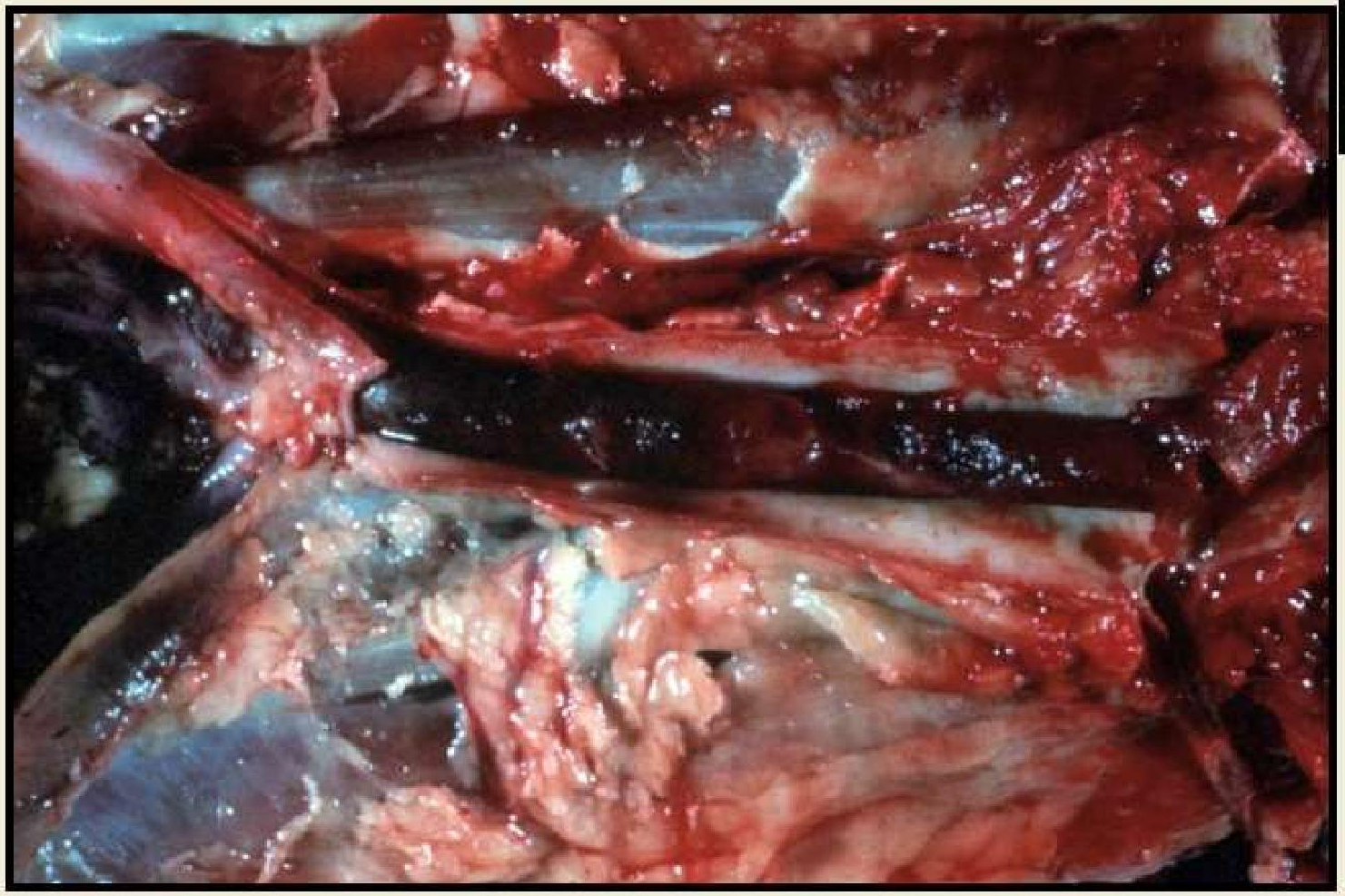


Fig. 2-35 Saddle thromboembolus, iliac-aortic bifurcation, cat. Cardiac thromboemboli usually lodge at the bifurcation of the aorta into the external iliac arteries with a portion of the thromboembolus entering each iliac vessel to form a saddle thromboembolus. A saddle thromboembolus is not attached to the wall of the aorta or iliac arteries and is easily removed at necropsy. The thromboembolus is composed of layers of platelets and fibrin in which there are enmeshed erythrocytes.

Infectious causes of Thrombosis or Thromboembolism

- bacteria or viruses can cause localized or widespread endothelial damage (thrombosis, +/- thromboembolism)

Bacterial endocarditis in cattle often involves the right AV valves. They often give rise to septic thromboemboli which shower and implant in small branches of the pulmonary artery, resulting in scattered inflammatory foci (ie embolic pneumonia)

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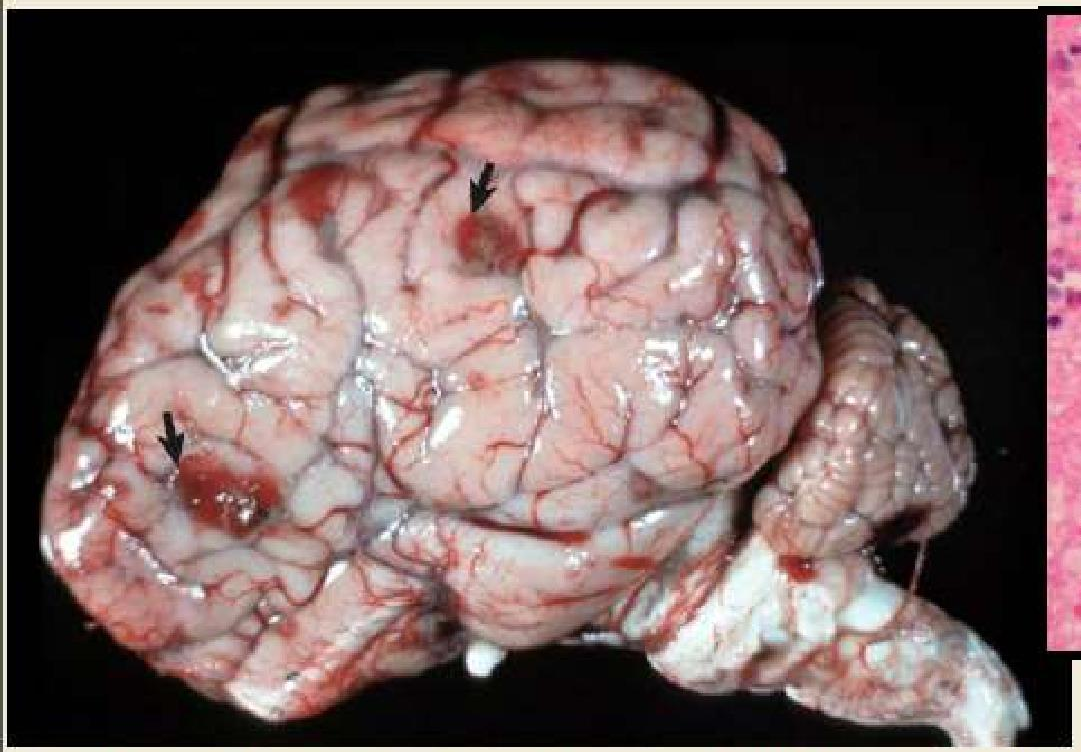


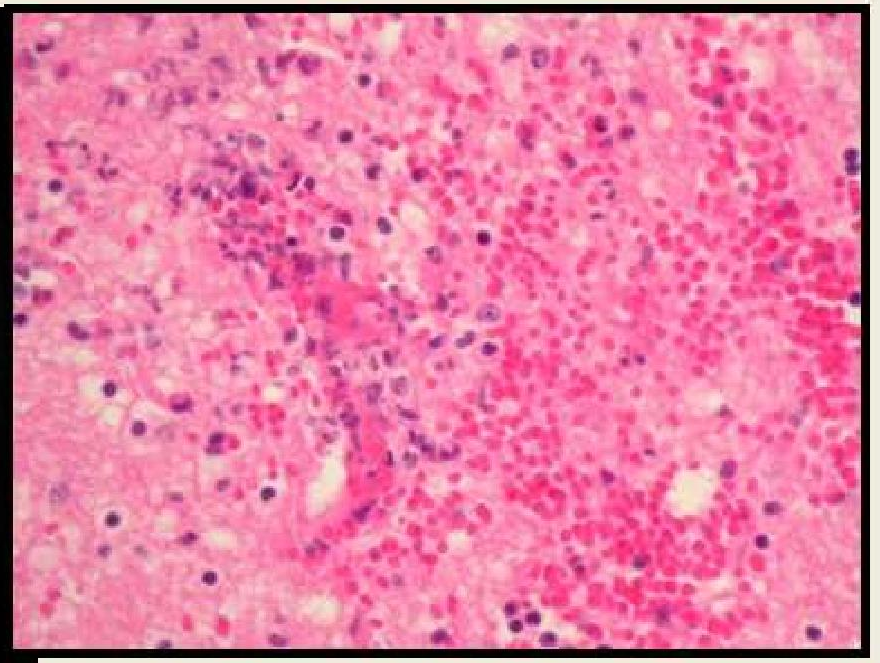
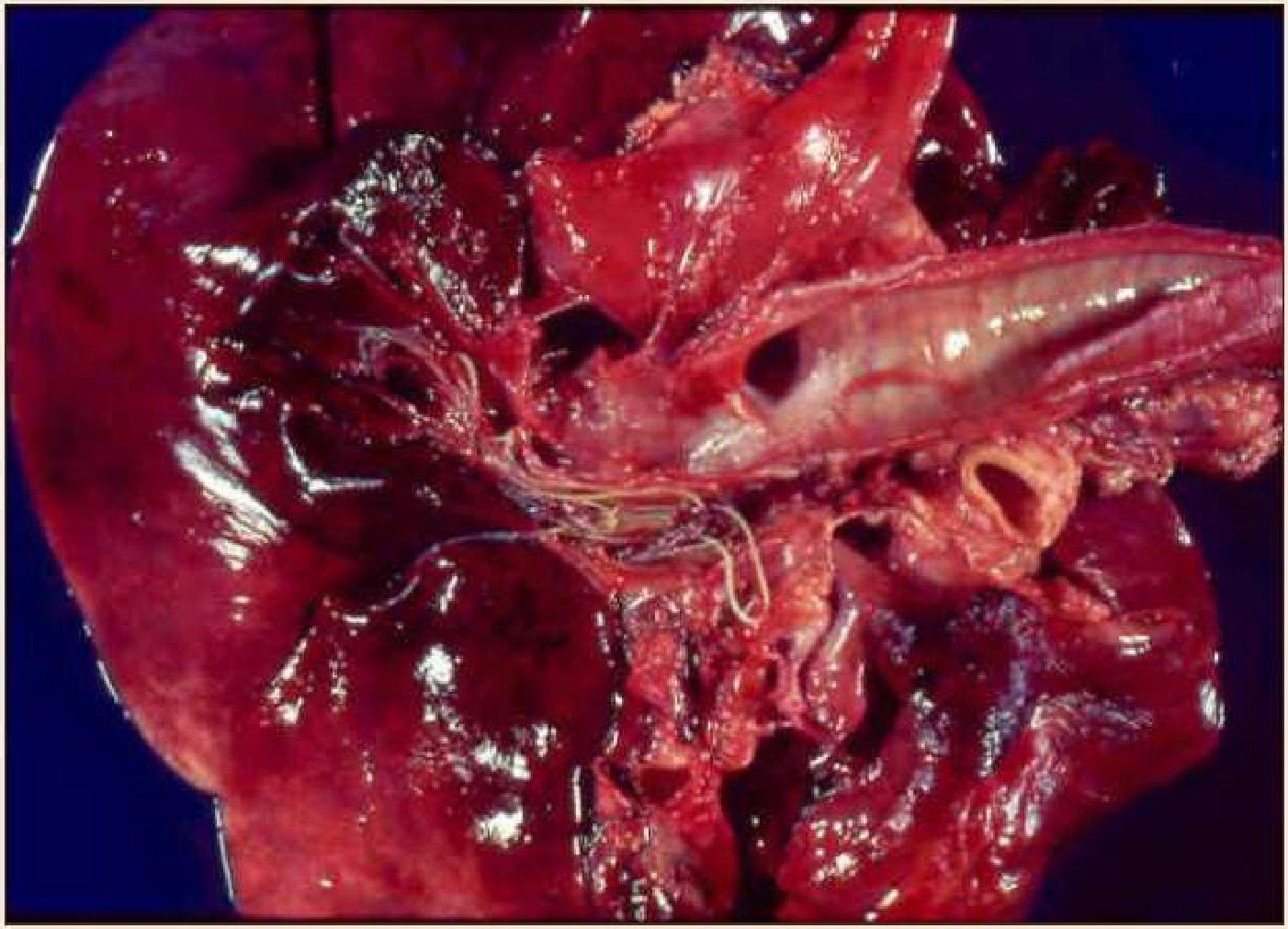


Fig. 14-48 Thrombotic meningoencephalitis (previously referred to as thromboembolic meningoencephalitis), cerebrum, steer. On the surface of the cerebral cortex (arrows) are several red-brown lesions. These lesions are areas of necrosis, hemorrhage, and inflammation secondary to vasculitis and thrombosis caused by Histophilus somni (formerly Haemophilus somnus). Such septic infarcts are distributed randomly (hematogenous portal of entry) throughout the central nervous system, including the spinal cord. The lesions depicted here are unusually severe.

Composition of Emboli

Parasites - nematodes

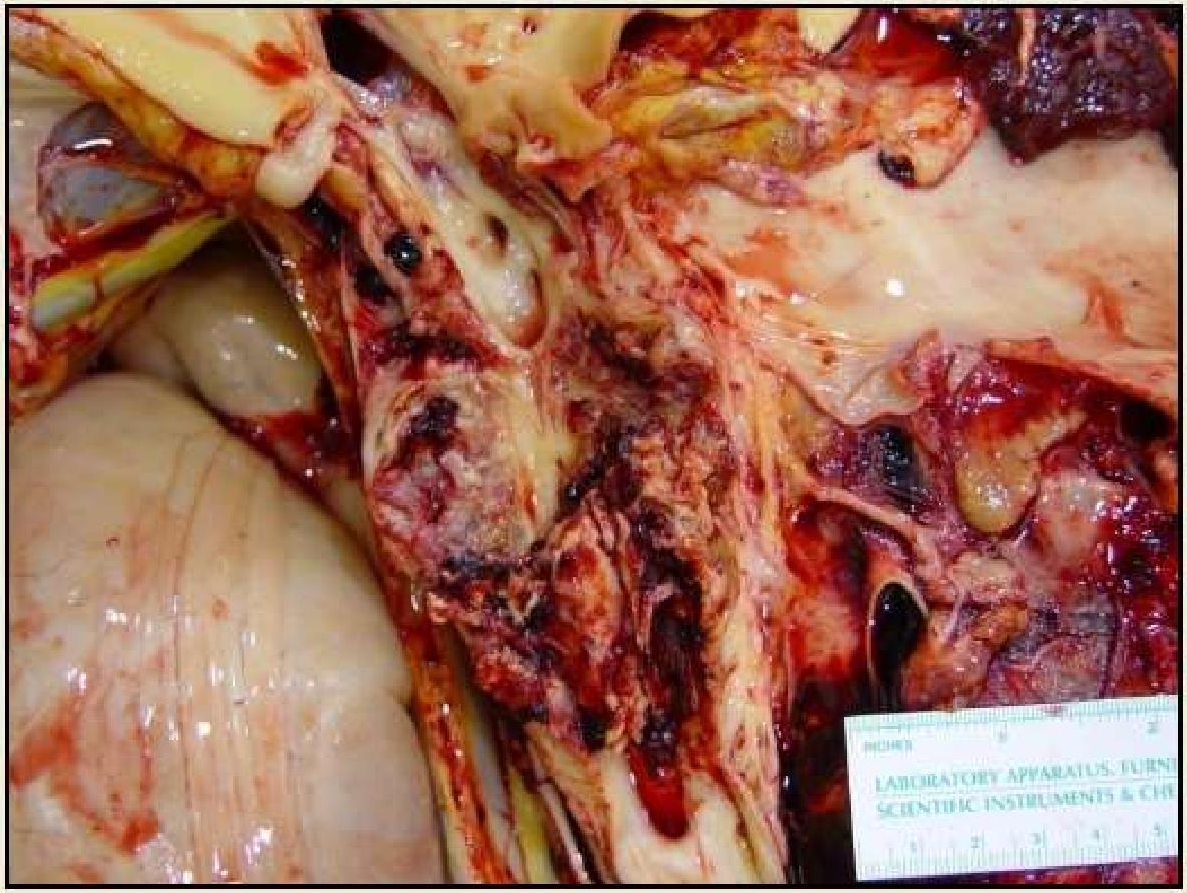
- Dirofilaria immitis



Composition of Emboli

Parasites - Nematode larvae

- *Ascarid sp*
- *Strongylus vulgaris*

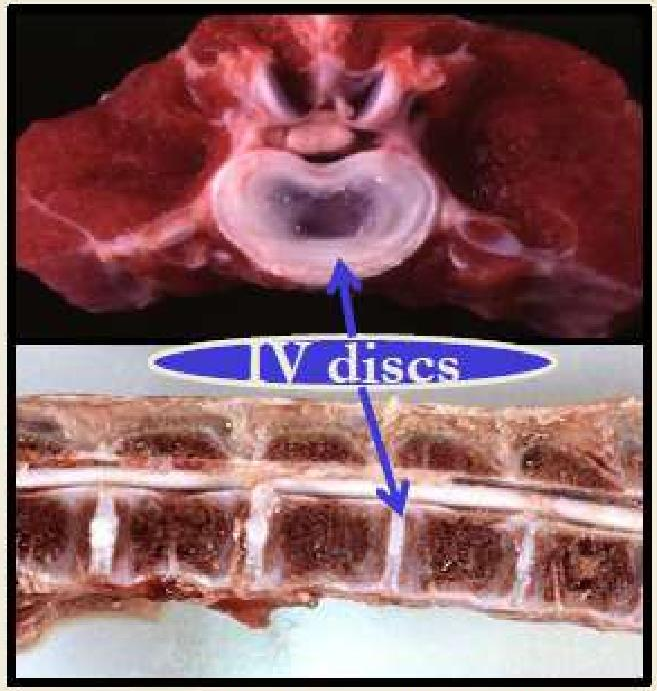


Arteritis with thrombosis, cranial mesenteric artery, horse. Damage to the cranial mesenteric artery, by strongyle larval migration is relatively common in horses. Strongyle larvae are often found within the lesion (image to the right) and occasionally may be found within the resulting thromboemboli (most emboli don't contain the larvae).

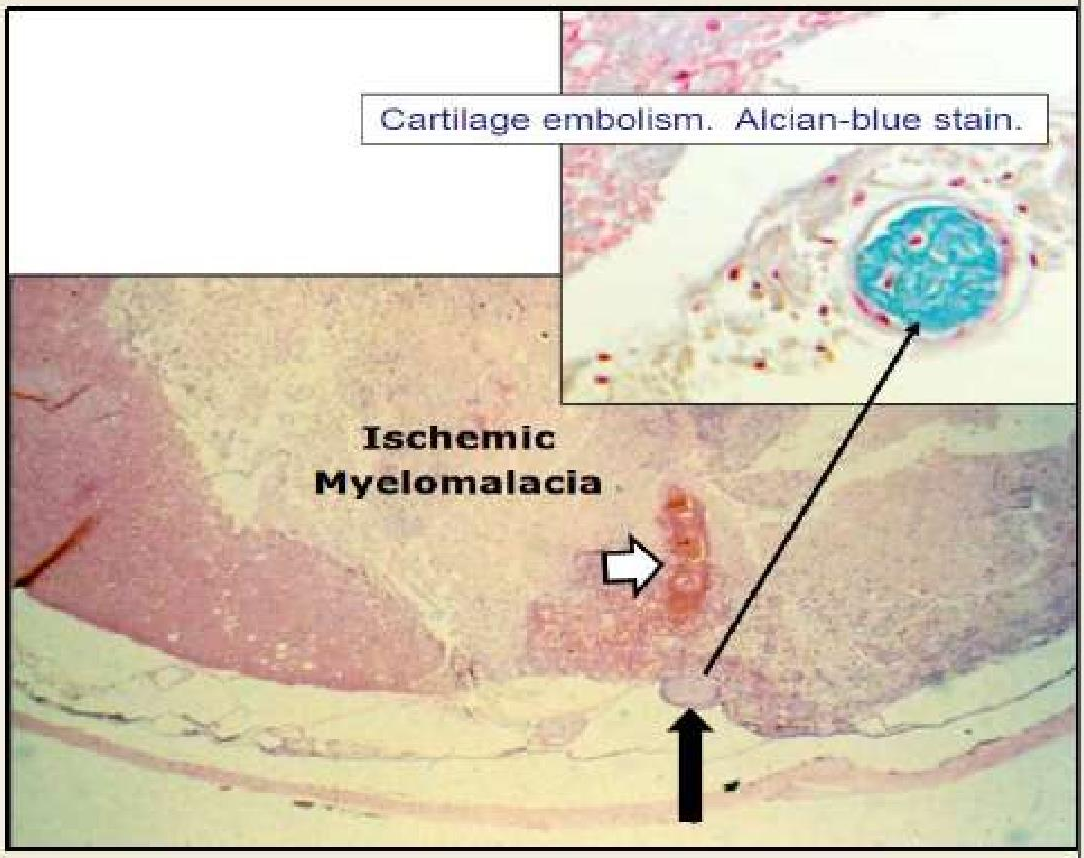
Composition of Emboli

Fibrocartilagenous emboli

- traumatic implantion of intervertebral disk material into spinal vessels
- causes necrotizing myelopathy (spinal cord infarcts)



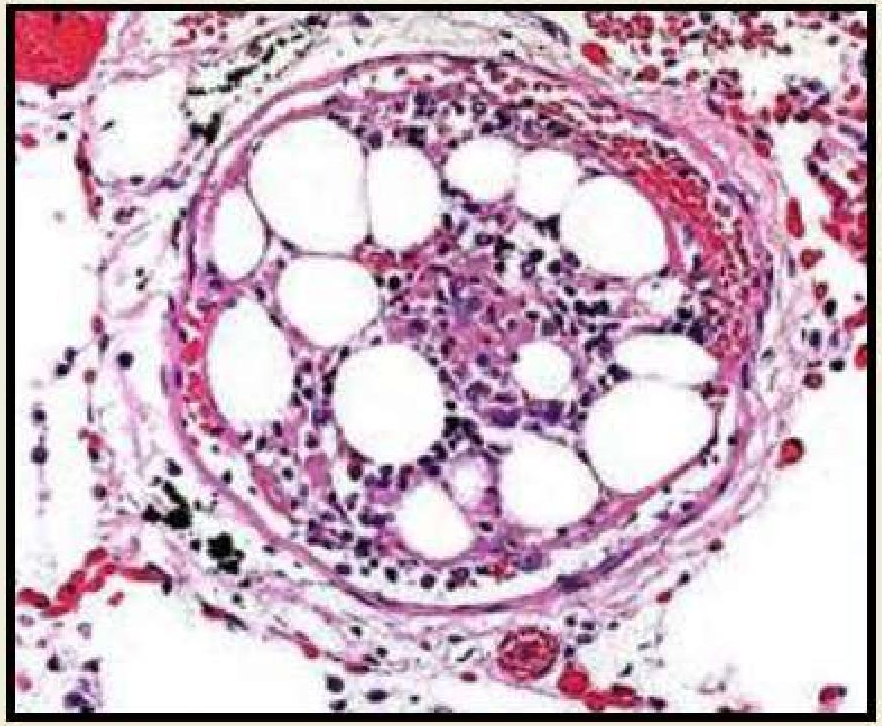
Fibrocartilagenous embolism, spinal cord, dog. Note vessel occluded with a cartilaginous embolism (large black arrow). As a result of the blocked vessel there is ischemic necrosis of the spinal cord (myelomalacia) with a focal area of hemorrhage (white arrow).
At higher magnification (inset) the cartilaginous material in vessels stains bright blue with an Alcian blue stain.



Composition of Emboli

Fat

- bone fractures
- prolonged surgery
- osteomyelitis

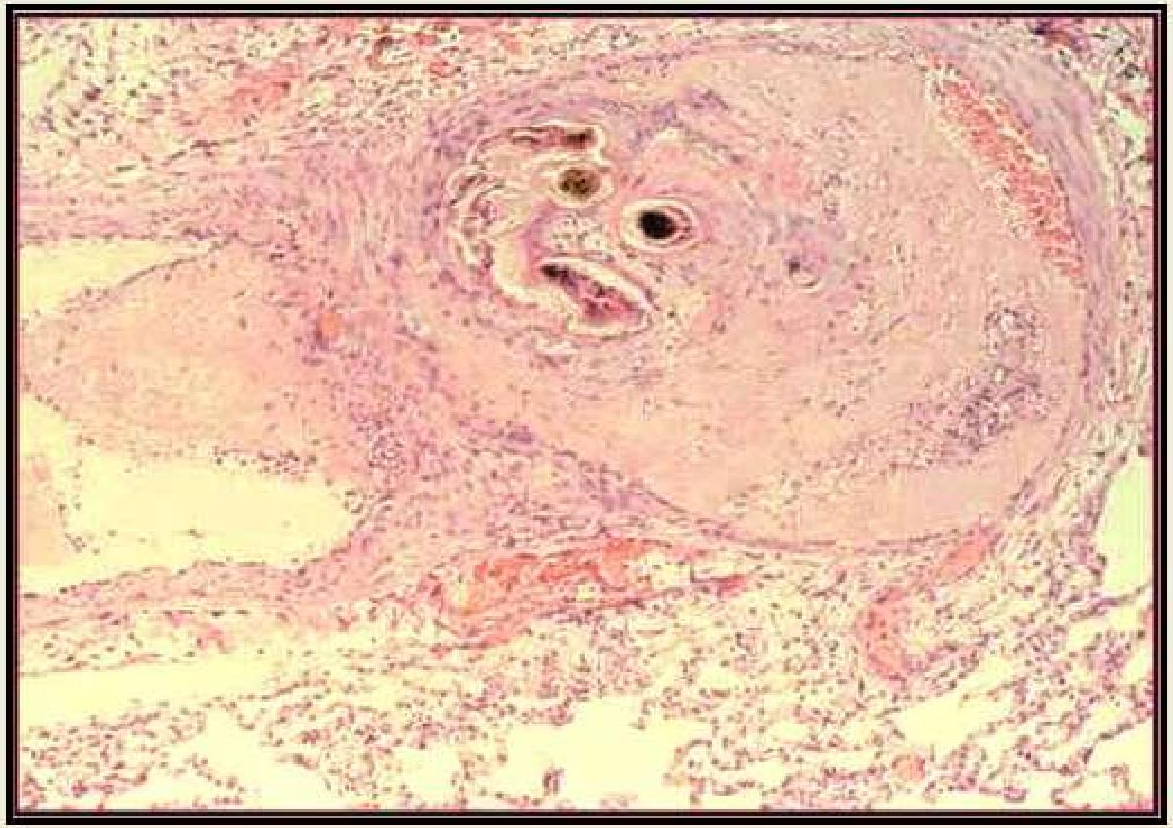


Fat embolus in a pulmonary artery, human. Note, bone marrow embolus in a medium-sized artery in the lung of a human patient following CPR (trauma).

Composition of Emboli

Other

- foreign material (eg hair, air bubbles)
- tumor cells
- amnionic fluid
- etc



note fragments of hair which have embolized to the lung (likely resulting from and injection or some other form of traumatic implantation of hair into a vein).

Composition of Emboli

Air or gas emboli

- Small amount of air during IV- no clinical significance
- More than 100 ml – clinical significance

Caisson disease/Decompression sickness (DCS) / Bends(pain) : is a condition that occurs when divers come back to the surface too quickly after being deep under water.

- It is caused by the formation of nitrogen bubbles in the bl
and, in the worst cases, c
death.

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Infarction

An infarct is an area of ischemic necrosis caused by occlusion of either the arterial supply, or rarely the venous drainage in a particular tissue

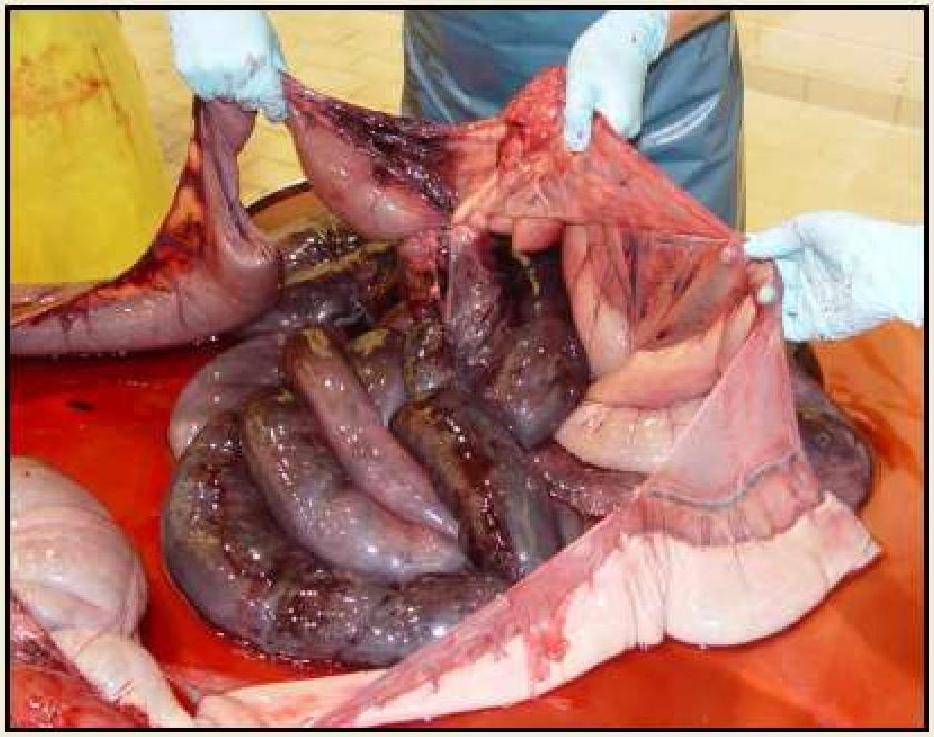
- ~40% of human deaths result of CV disease, esp infarction (heart & brain)
- Pulmonary, intestinal and renal infarction most common in animals

Infarction :

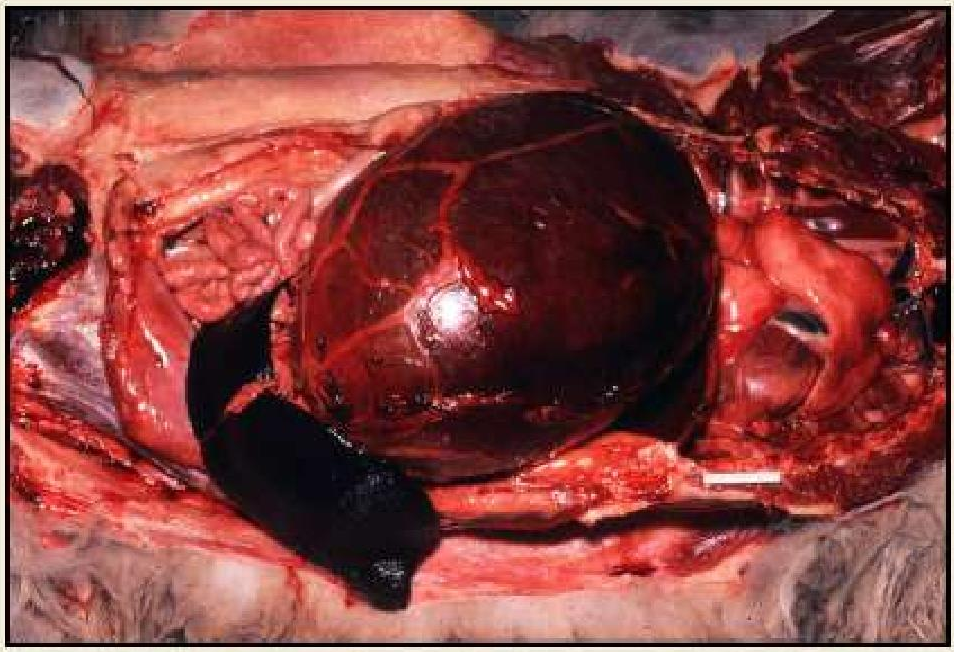
Etiology

- Artery or Venous obstruction
  - Thrombi and emboli : Most common
  - Torsion, volvulus
  - Narrowing of an artery
  - Compression of arteries by expanding tumours, abscesses, cysts, or by inflammatory fibrous adhesions
  - Poisonous compounds or drugs, such as ergot by inducing contraction of the musculature of arterial walls

Acute Blockage of Portal Venous System



Venous infarction of a segment of small intestine due to strangulation by a pedunculated lipoma

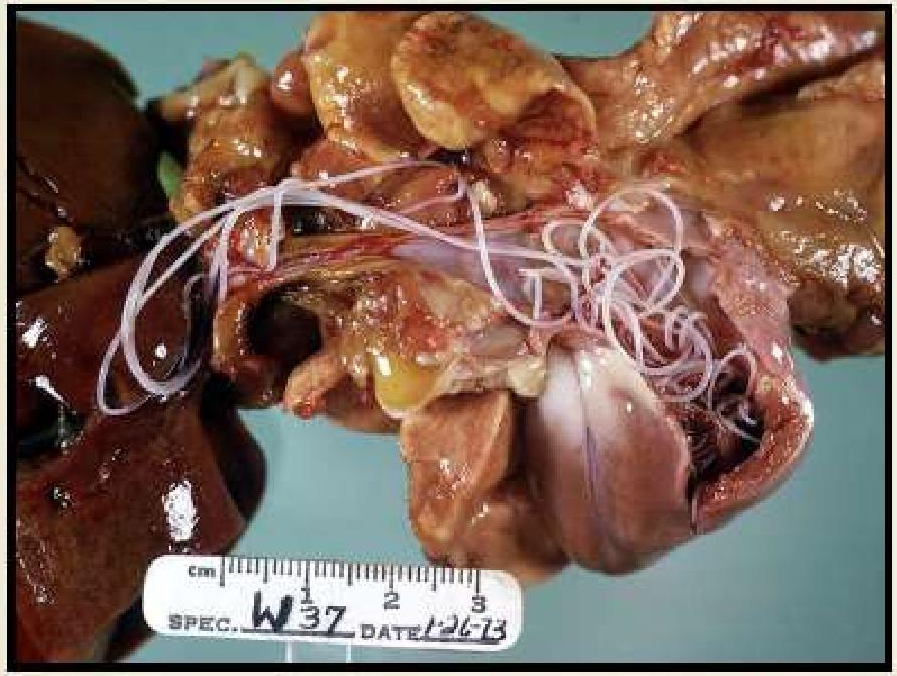


Gastric volvulus (torsion) in a dog → twisting of vessels → obstructs gastric portion of portal venous system → severe venous congestion (acute, local, congestion) → ischemia (necrosis) → loss of endothelial integrity → hemorrhage → shock → death

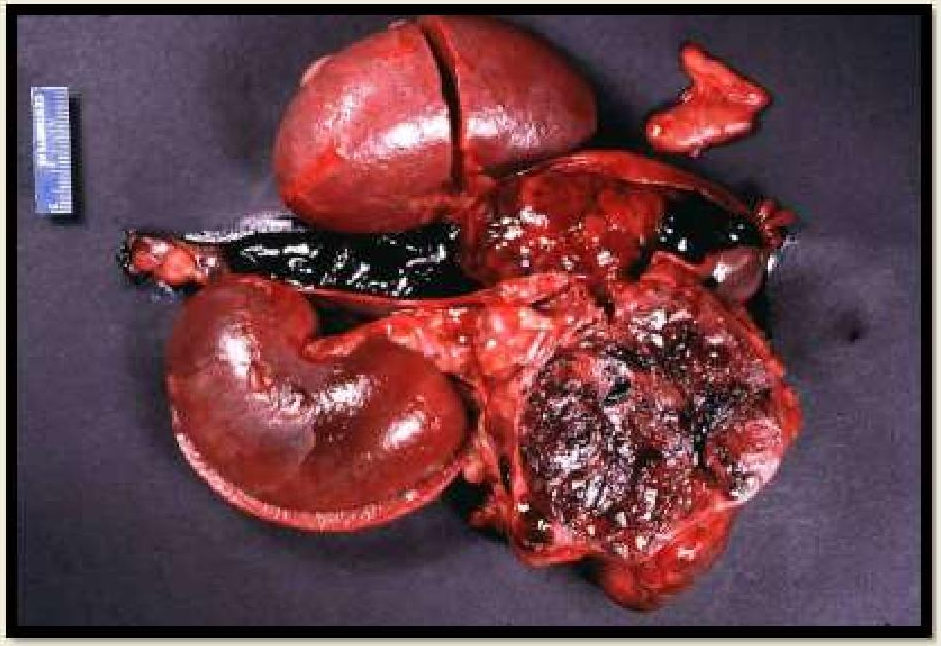
Blockage of Posterior Vena Cava

Etiology

- in dogs, heartworm (high burdens) or tumor invasion



With heavy burden of heartworm, adults can sometime be in right heart (ventricle & atria) and caudal vena cava.

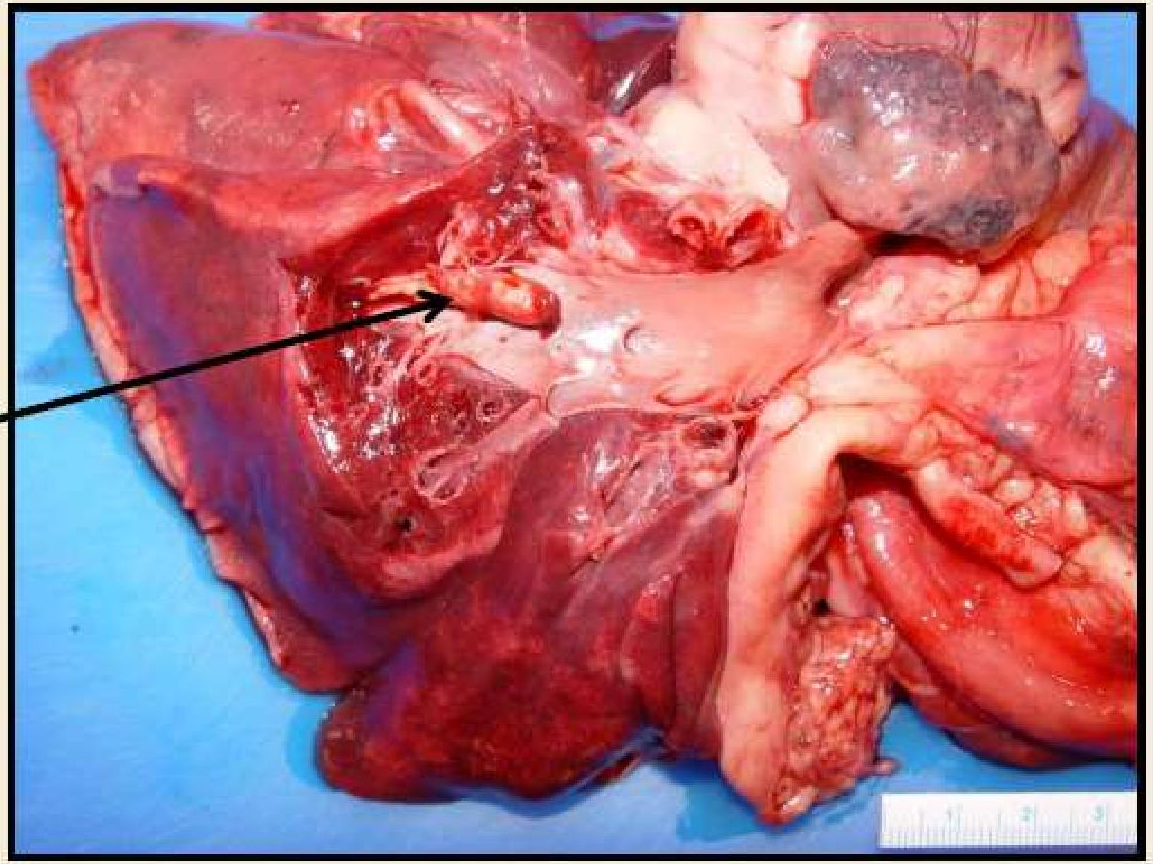


Pheochromocytoma (adrenal medullary tumor), dog. Note local invasion of vena cava which would impair venous return.

Pulmonary Artery Thrombosis

- can be due to a variety of causes:
  - pneumonia
  - parasities (eg heartworm)
  - hypercoagulability (eg nephrotic syndrome, hyperadrenocorticism)
  - liver abscess (ruminants)
  - deep vein thromboembolism (humans)

note large thrombus in the pulmonary artery of a dog with hypercoagulability due to Cushing's disease (hyperglucocorticoidism)



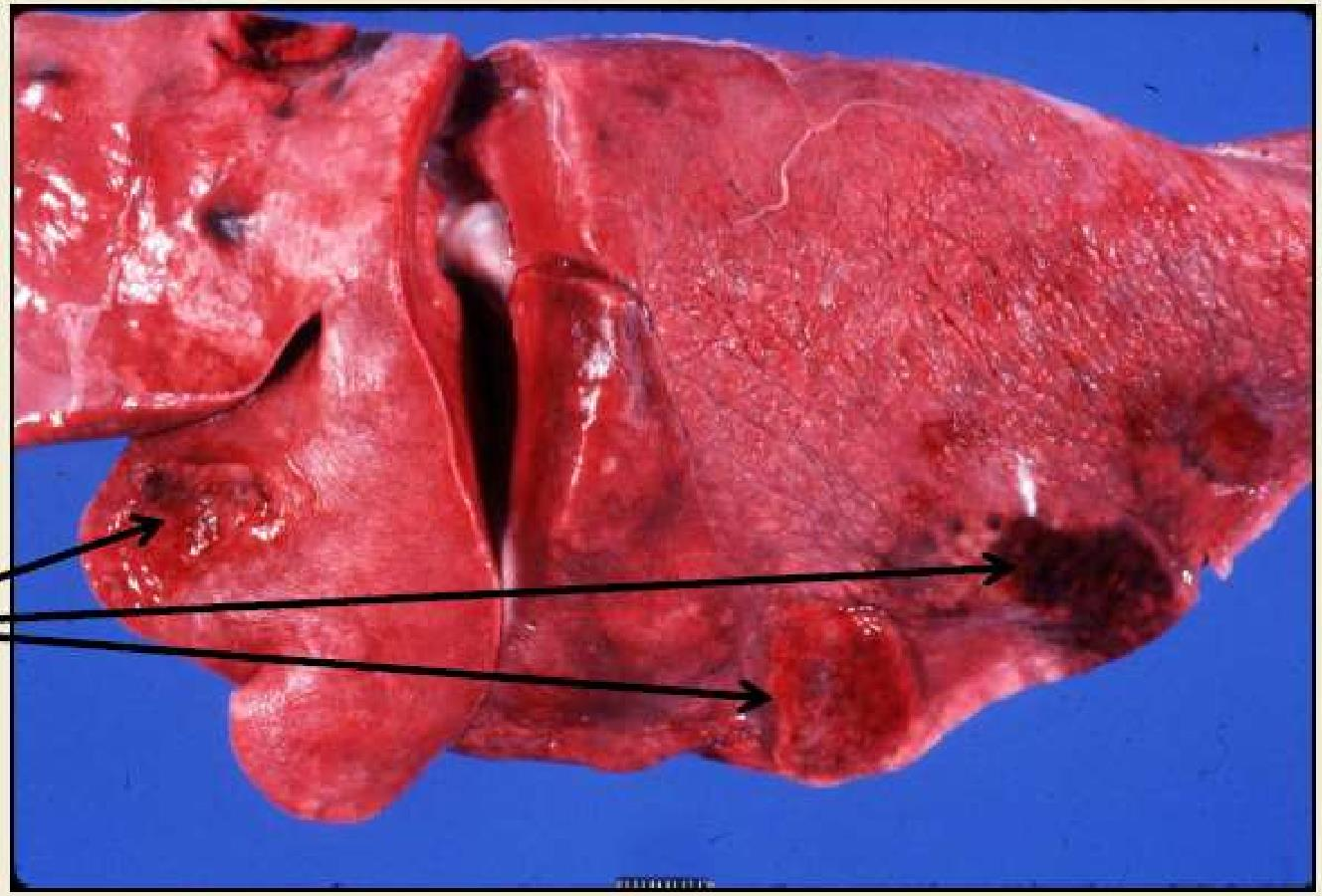
Pulmonary Artery Thrombosis

Result

- depends upon size of artery blocked: Large artery → death

  Small artery → infarction (usually red)

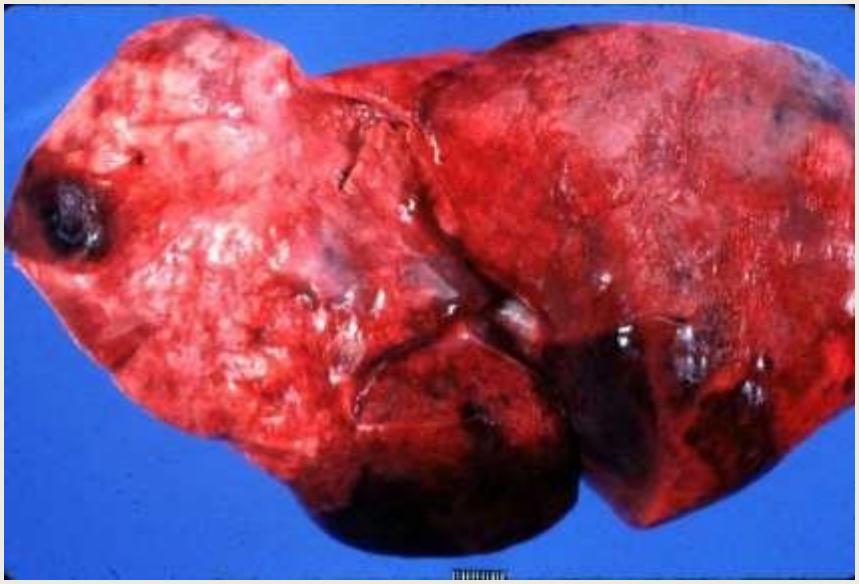
Note several pulmonary infarcts



Types of Infarcts or Gross Morphology

1. Red Infarct or hemorrhagic

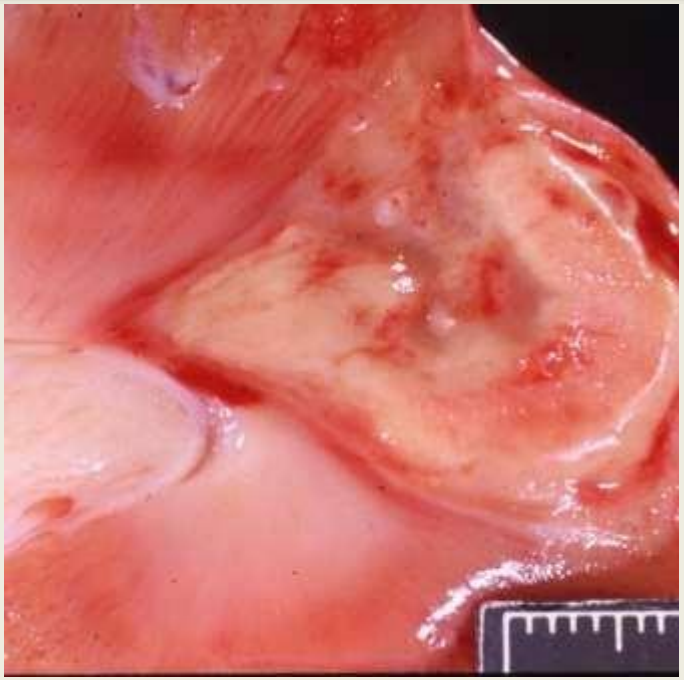
- Venous infarction (no drainage)
- In loose tissues (e.g., lung) where blood can collect in infarcted zones
- in tissues with dual circulations such as lung and small intestine



Types of Infarcts or Gross Morphology

2. White infarcts or Pale/anaemic

- Arterial occlusions
- In solid organs with end-arterial circulations (e.g., heart and kidney)
- Tissue density limits the seepage of blood from adjoining vascular beds



Types of Infarcts or Gross Morphology

3. Septic Infarct or Bend

• mostly from a septic (bacterial infected) thromboembolus

• occasionally necrotic tissue seeded by opportunistic bacteria

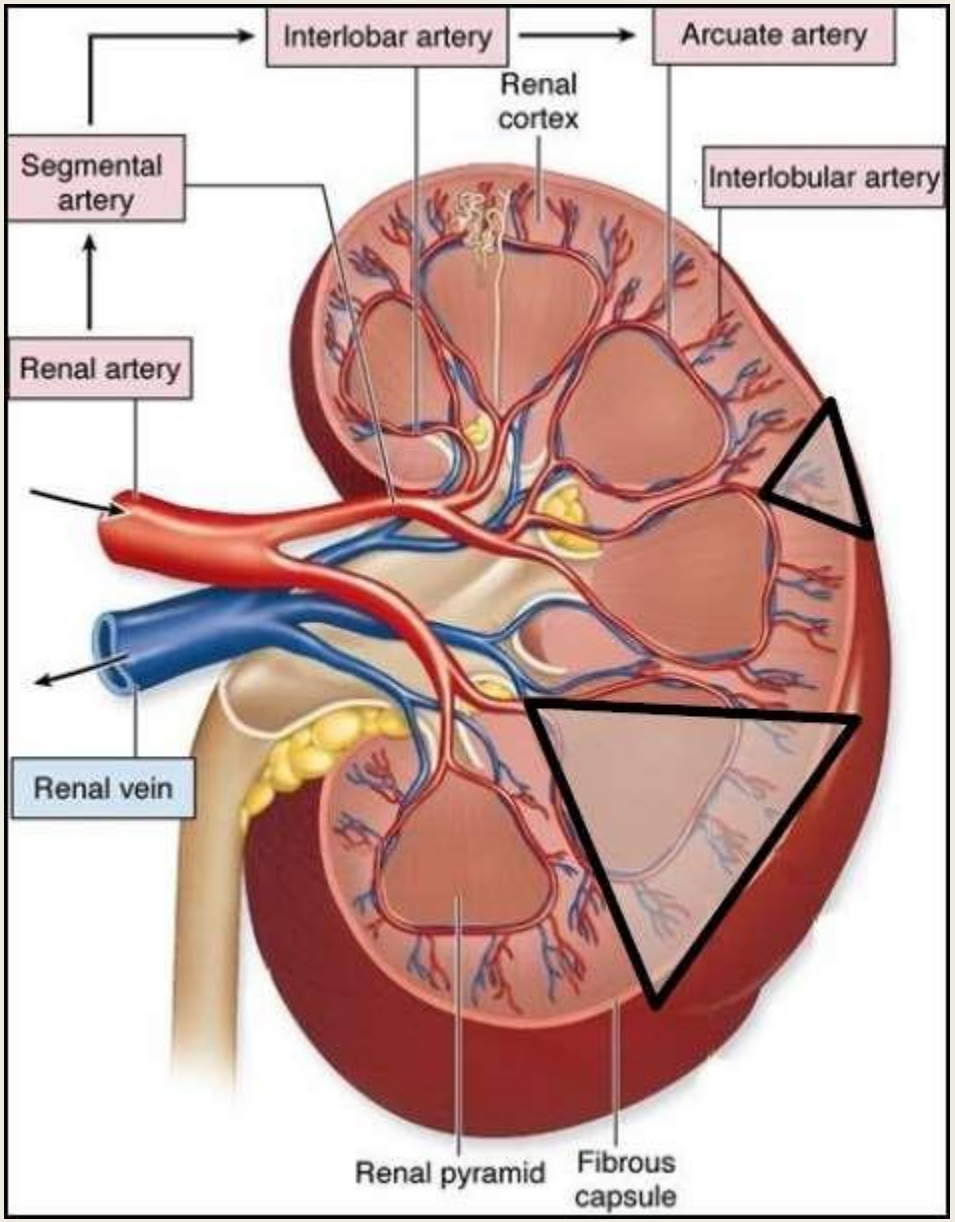
Myocarditis, necrosuppurative focal. dog. This focal area of myocardial necrosis and inflammation is due to a septic infarct resulting from valvular endocarditis due to *Staphylococcus spp*

Pathogenesis of Infarction

- Often wedge-shaped

- Early – ill defined & hyperemic/congested
  - red – After 24 hrs. - coagulation necrosis - sharply defined within 72 hours

- Later – may become more pale

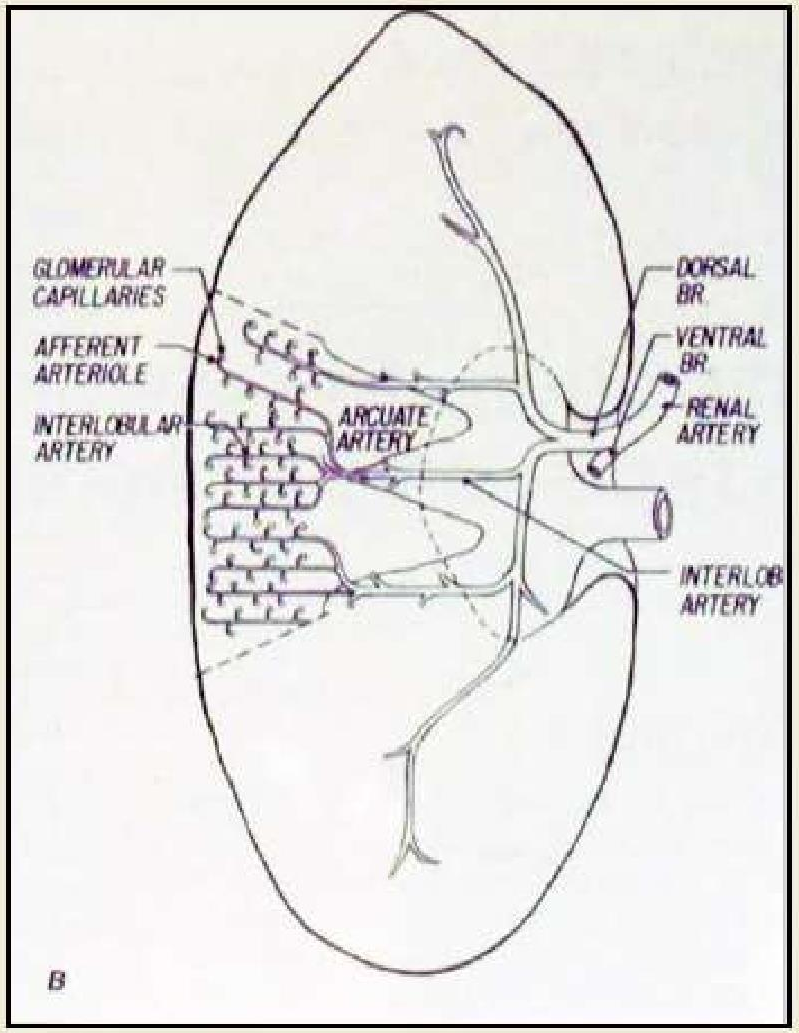


Factors that Influence Development of an Infarct

1. **Nature of the vascular supply**
   - collateral circulation : liver & lung- Not common
   - kidney + spleen -single arterial supply – More
2. **Rate of development of occlusion**
   - Slowly developing occlusions - ↓ % of infraction development
3. **Vulnerability to hypoxia**
   - Neurons : 3 to 4 minutes
   - Myocardial cells : 20 to 30 minutes
4. **O2 content of blood at time of infarct**
   - Abnormally low blood O2 due to anemia
   - Increases both the likelihood and extent of infarction.

Infarction – Histology

- ischemic necrosis of affected tissue



Infarction – Repair

- scar tissue replaces parenchyma
- fibrous tissue contracts forming depression / indent on surface

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SHOCK

- “haemodynamic disorder characterized by inadequate systemic blood circulation (hypoperfusion) due to a reduction either in the cardiac output, or in the effective circulatory blood volume”

SHOCK

Final common pathway for:

- Microbial sepsis
- Severe hemorrhage
- Extensive trauma or burns
- Myocardial damage
- Severe pulmonary embolism

- Results in impaired tissue perfusion and cellular hypoxia
- Brain and heart are organs most susceptible to ischemic damage

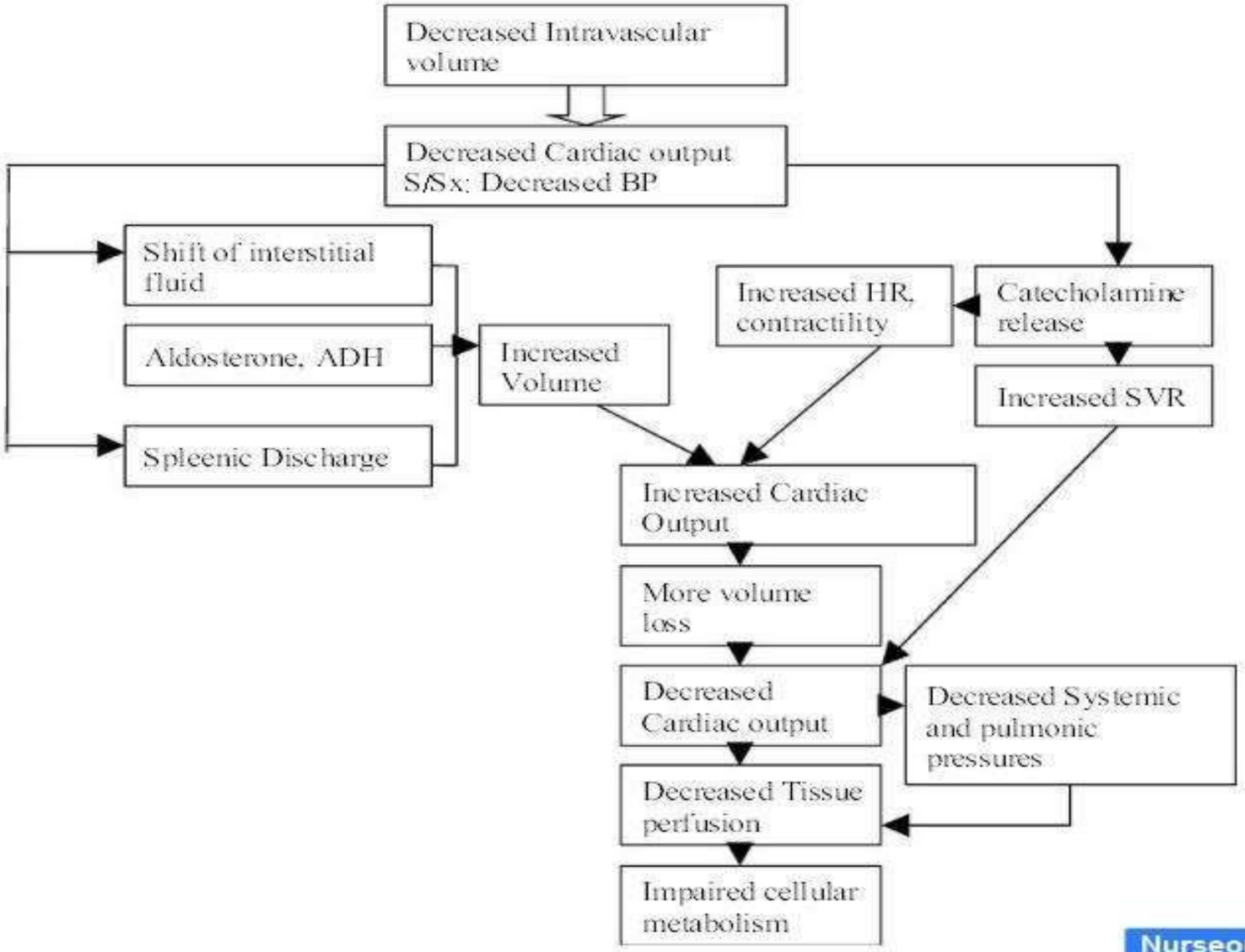
SHOCK

- On basis of clinical conditions is best divided into four pathophysiological types:
  - **(1) Hypovolemic shock**
  - **(2) Cardiogenic or cardiac shock**
  - **(3) Blood Maldistribution (Vasogenic Shock)**
    - **a. Anaphylactic Shock - release of vasoactive amines**
    - **b. Neurogenic Shock**
    - **c. Septic Shock - release of chemical**

Hypovolemic shock

- Decreased circulating blood volume due to
  - Blood loss from hemorrhage
  - Fluid loss (eg vomiting, diarrhea, burns)

Hypovolemic shock – Pathogenesis



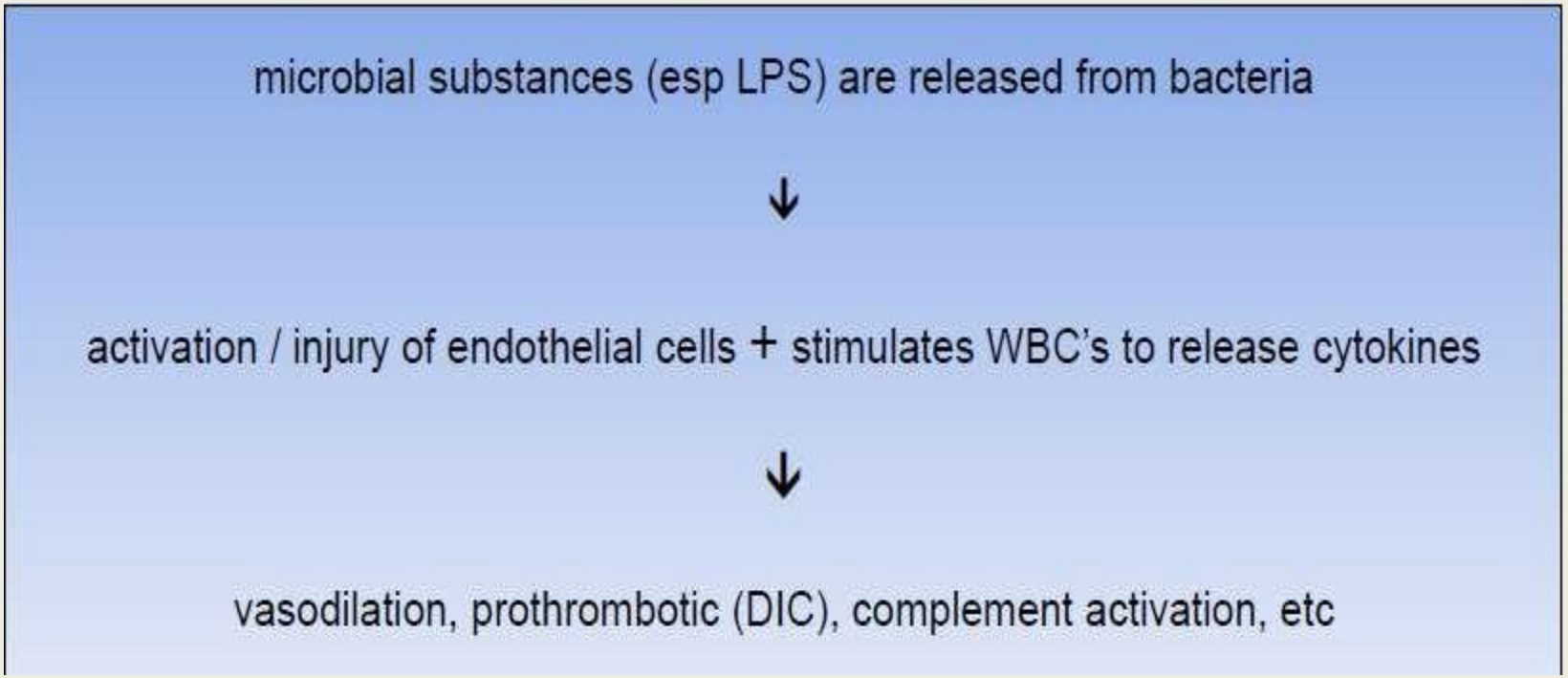
Cardiac shock

- Failure of heart to adequately pump blood may be due to
  - Myocardial infarction
  - Arrhythmias (eg ventricular tachycardia)
  - Cardiomyopathy
  - Obstruction of blood flow

Similar Pathogenesis like hypovolemic shock

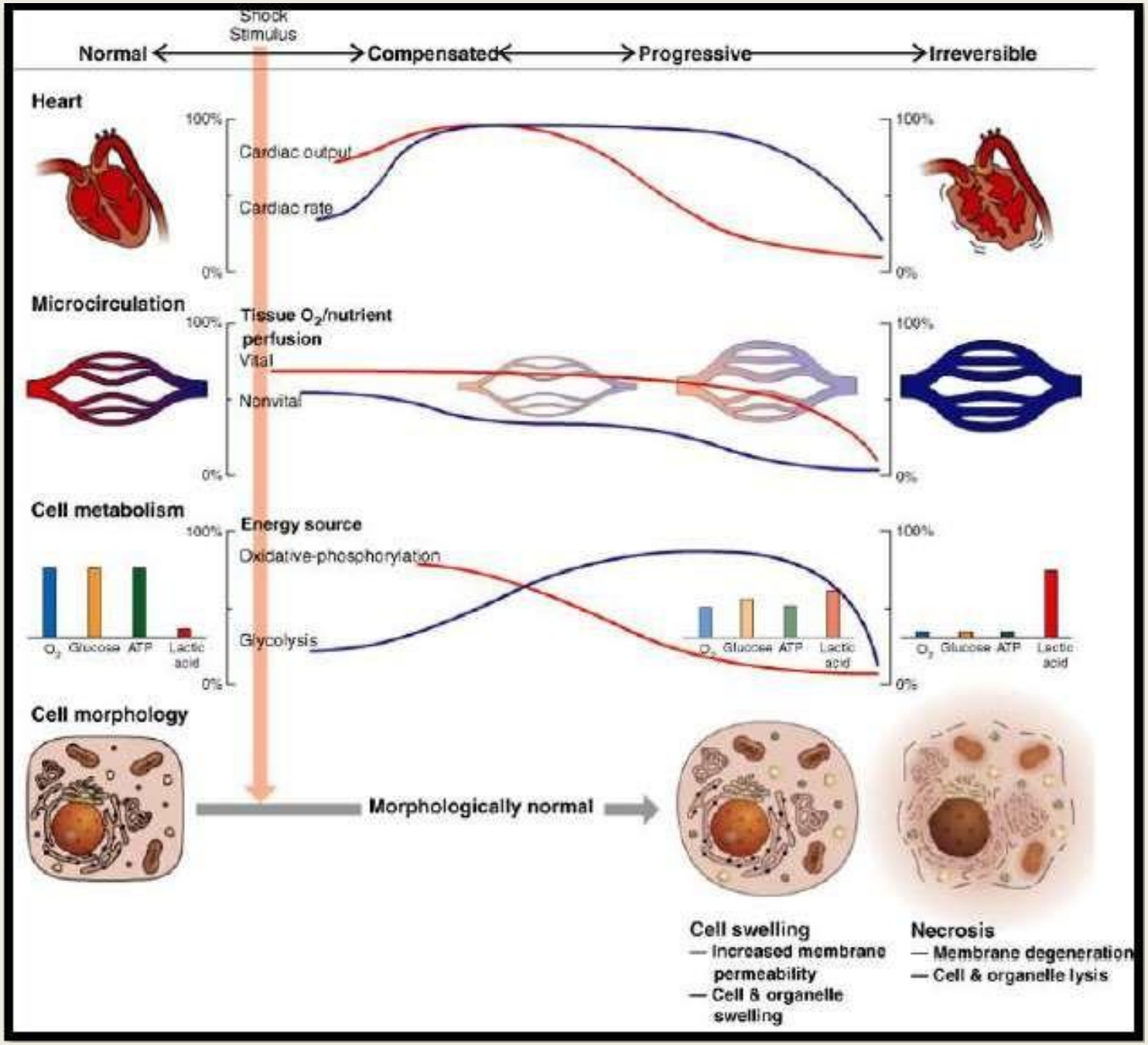
Septic or endotoxic shock

Pathogenesis



Stages of Shock

1. Initial Nonprogressive shock
2. Progressive stage
3. Irreversible stage


Shock Stimulus
Normal
Compensated
Progressive
Irreversible
Heart
100%
Cardiac output
Cardiac rate
0%
Microcirculation
Tissue O_2/nutrient perfusion
Vital
Nonvital
Cell metabolism
Energy source
Oxidative-phosphorylation
Glycolysis
O_2 Glucose ATP Lactic acid
Cell morphology
Morphologically normal
Cell swelling
— Increased membrane permeability
— Cell & organelle swelling
Necrosis
— Membrane degeneration
— Cell & organelle lysis

Lesions of Shock

- Pulmonary congestion & edema (cattle and horses)
- Hepatic congestion (dog)
- Kidneys - acute tubular necrosis
- Heart - hemorrhage and necrosis
- Blood vessels - endothelial damage (thrombosis / DIC)
- Brain - neuronal cell death
- Adrenal glands - hemorrhage and necrosis
- GI tract - congestion and necrosis