MJF COLLEGE OF VETERINARY AND ANIMAL SCIENCE, CHOMU, JAIPUR

DEPARTMENT OF VETERINARY PATHOLOGY

EDEMA

HYPEREMIA AND CONGESTION

HEMORRHAGE

HEMOSTASIS

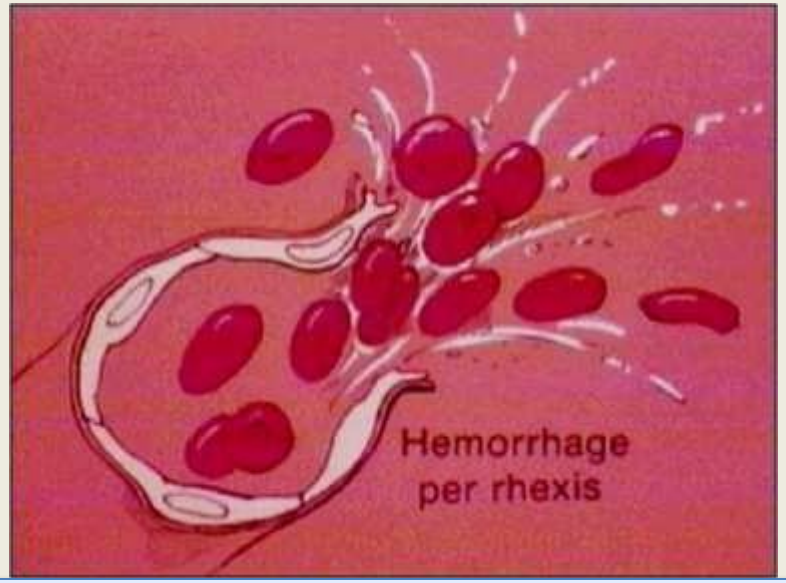
THROMBOSIS

EMBOLISM

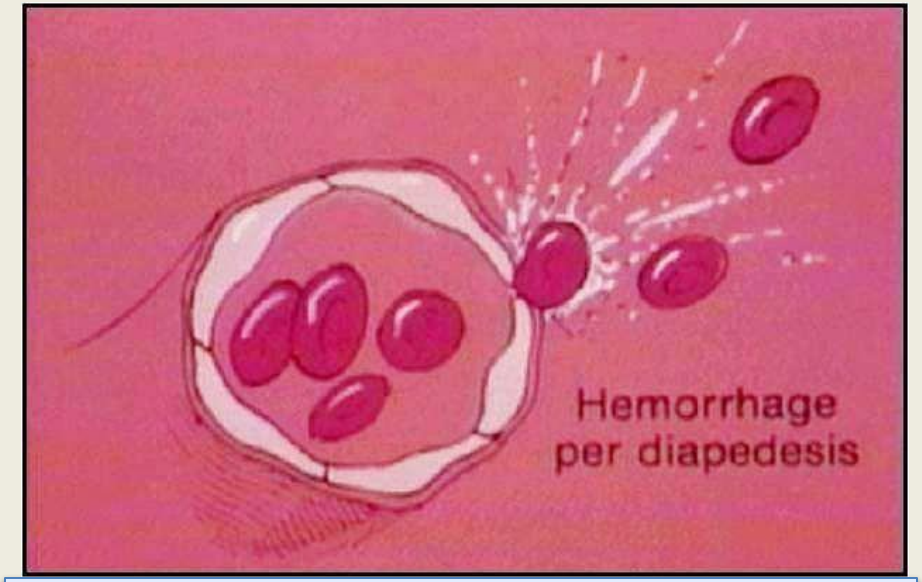
INFRACTION

HEMORRHAGE

- *Definition: Extravasation of blood from* vessels
- Internal or External



hemorrhage from a tear in blood vessel or heart
•moderate to marked flow of blood out of cardiovascular system



Bleeding from a small defect
- RBC's passing through vessel wall in hyperemia of inflammation

HEMORRHAGE

Etiology:

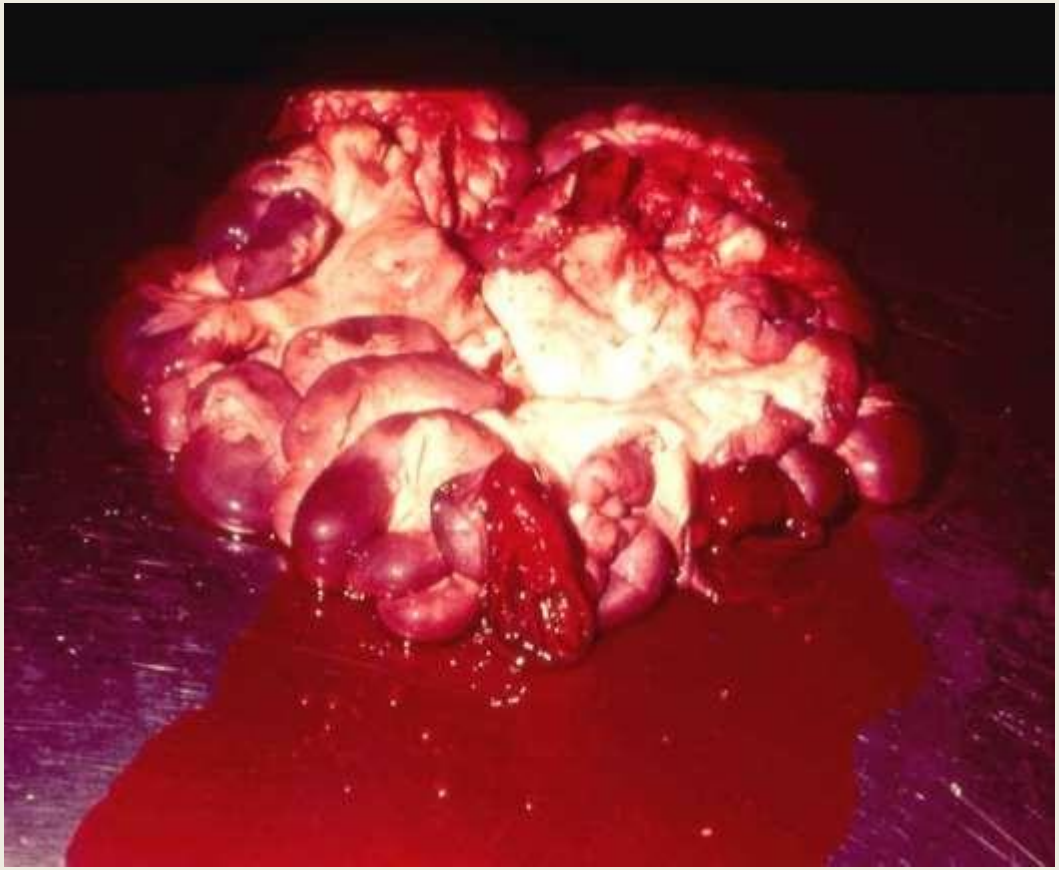
- Physiological : parturition
- Trauma
- Infectious agent: Bacteria, Virus, Parasite
- Toxic chemical
- Haemorrhagic diatheses
  - Group of clinical disorders characterized by increased bleeding tendency
  - Increased vascular fragility
    - vitamin C deficiency or scurvy
  - Reduced platelet number
  - Defective platelet function
  - Abnormalities in clotting factors
    - Hemophilia A – factor VIII deficiency

Hemorrhage Significance

Site
- CNS
- heart

Rate
- How fast

Volume of Blood Loss
- Hemorrhagi

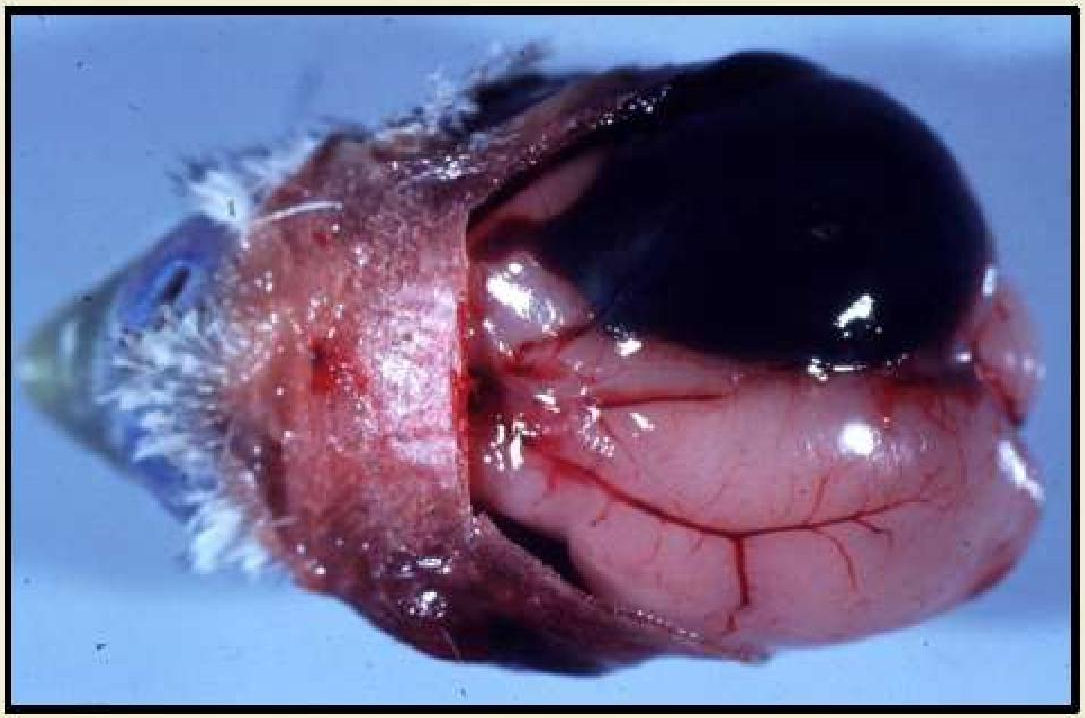


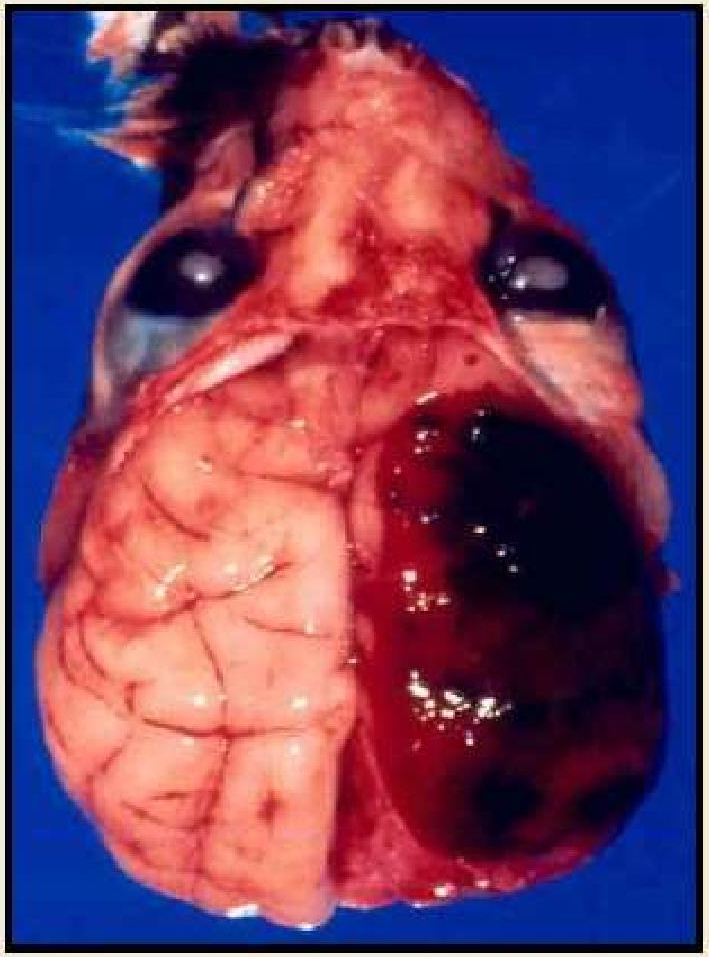
Bleeding into the intestinal lumen in a hemorrhagic enteritis / enteropathy; which can be caused by bacteria (eg *Salmonella spp), viruses (eg parvovirus), toxins (eg arsenic), etc*

Hemorrhage

Subdural (or Epidural) Hematoma

- blood accumulation beneath (or above) the dura
- compresses brain

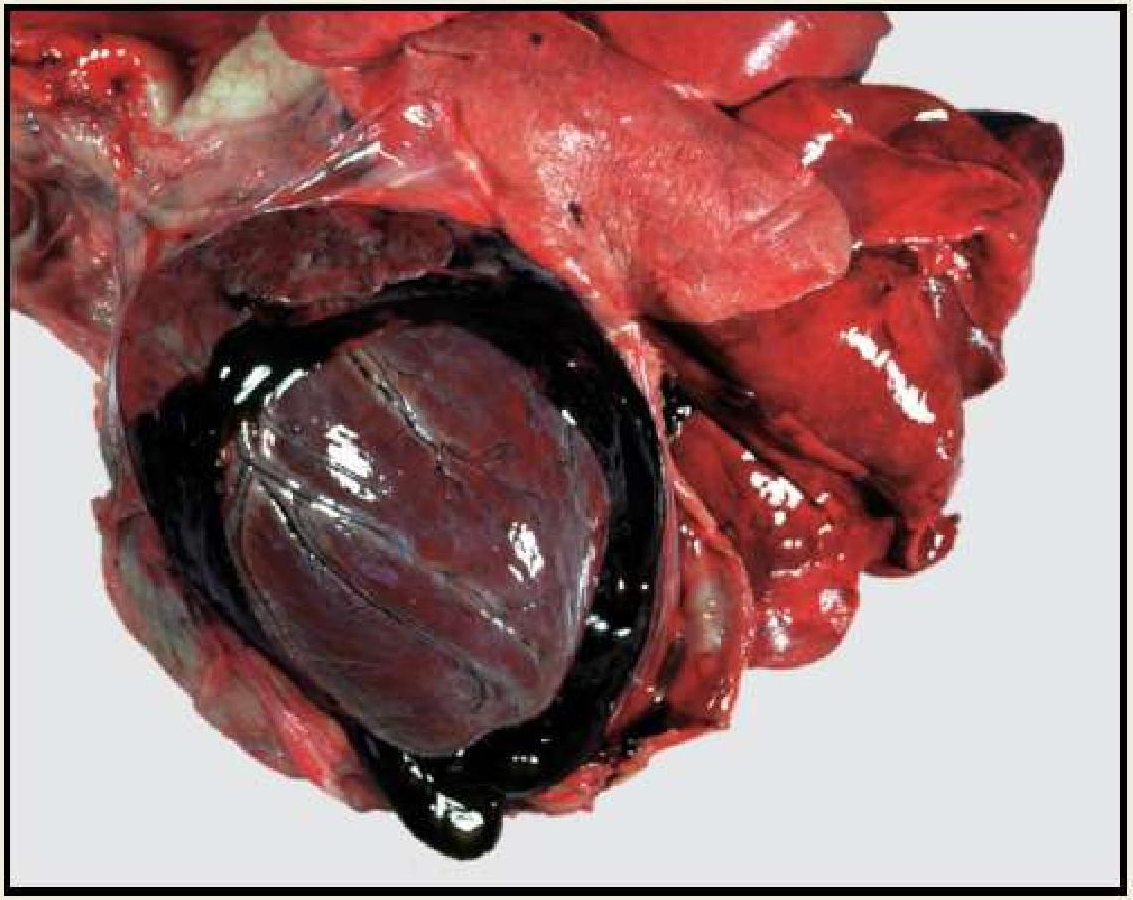




Hemorrhage

Cardiac Tamponade

- heart failure due to massive accumulation of fluid in pericardial sac
- compresses atrium and ventricles (restriction of cardiac filling)

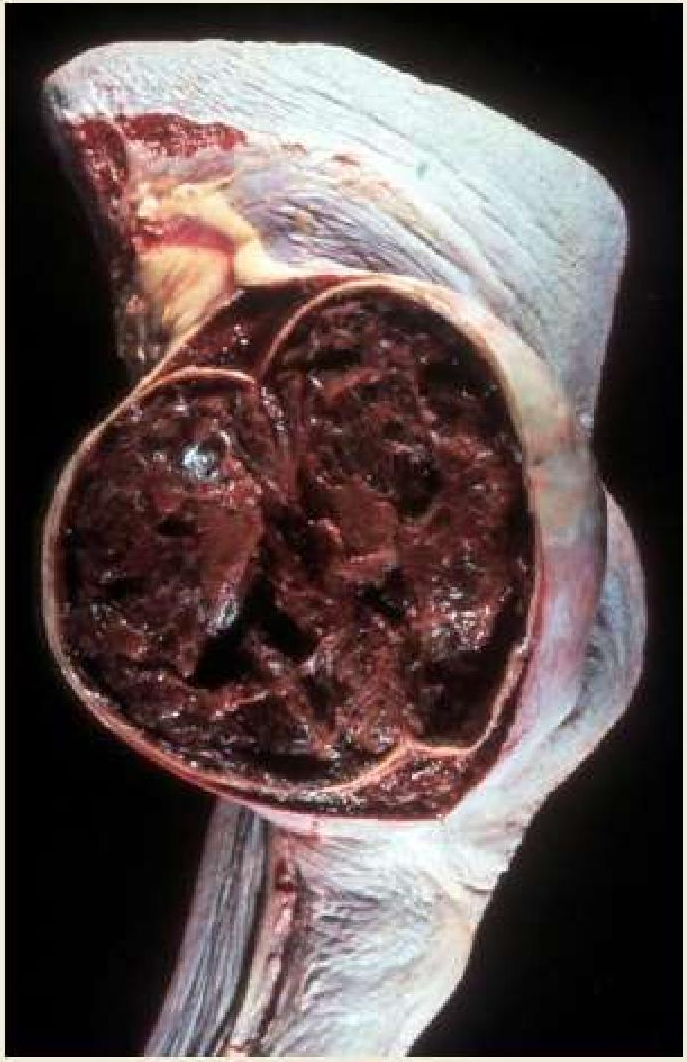


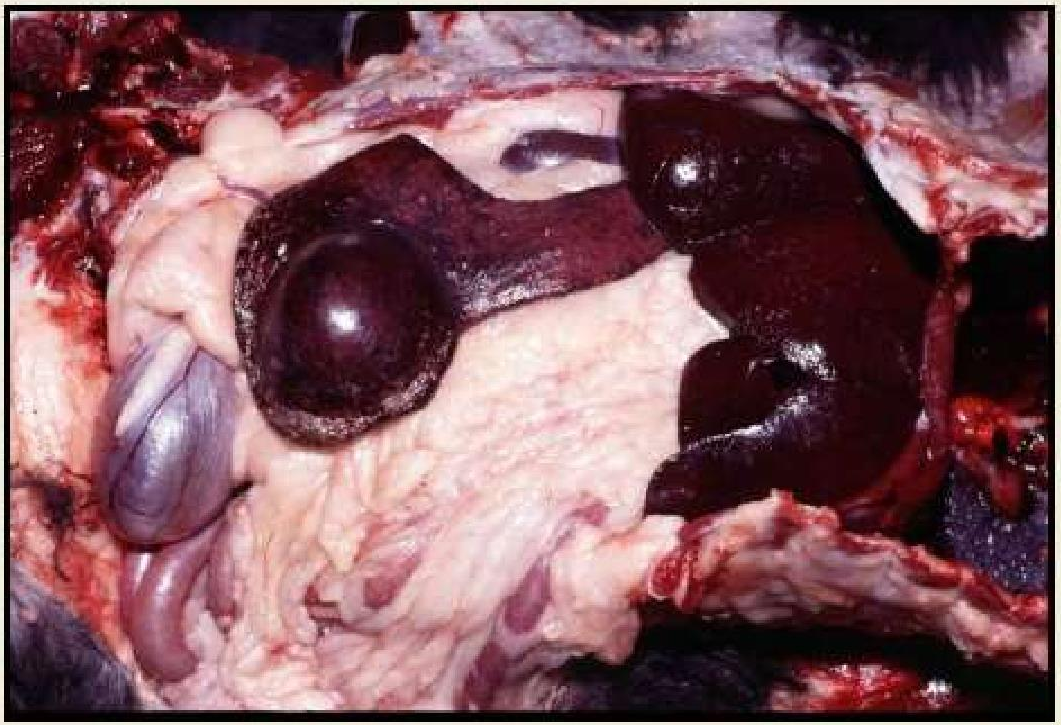
Hemopericardium

- blood present within the pericardial sac.

Hematoma

- extravascular, 3-D blood clot, enclosed within a tissue



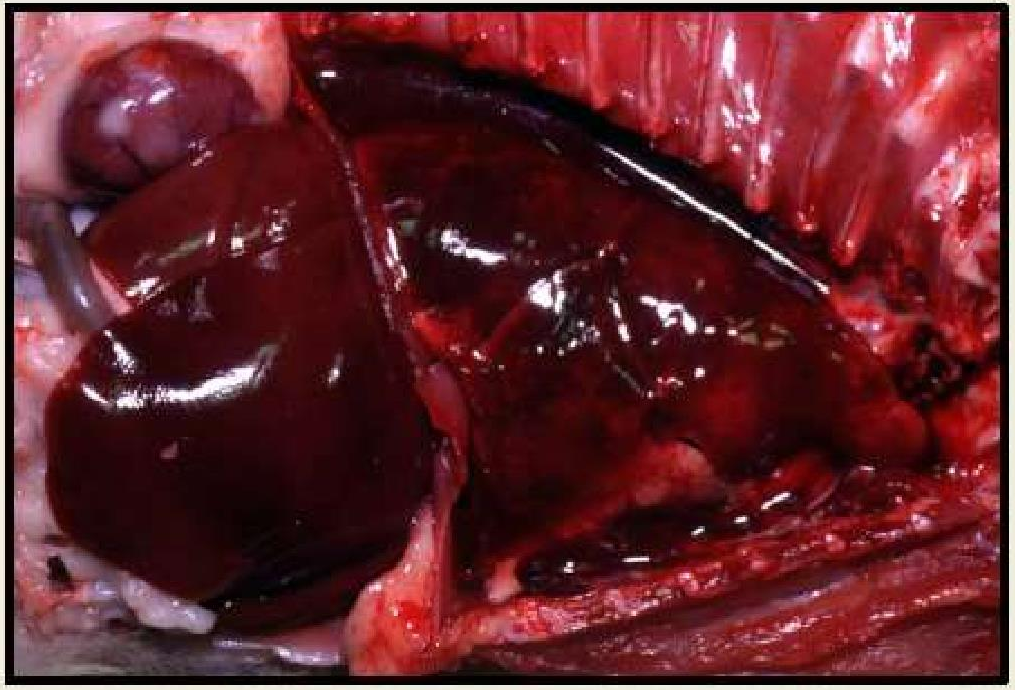


Splenic hematoma, dog

Fig. 2-22 (Zachary) Organizing hematoma, spleen, horse. Trauma to the spleen has caused damage to the splenic red pulp and its vessels, resulting in bleeding into the splenic parenchyma, forming a hematoma. Note that this hematoma is not acute but is several days old, because the blood clot is being degraded. The hematoma is contained by the splenic capsule.

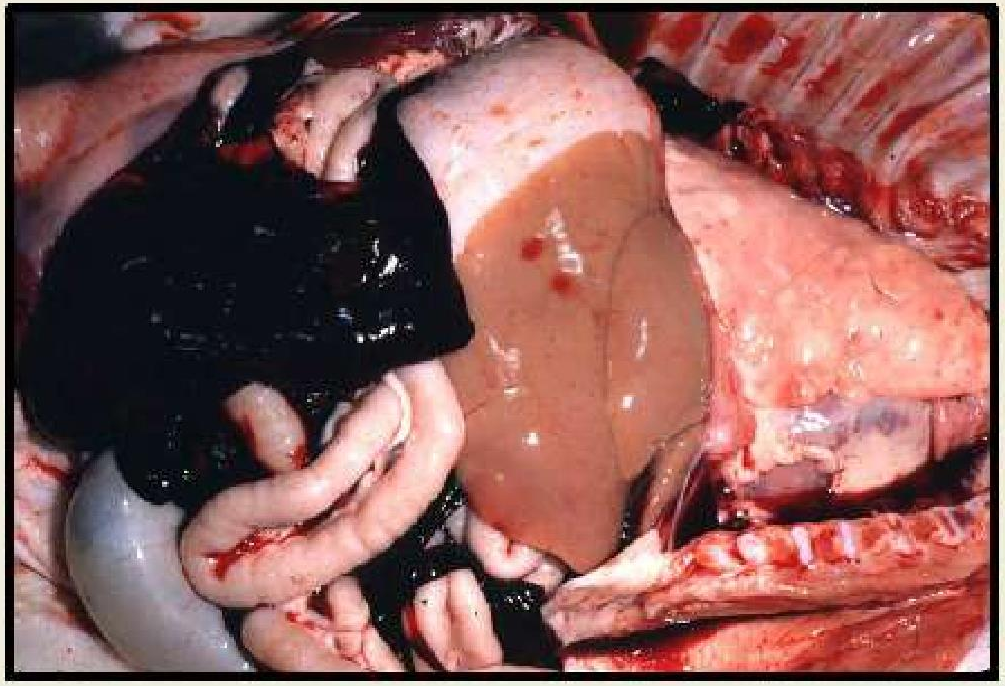
Hemothorax

- blood in the pleural cavity



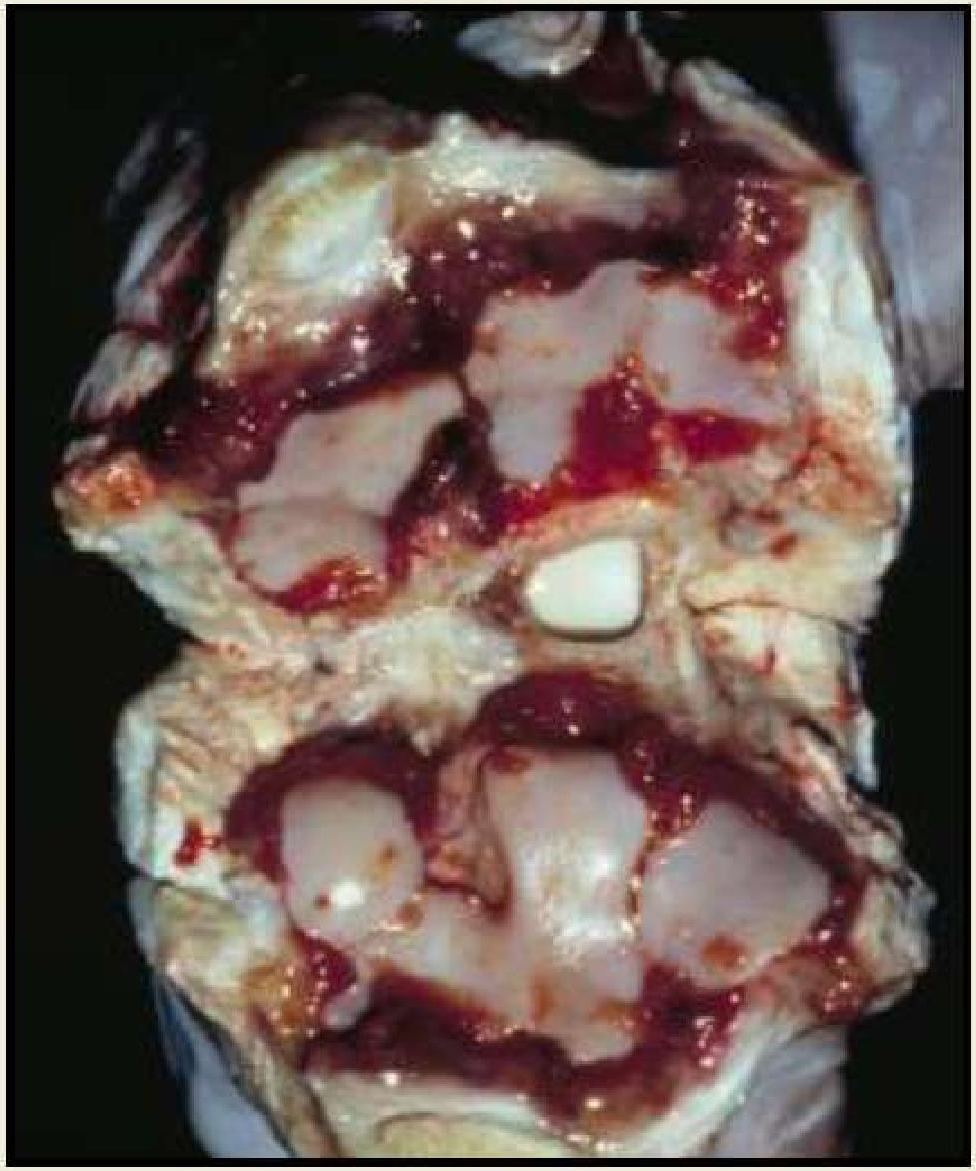
Hemoperitoneum

- blood in the peritoneal cavity



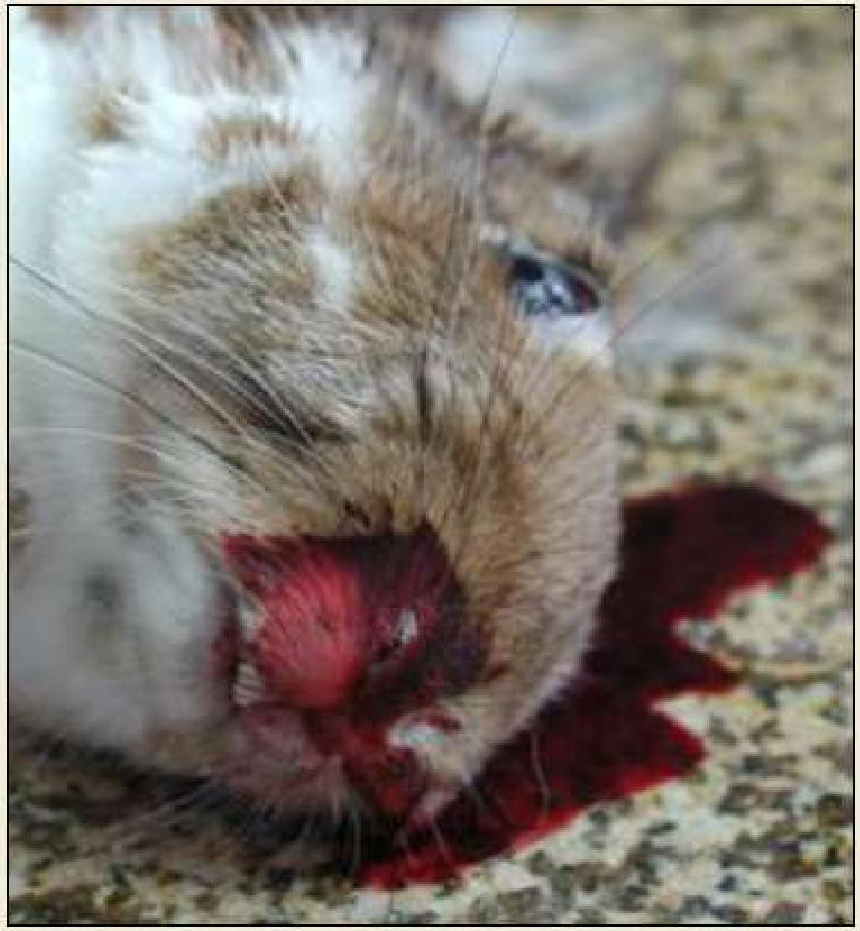
Hemarthrosis

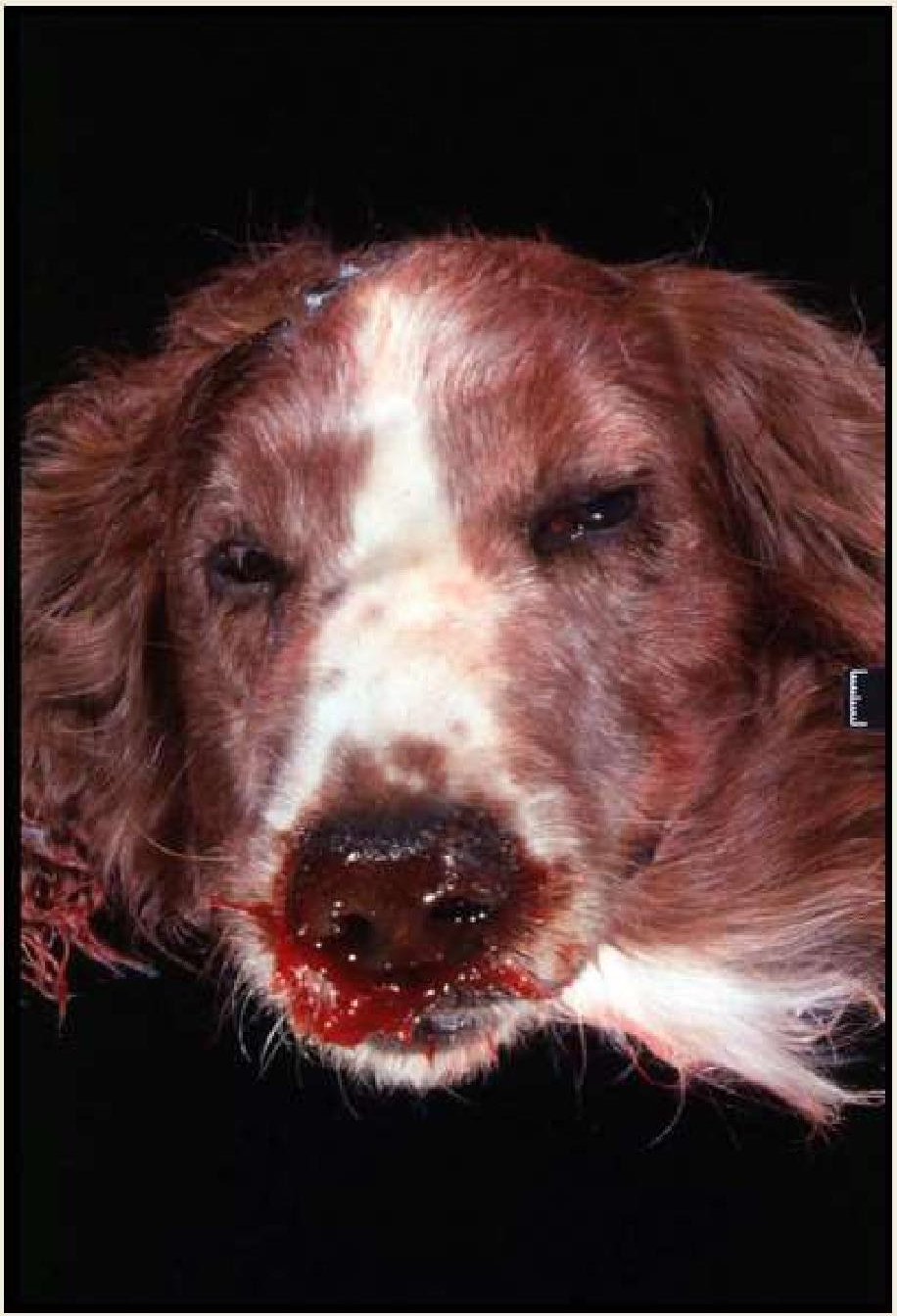
- blood present in joint spaces



Epistaxis

- bleeding from the nose





Hemoptysis

- coughing of blood from the lungs or airways

Haematemesis

- Blood in vomit

Metrorrhagia

- Uterine bleeding

Haematuri

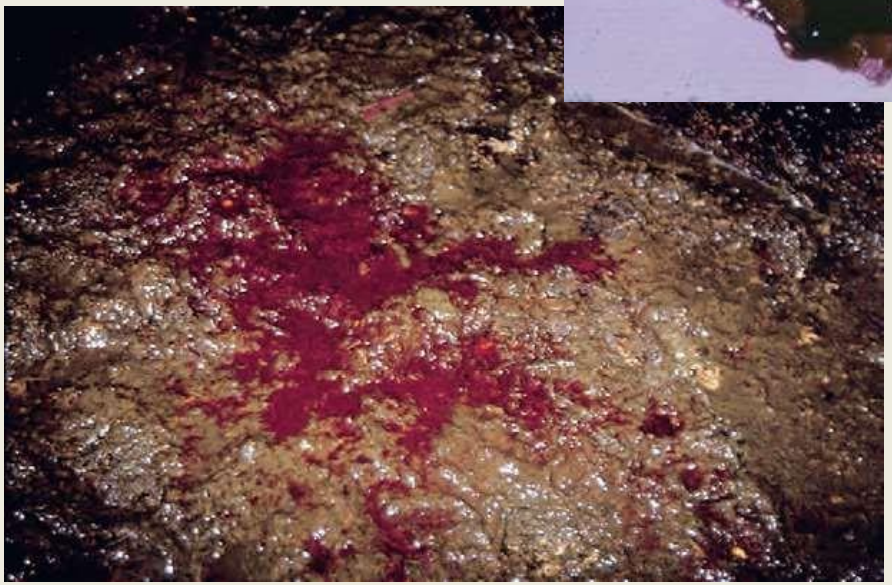
- Blood in

Melen

- Blood in stools/faeces
- Not fresh blood /digested dark colour)

Hematochez

passage of fresh blood through the anus, usually in or with stools



Haematocele

- bleeding in tunica vaginalis - serous lining of the testicle

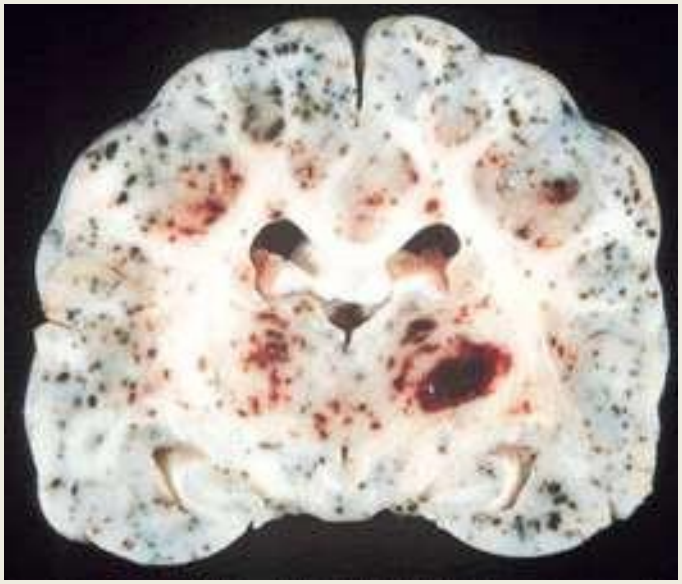
Haemosalpinx

- bleeding in oviducts

Apople

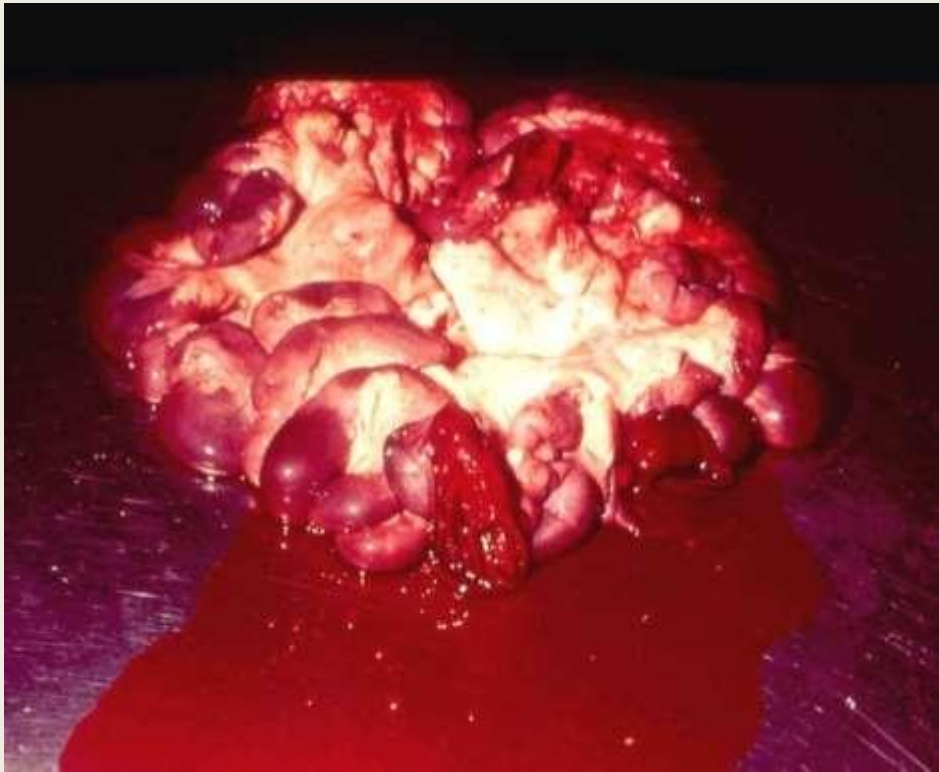
x

- Haemorrhage in organ like brain or pituitary



Enterorrhag

intestinal haemorrhage

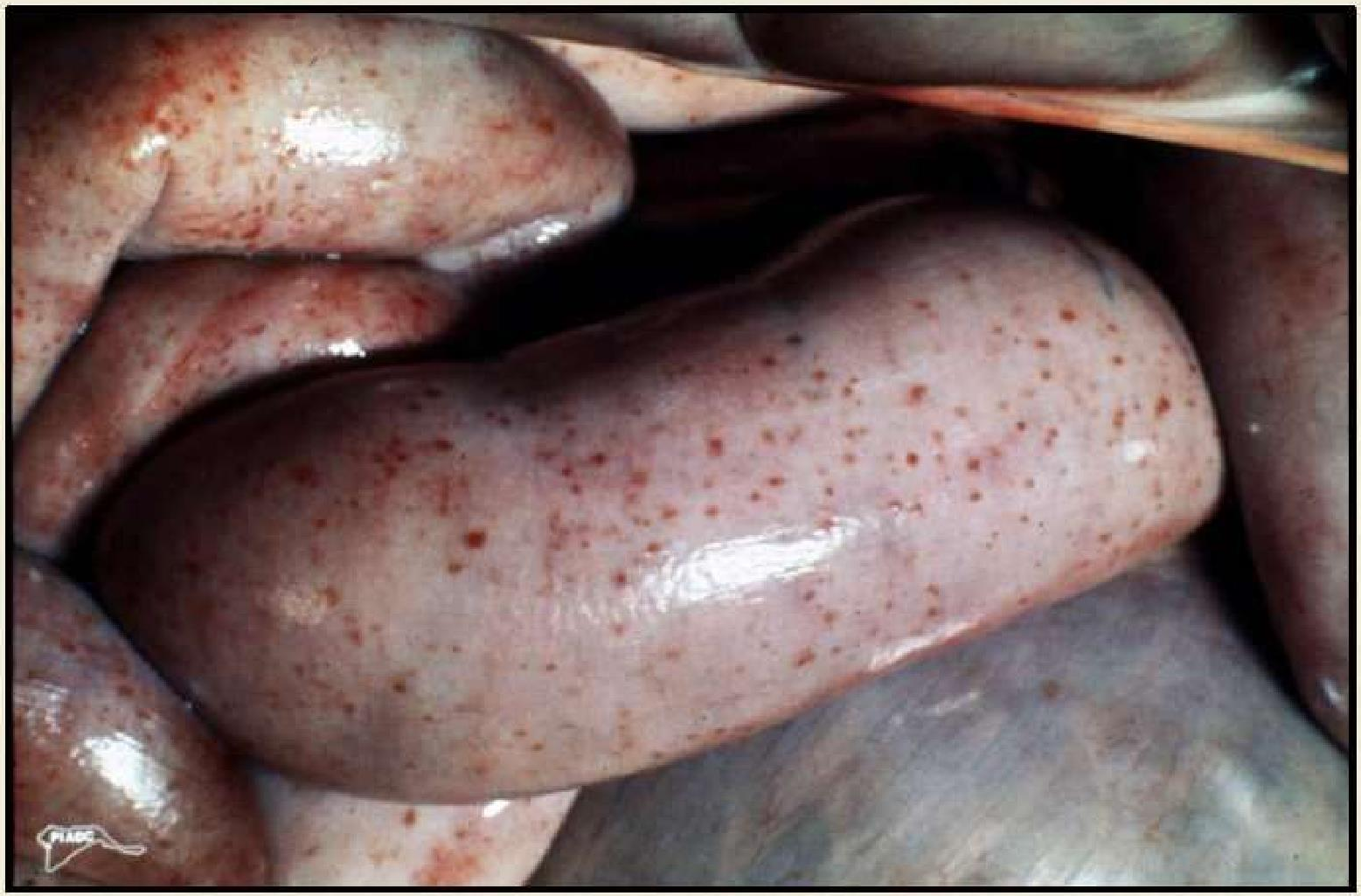


Linear h

- Haemorrhages that appear as lines

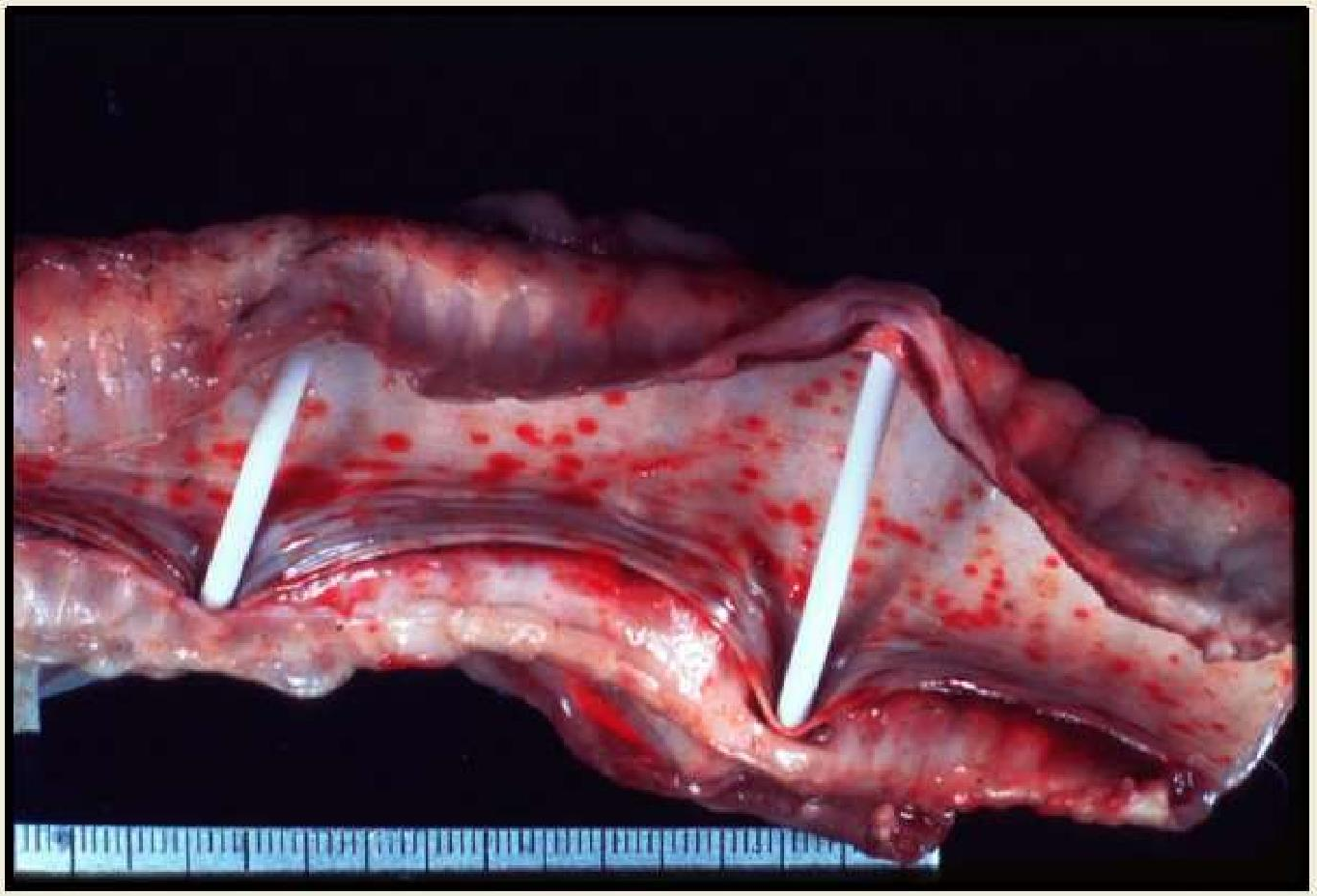
Petechia (pl. petechiae):

- minute, up to 1-2 mm, hemorrhages, esp skin / mucosa / serosa.



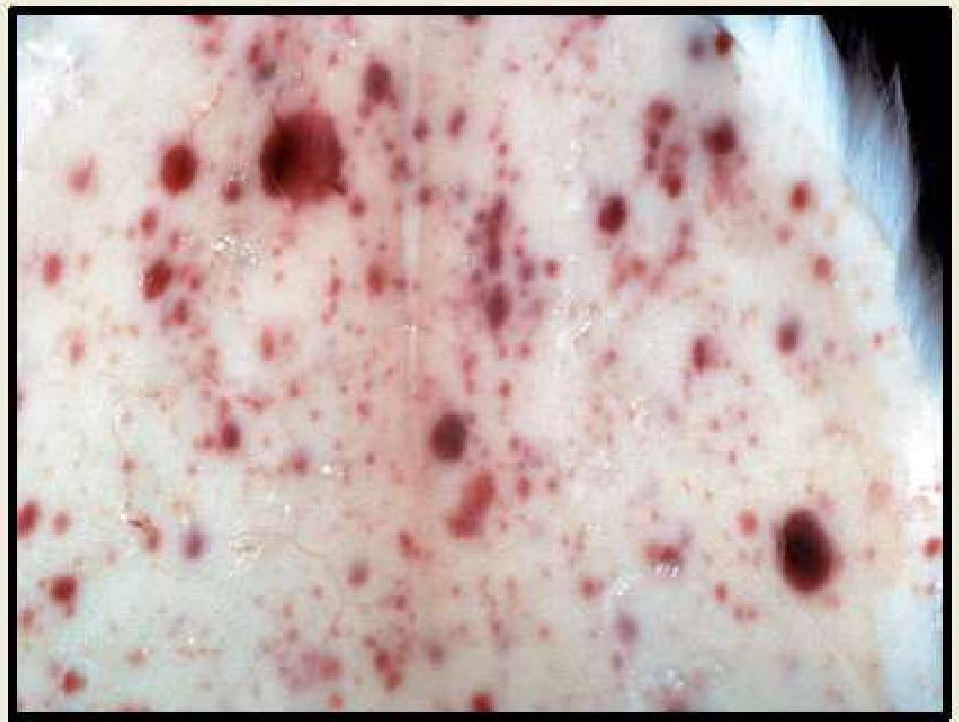
Purpura

- hemorrhage 3 mm – 1cm, often scattered on body surfaces (skin, mucosa)
- often with diseases which cause petechiae; vascular inflammation / damage



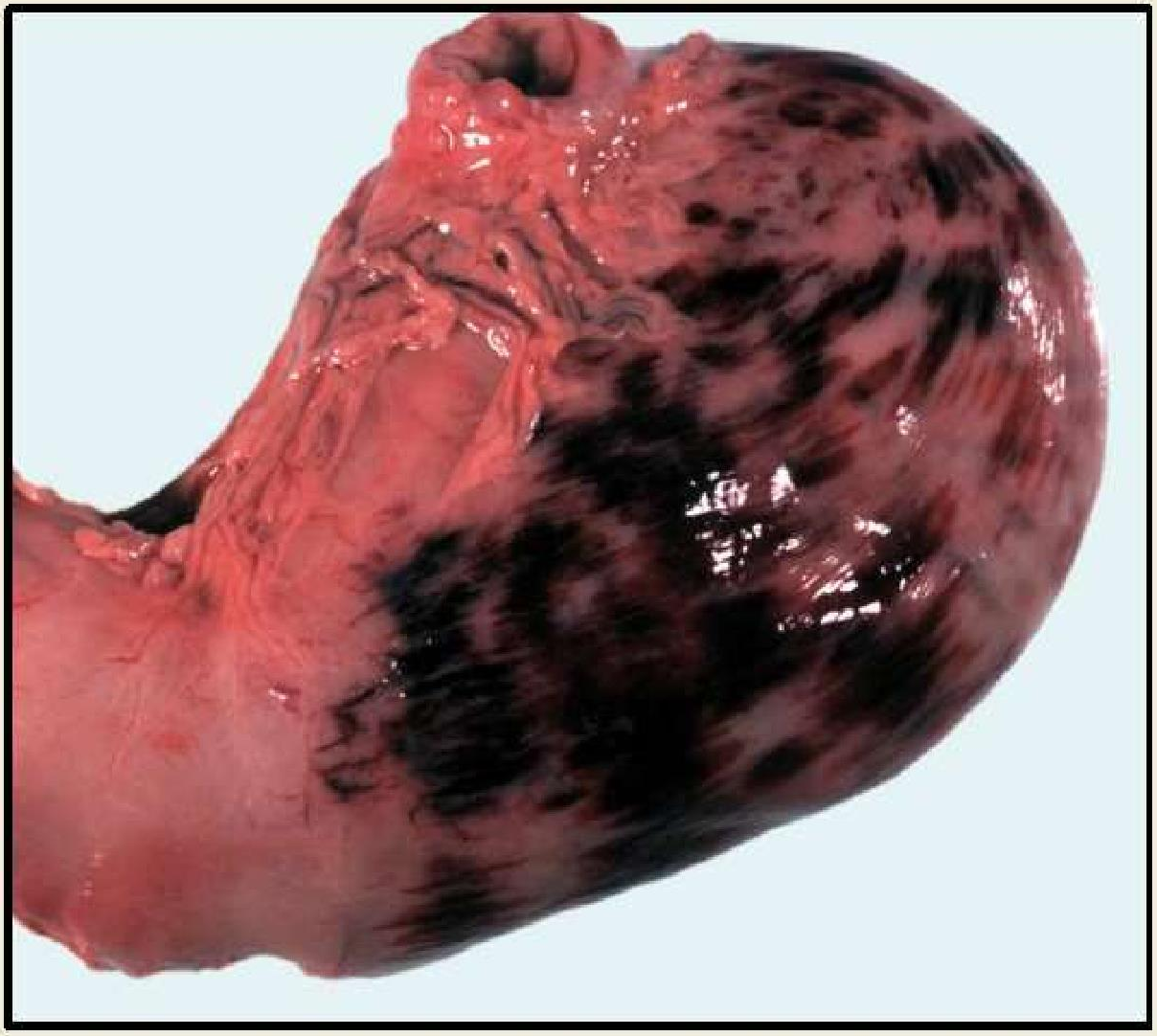
Ecchymosis (pl. ecchymoses)

- hemorrhage larger than petechiae / purpura (> 1 cm)
- often blotchy or irregular



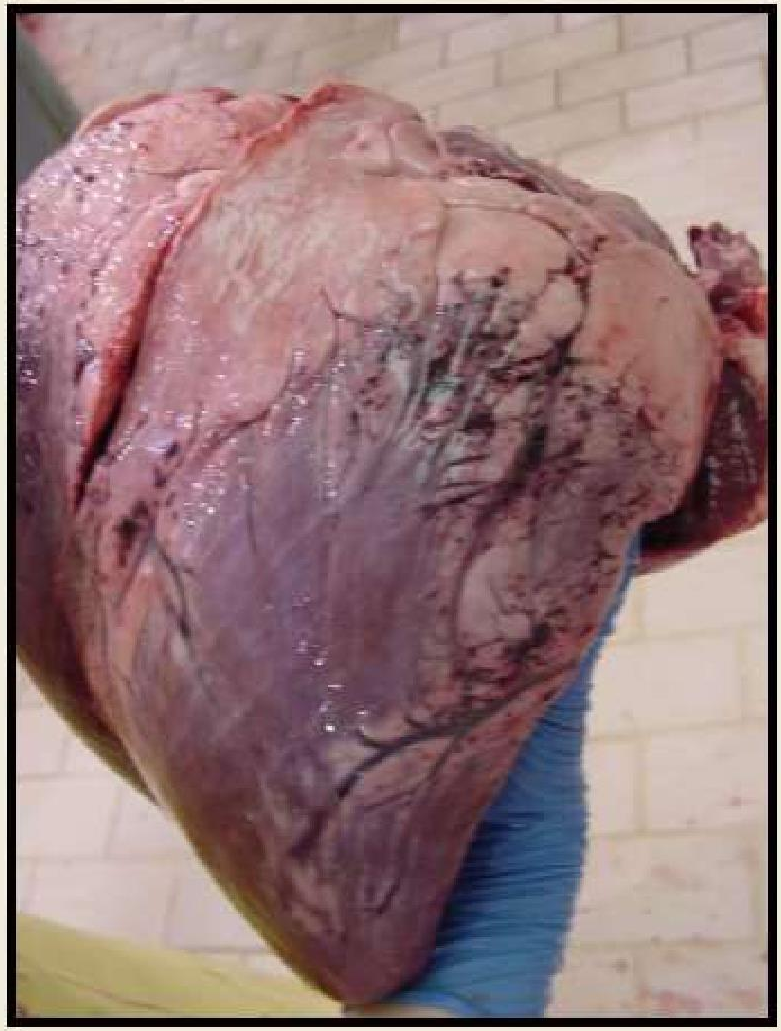
Suffusive Hemorrhage

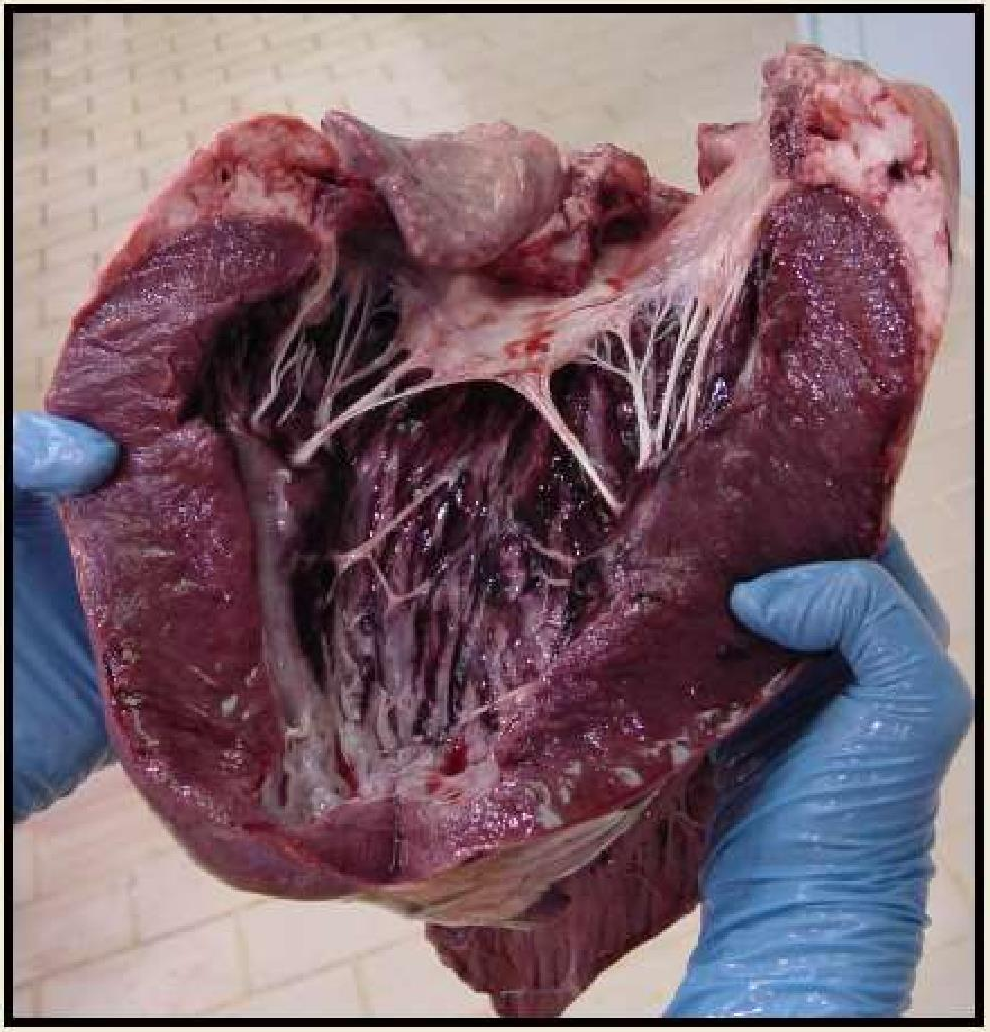
- areas of hemorrhage larger than ecchymoses and contiguous.



Agonal hemorrhages

- refer to petechiae and ecchymoses that are associated with the death struggle (terminal hypoxia).





Resolution of Hemorrhage

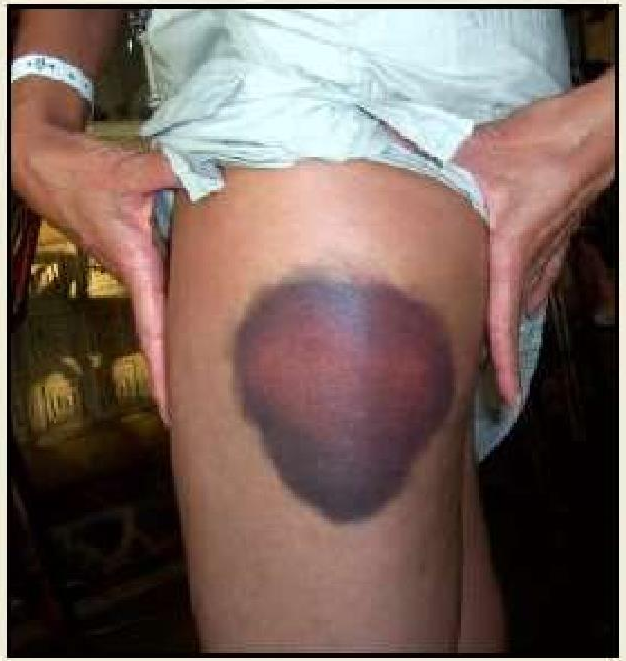
Dependent on amount

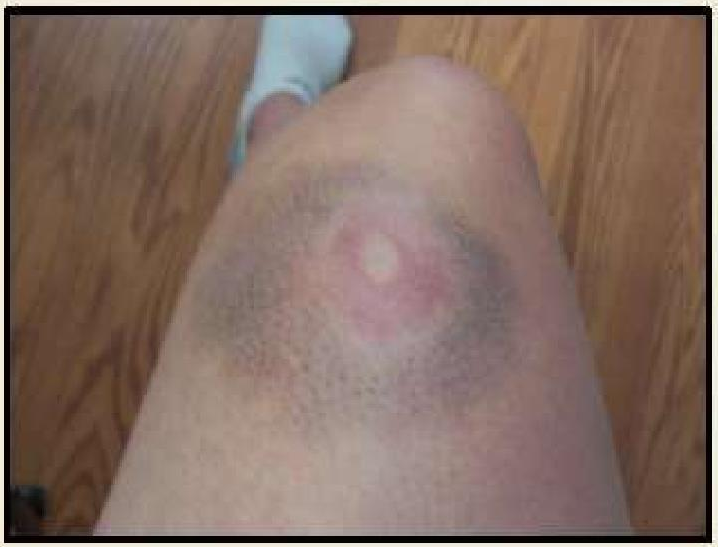
- small amount can be reabsorbed
- larger areas require breakdown & removal of rbc's

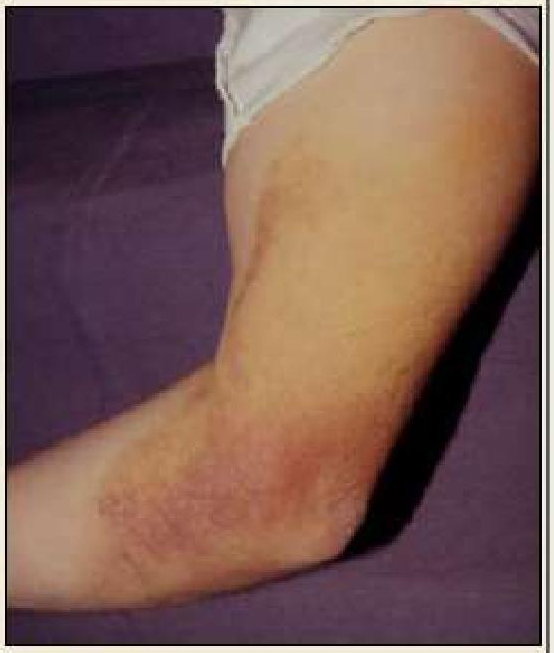
Resolution of Hemorrhage

Hemoglobin → Bilirubin → Hemosiderin

Red - Blue | Blue-Green | Yellow-Brown



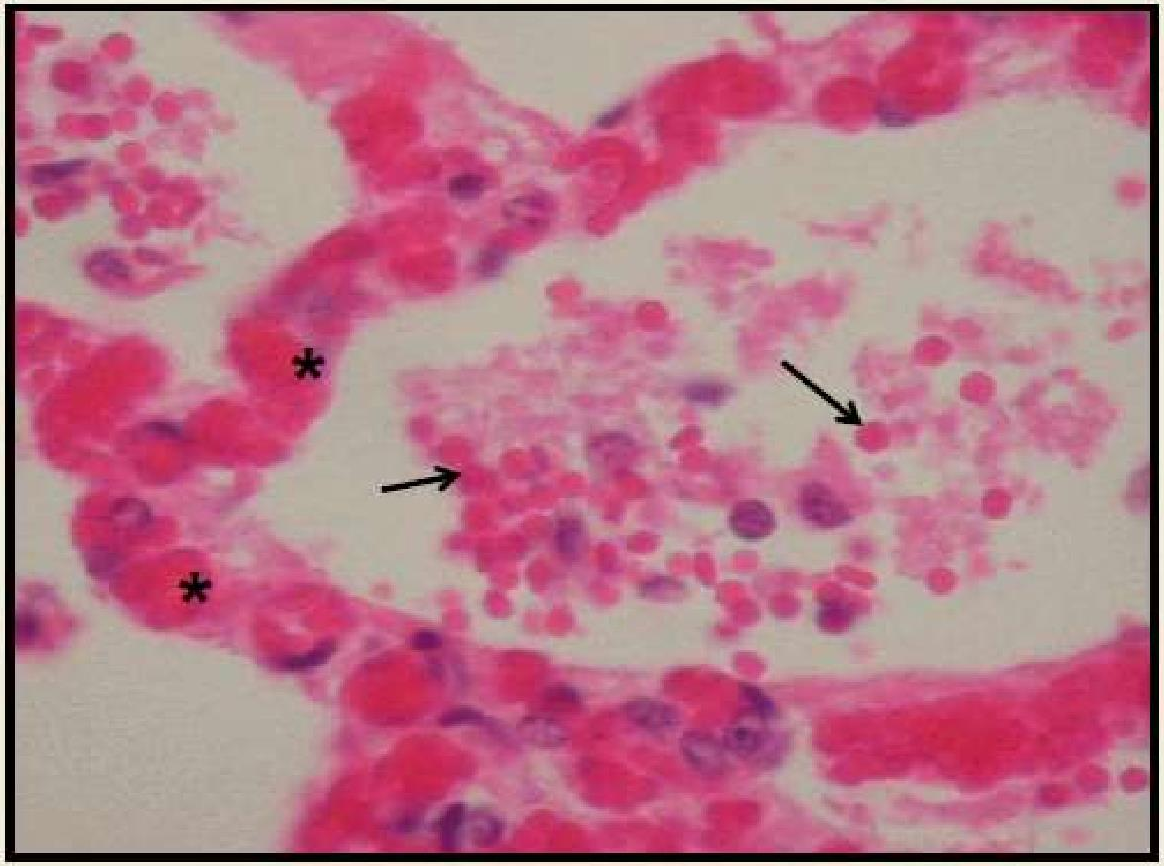




Hemorrhage Vs Hyperemia/Congestion

Congestion / Hyperemia – rbc's are within the blood vessels (asterisks)

Hemorrhage – rbc's are outside vessels (arrows)



EDEMA

HYPEREMIA AND CONGESTION

HEMORRHAGE

HEMOSTASIS & THROMBOSIS

EMBOLISM

INFRACTION

SHOCK

Hemostasis

HEMOSTASIS = refers to the arrest of bleeding

Well-regulated process to:

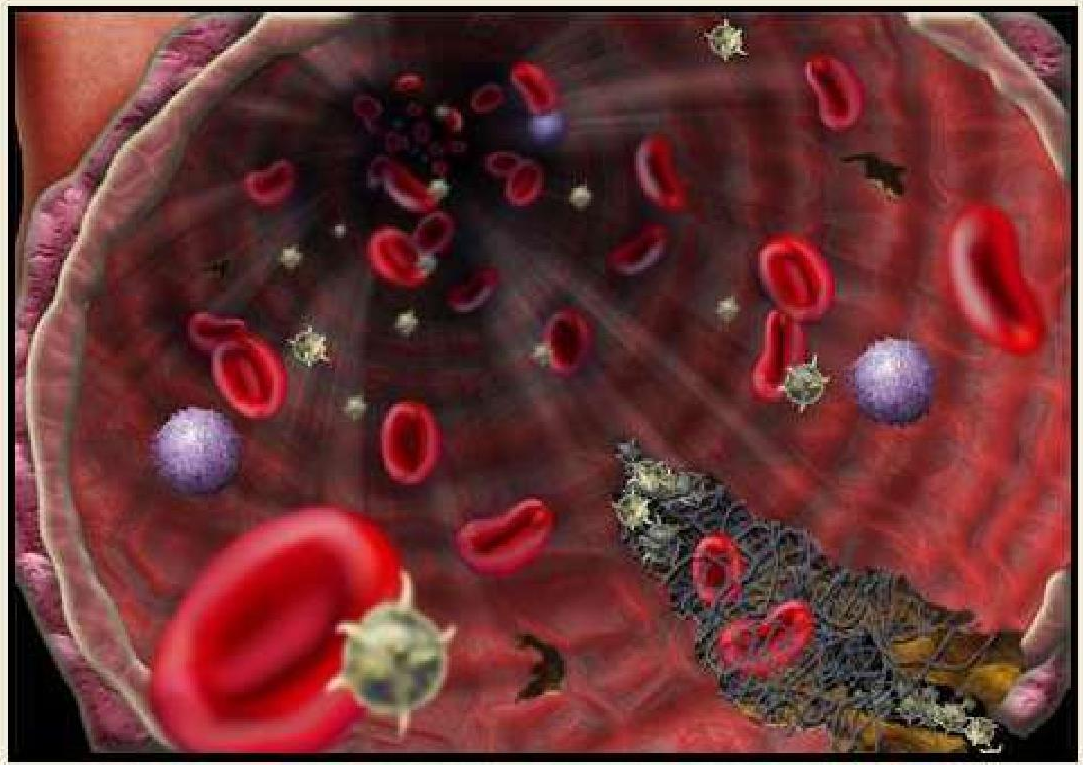
- Keeps blood fluid (clot free) within a normal vessel
- Rapid clot formation (hemostatic plug) occurs when vessel injured
- A hemostatic clot is normal in cases of vessel

Thrombosis: the formation of blood clot (thrombus) within intact vessels

Hemostasis

Three General Components

- Vascular wall - endothelium
- Platelets
- Coagulation Cascade

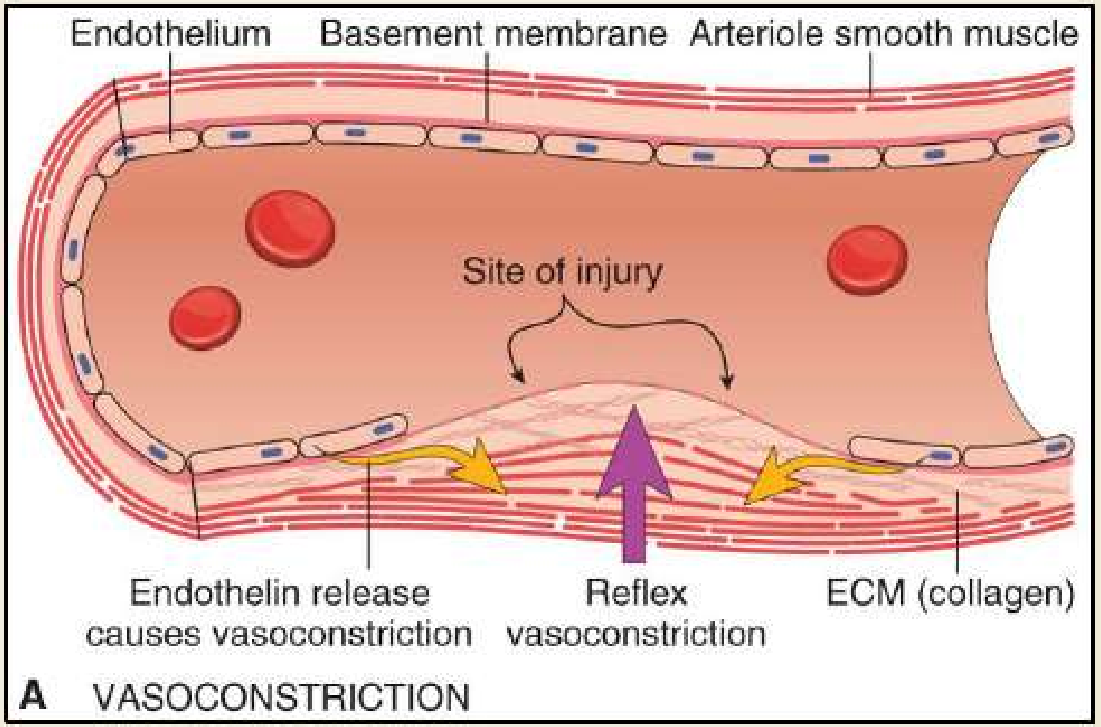


Normal Hemostasis

Sequence of Events

1. Arteriolar Vasoconstriction

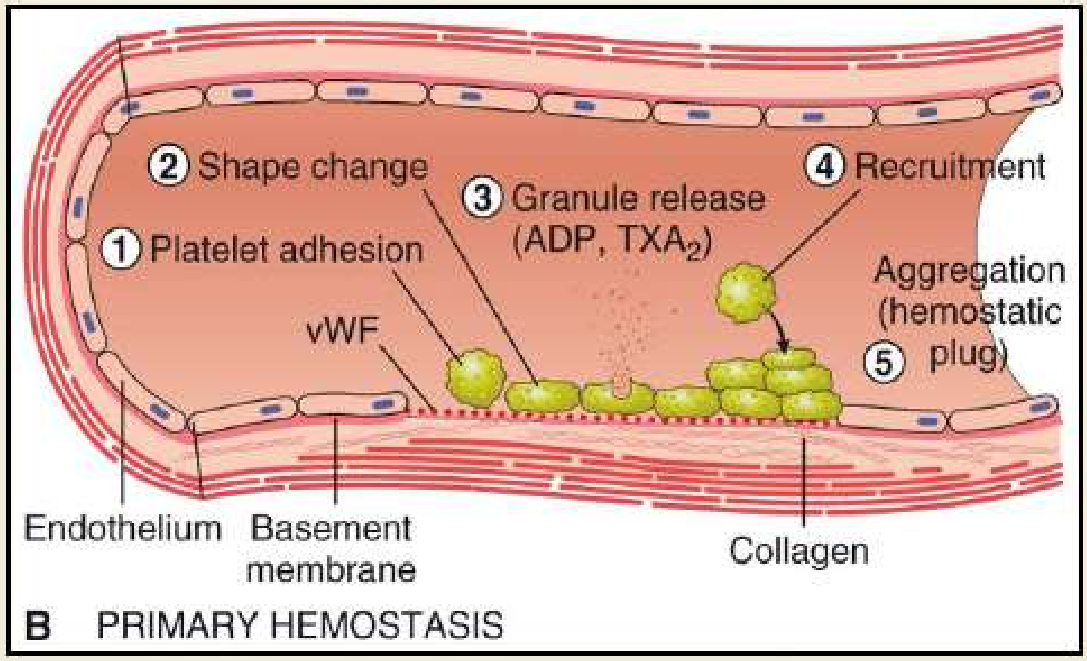
- reflex neurogenic mechanism
- local secretion of endothelin



Normal Hemostasis

Sequence of Events

1. Arteriolar Vasoconstriction
2. **Primary Hemostasis - PLATELET**
   - Platelet respond to exposure of subendothelial ECM by:
     - Adhesion
     - Shape Change
     - Granule Release
     - Recruitment
   - Platelet aggregation (1º hemostatic plug)

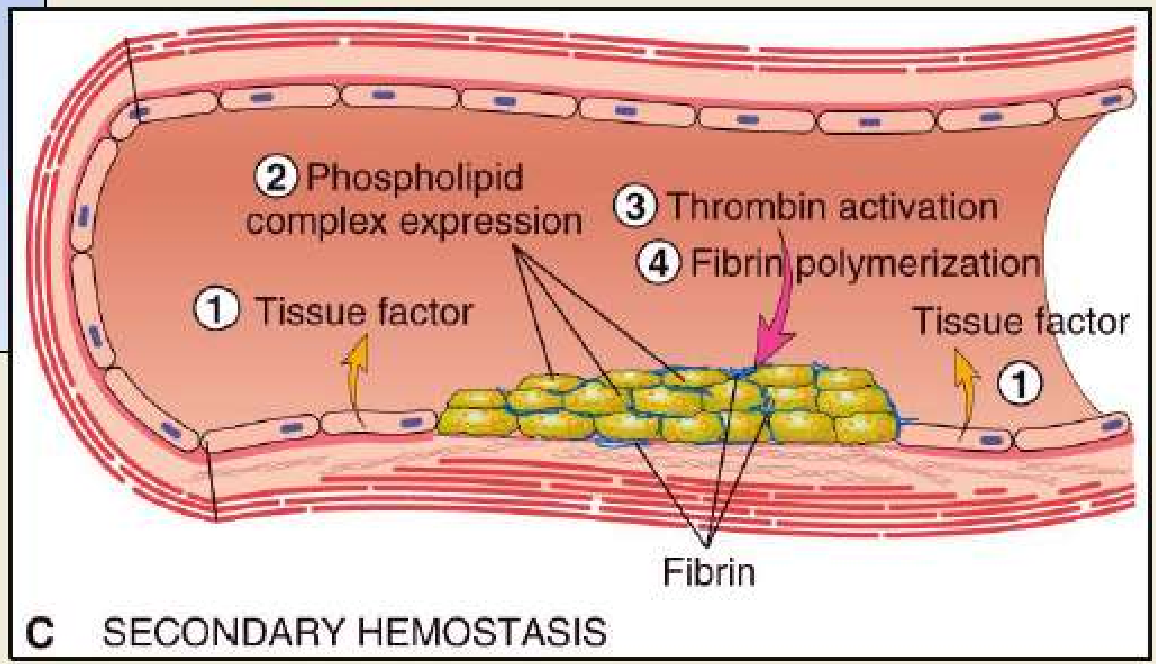


B PRIMARY HEMOSTASIS

Normal Hemostasis

Sequence of Events

1. Arteriolar Vasoconstriction
2. Primary Hemostasis - PLATELET
3. **Secondary Hemostasis - COAGULATION**
   - Tissue factor release
   - Phospholipid complex expression
   - Thrombin activation
   - Fibrin polymerization (2º hemostatic plug)

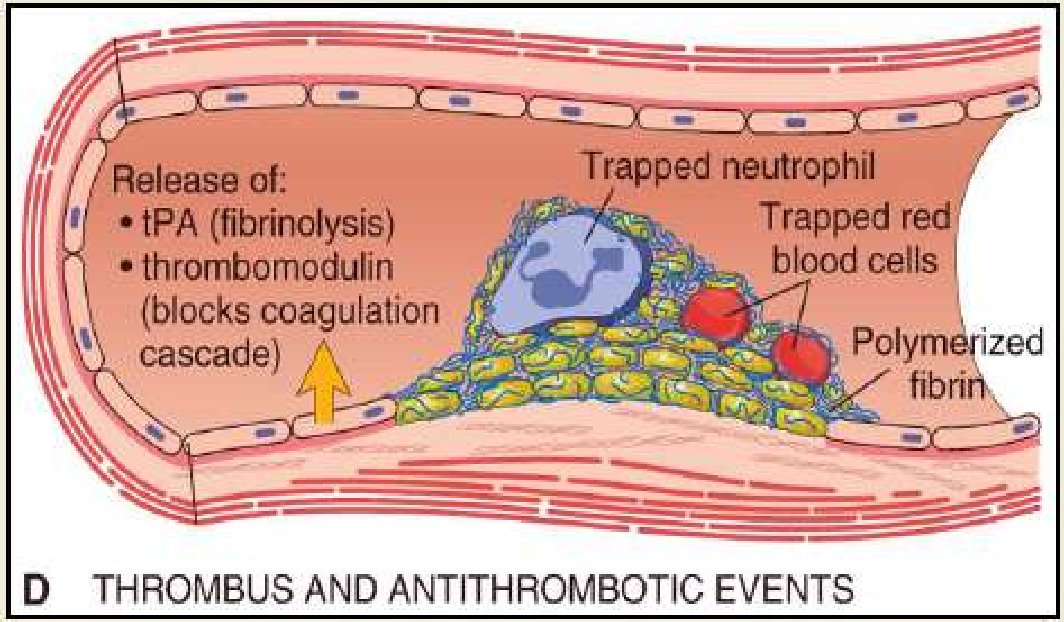


C SECONDARY HEMOSTASIS

Normal Hemostasis

Sequence of Events

1. Arteriolar Vasoconstriction
2. Primary Hemostasis - PLATELET
3. Secondary Hemostasis - Coagulation
4. **Antithrombotic Counter-Regulation**
   - Factors released to limit the size of the hemostatic plug

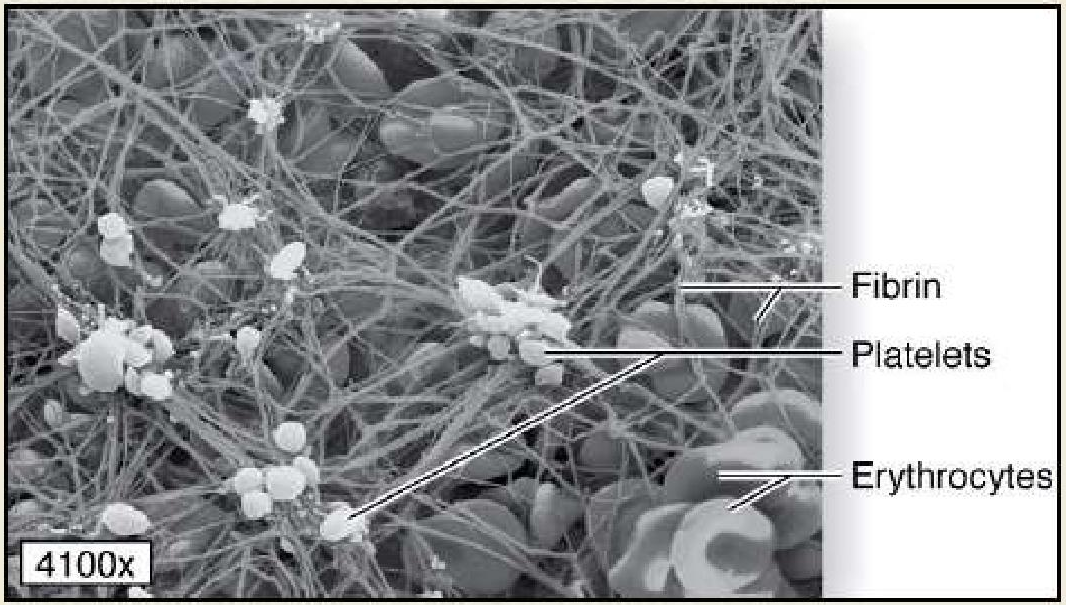


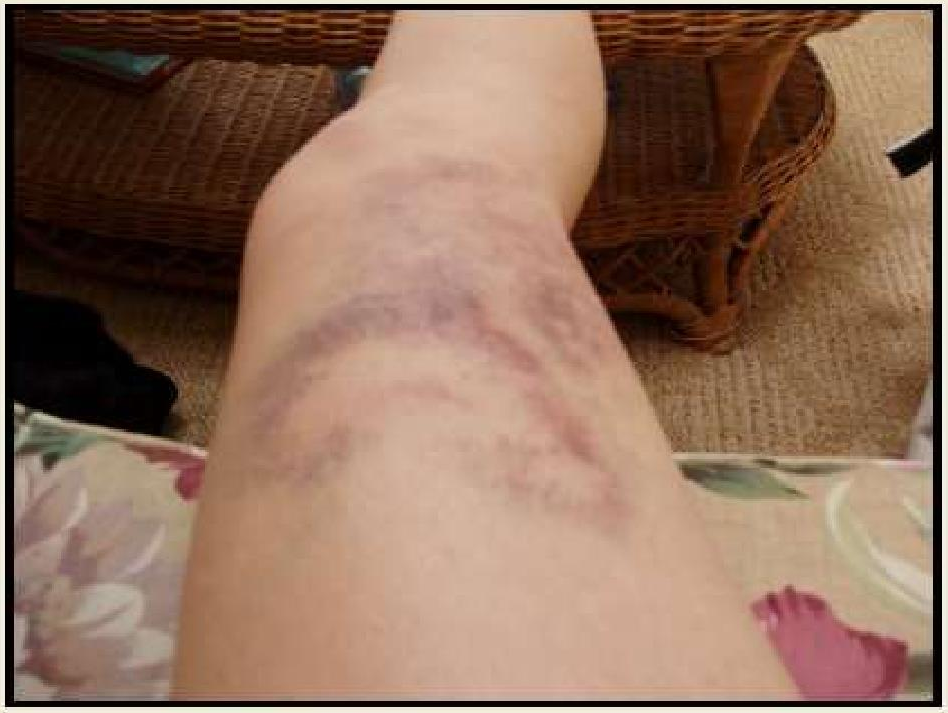
D THROMBUS AND ANTITHROMBOTIC EVENTS

Normal Hemostasis

Sequence of Events

1. Arteriolar vasoconstriction
   - VASCULAR WALL
2. Primary hemostasis
   – PLATELET
3. Secondary Hemostasis
   – COAGULATION
4. Antithrombotic Counter-Regulation



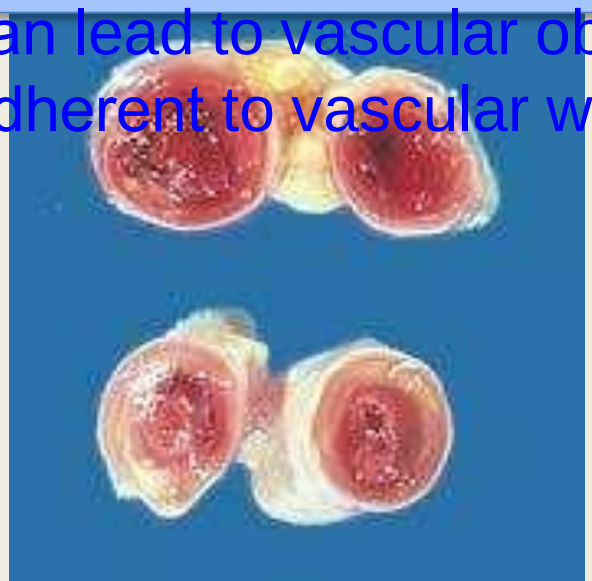


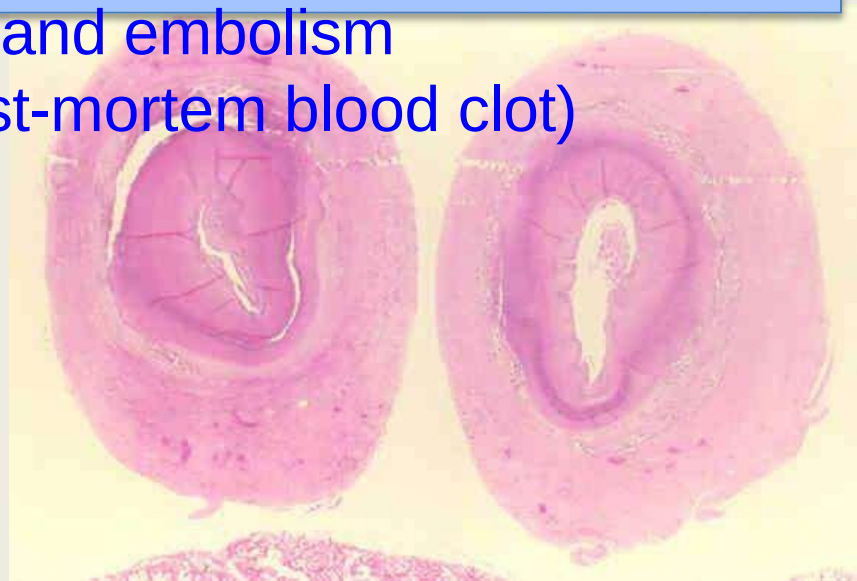
Thrombosis is inappropriate activation of hemostatic process in uninjured or slightly injured vessels

- formation or presence of a solid clotted mass (thrombus) within CV system

Thrombus (*pl. Thrombi*)

- aggregate of platelets and fibrin with entrapment of rbc's/wbc's
- can lead to vascular obstruction and embolism
- adherent to vascular wall (vs post-mortem blood clot)





Causes of Thrombosis : Virchow's

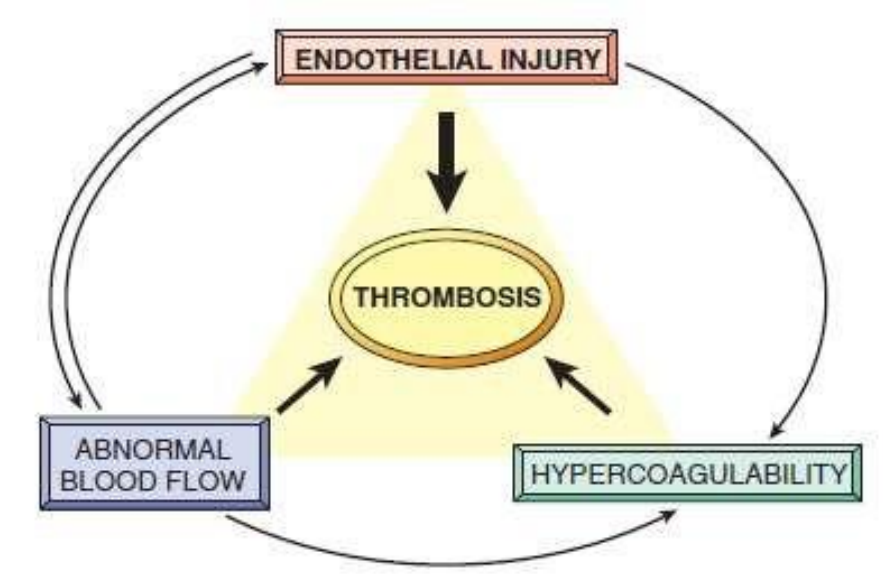
Endothelial injury

- Dominant influence
- Thrombosis by itself

Alterations in blood flow

- turbulence or stasis
- Platelets contact endothelium
- Endothelial injury / activation
- No dilution of clotting factors
- Build-up of thrombi

Hypercoagulability



Causes of Thrombosis : Virchow's Triad

1. ENDOTHELIAL INJURY

- **Viruses :** Herpes virus and arterivirus & pestivirus
- **Bacteria :** *Mannheimia hemolytica, Erysipelothrix rhusiopathiae*
- **Fungi :** *Aspergillus, Mucor*
- **Nematode parasites** : *Strongylus vulgaris larvae, Dirofilaria,*
- **Immune-mediated vacuities :** (e.g., purpura hemorrhagica,
- **Toxins**
- **Vitamin E/Selenium deficiency** (microangiopathy)
- local extension of infection
- Disseminated intravascular coagulation (DIC)
- Faulty intravenous injections

Causes of Thrombosis : Virchow's Triad

2. ALTERATIONS IN BLOOD FLOW

- **local stasis or reduced flow**
  - Gastric dilation and volvulus
  - Intestinal torsion and volvulus
  - External compression of vessel
- **Cardiac disease**
  - Cardiomyopathy, cardiac hypertrophy
- **Aneurysm :**
  - *Strongylusvulgaris, Spirocerca lupi*
- **Hypovolemia**
  - shock, diarrhea, and burns

Causes of Thrombosis : Virchow's Triad

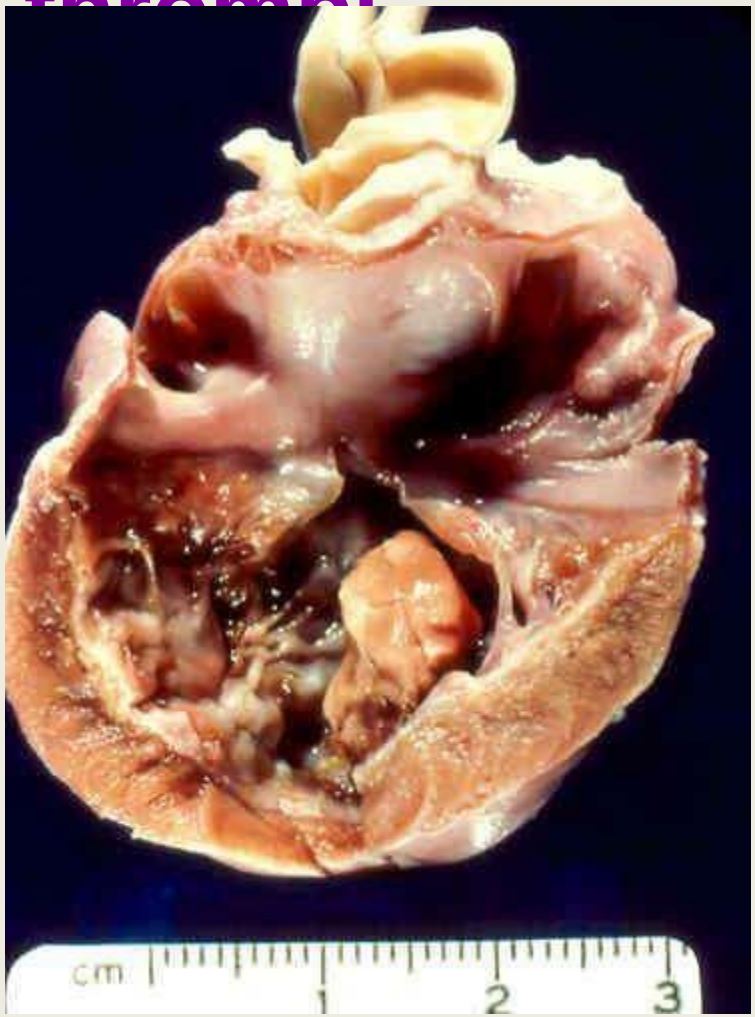
3. HYPERCOAGULABILITY

- **Enhanced platelet activity**
  - Diabetes mellitus, nephrotic syndrome , heartworm disease, uremia
- **Increased clotting factor activation**
  - Nephrotic syndrome, DIC, neoplasia
- **Antithrombin III deficiency**
  - DIC, hepatic disease, Glomerular amyloidosis)
- **Metabolic abnormalities**
  - Hyperadrenocorticism, hypothyroidism, Glomerulopathies

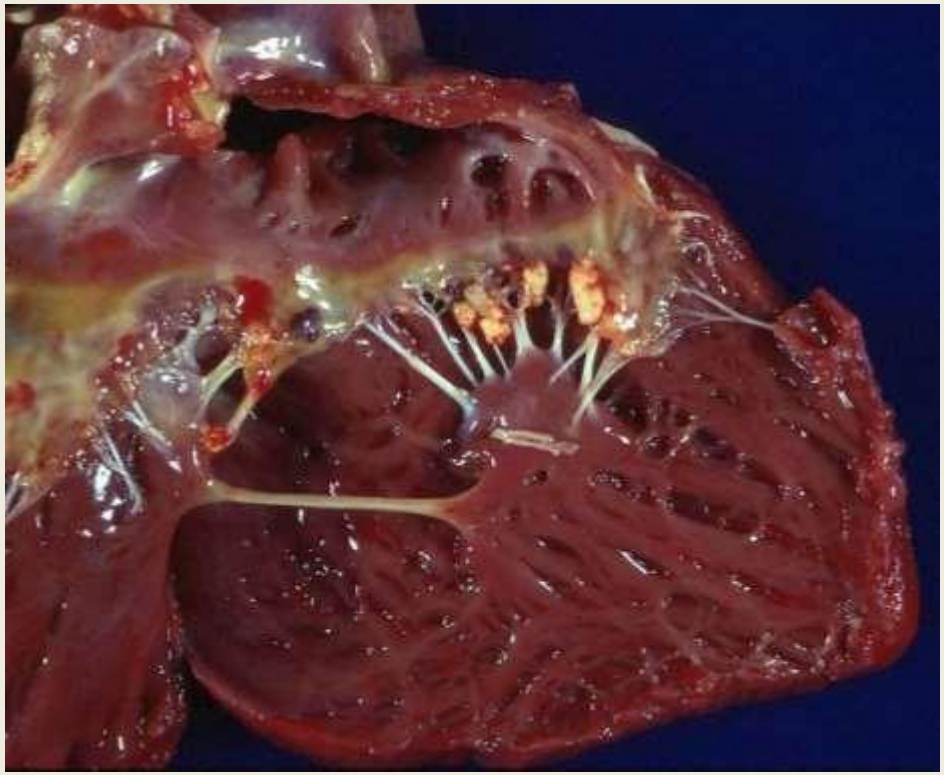
Pathogenesis of Thrombosis

(1) Platelets adhere to the exposed subendothelial collagen
(2) Activation of platelets secrete granule products (e.g., ADP and thromboxane A2
(3) Activation of intrinsic & extrinsic coagulation pathway
(4) Formation of a reversible primary haemostatic plug
(5) Converted into a larger irreversible secondary plug
(6) Deposition of fibrin within and around the aggregated platelets stabilizes the mass

Location of thrombi



Mural thrombus: heart chambers or in the aortic lumen



Valvular thrombi: attached to valves

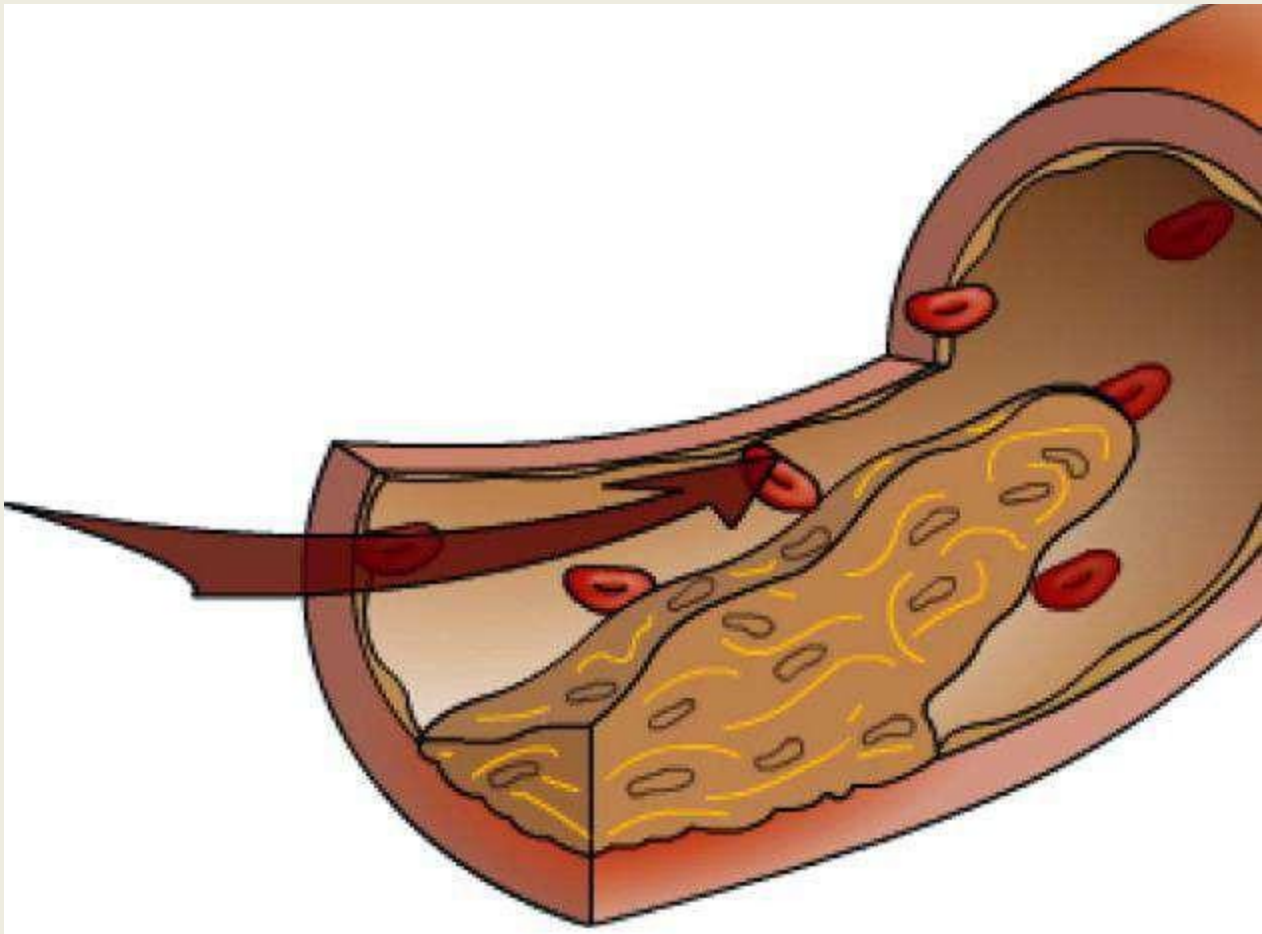
Location of thrombi

Arterial thrombi

- grow away from heart

more common than cardiac or venous thrombi

Strongylus vulgaris larvae in the anterior mesenteric artery



Arterial Thrombi

- usually form at sites of endothelial injury
- often paler & "meater" than venous thrombi

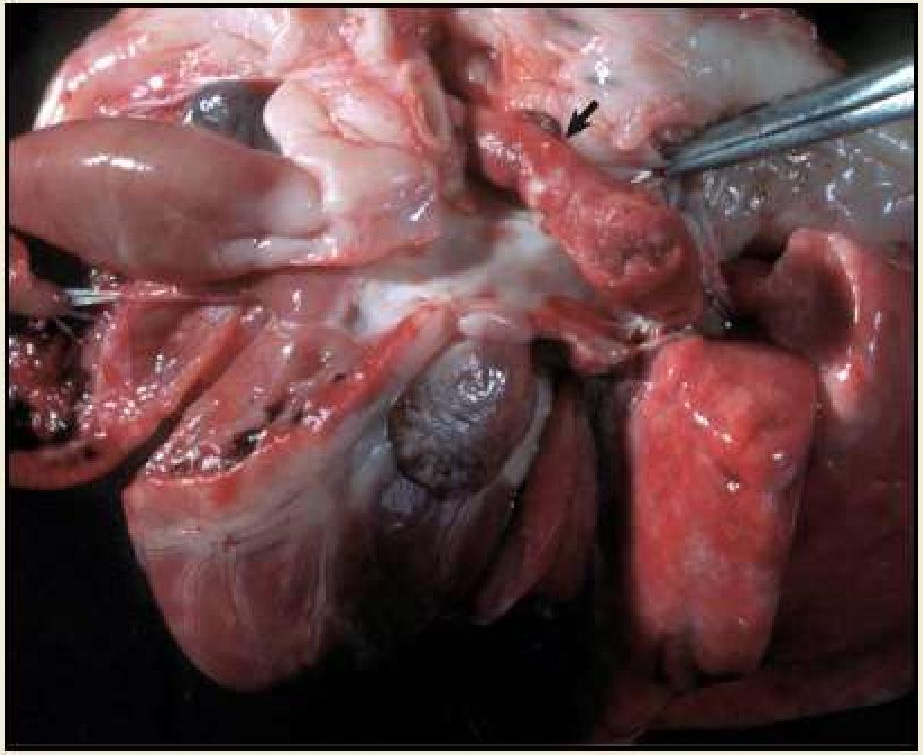


Figure 02-28. Arterial thrombus, pulmonary artery, dog. Arterial thrombi are composed primarily of platelets and fibrin because of the rapid flow of blood, which tends to exclude erythrocytes from the thrombus; thus they are usually pale beige to gray *(arrow)*.

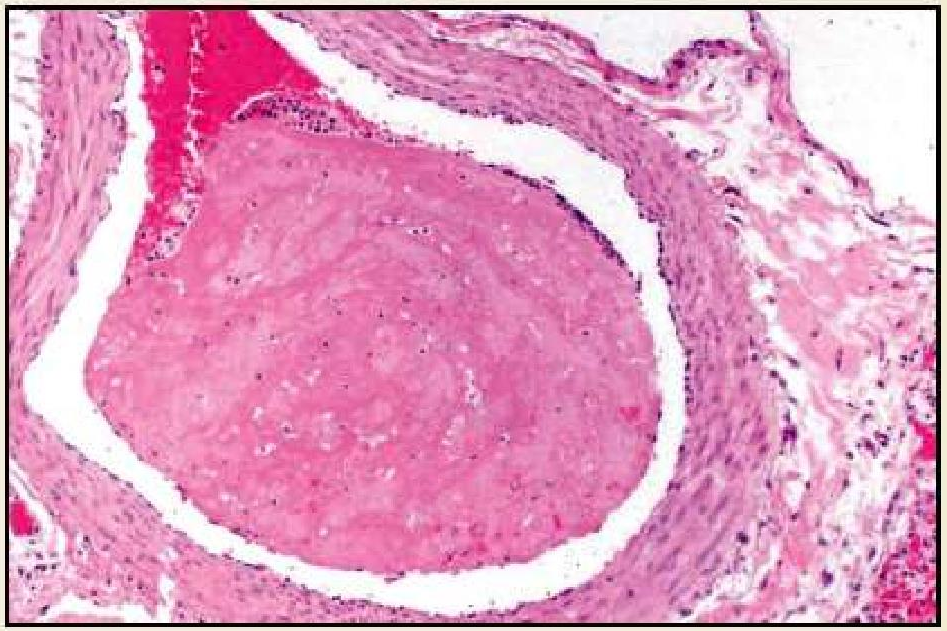


Fig. 2-32 Large thrombus, pulmonary artery, cow. Large thrombi are less readily dissolved by thrombolysis and therefore heal by other methods. This thrombus consists of a large coagulum of fibrin that has undergone little to no resolution.

Arterial Thrombi

- sometimes see alternating lines of rbc & fibrin ("lines of Zahn")

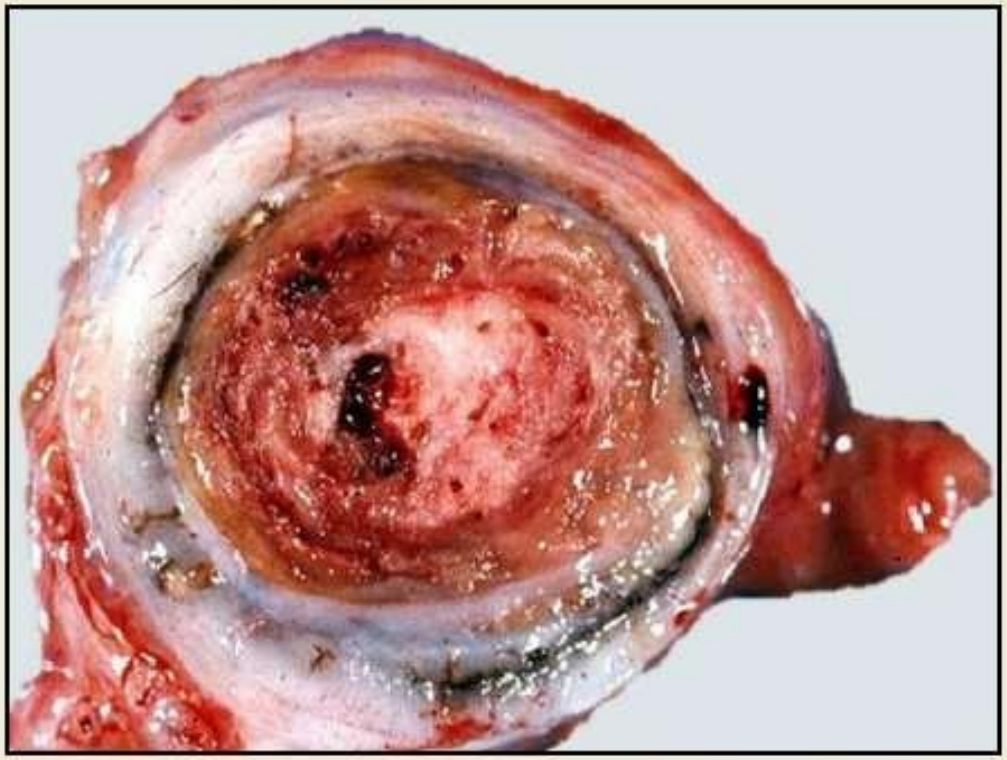
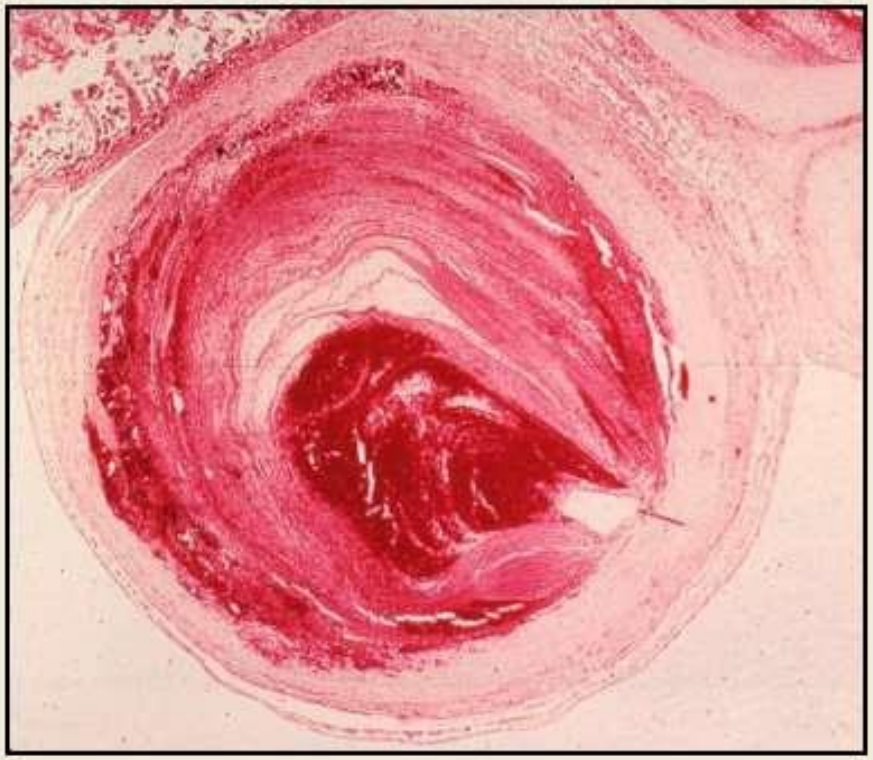


Figure 02-29. Arterial thrombus, lines of Zahn, cranial mesenteric artery, horse. Cardiac and larger arterial thrombi often have a laminated appearance characterized by alternating layers of platelets (dark) and fibrin (pale) intermixed with erythrocytes and leukocytes (lines of Zahn). These lines are the result of rapid blood flow in the heart and arteries/ arterioles that favors the deposition of fibrin and platelets and the exclusion of erythrocytes from the thrombus. This horse had verminous arteritis (*Strongylus vulgaris* fourth stage larvae) in the affected artery.

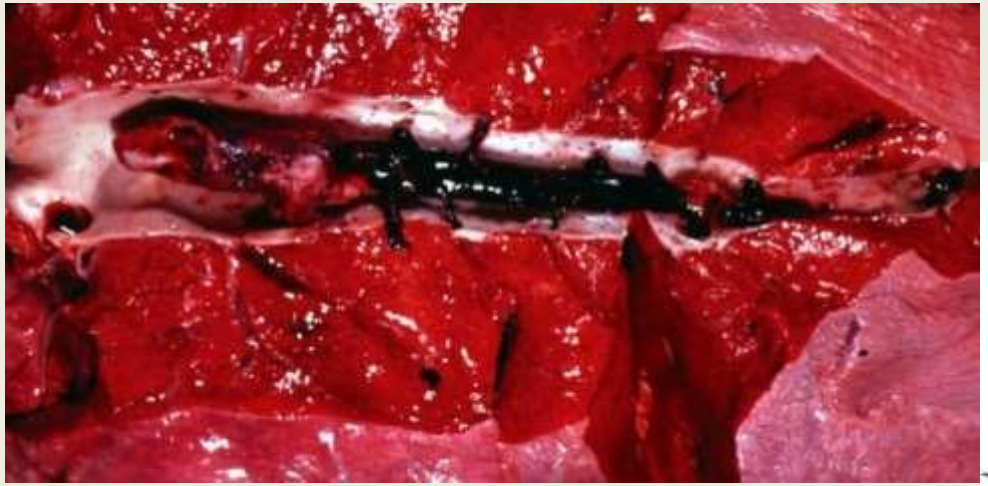


Arterial thrombus, histology. Note laminated appearance ("lines of Zahn")

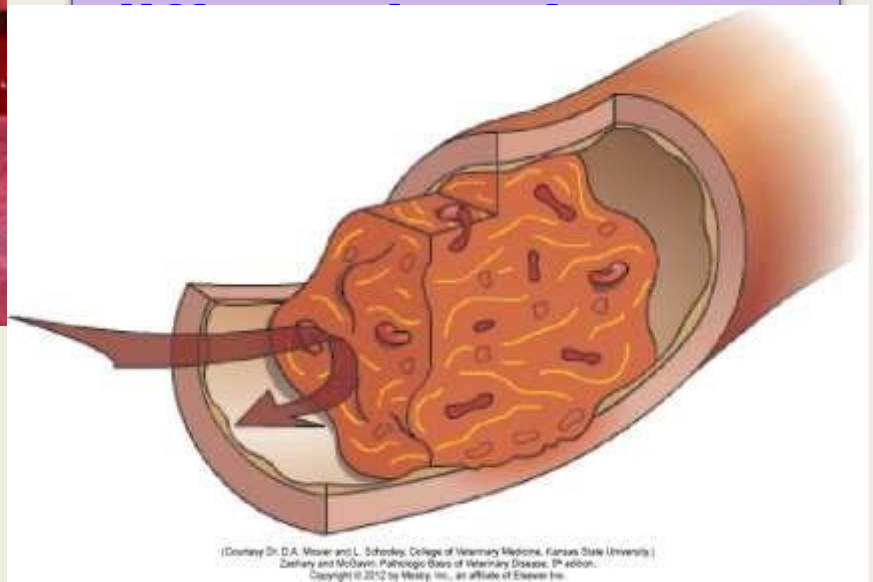
Venous Thrombi

- Toward heart
- Most venous thrombi are occlusive.

- **Usually form in slow flow environments**
- **Fibrin strands with entrapped rbc's**
- **More red colored**
- **Sometimes difficult to**

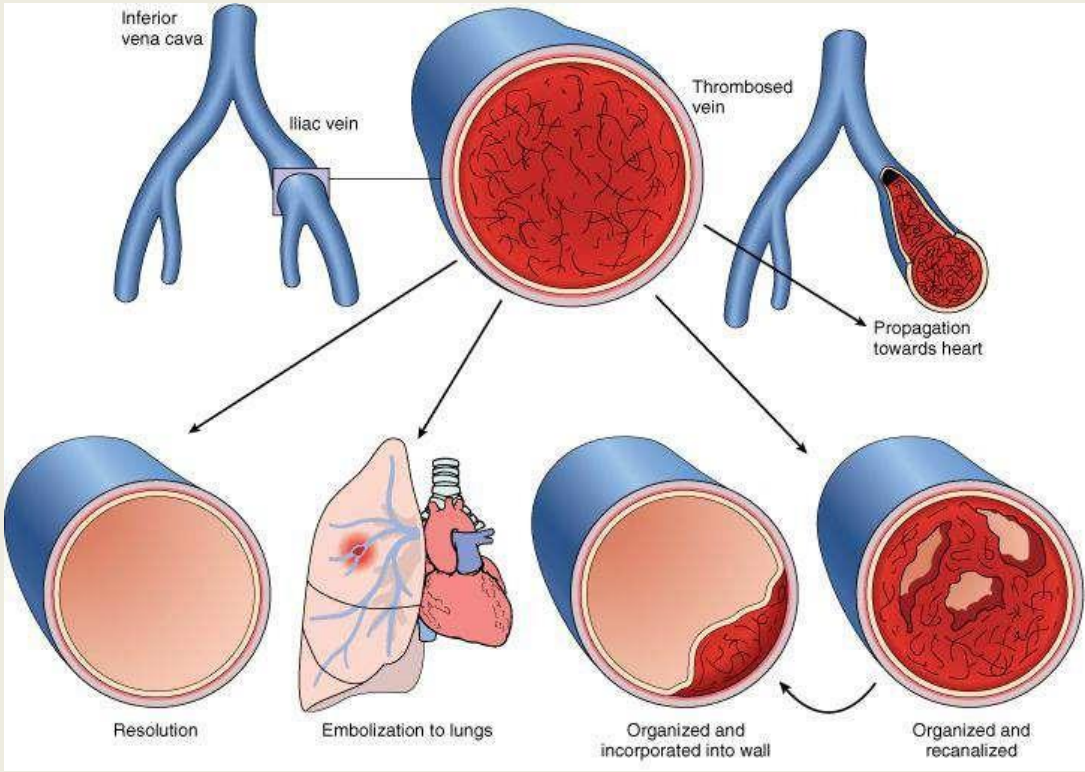


Venous thrombi, pulmonary vein, lung, horse. Venous thrombi become molded to the shape of the lumen of the vein

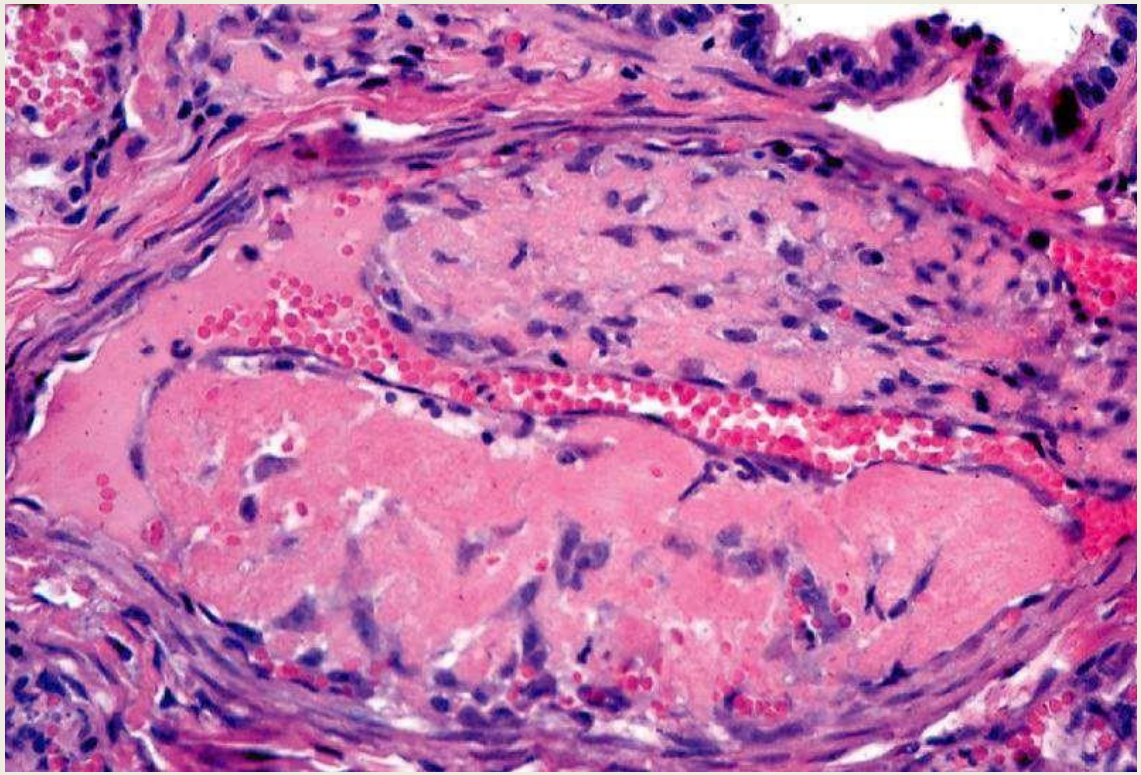


Fate of the Thrombus

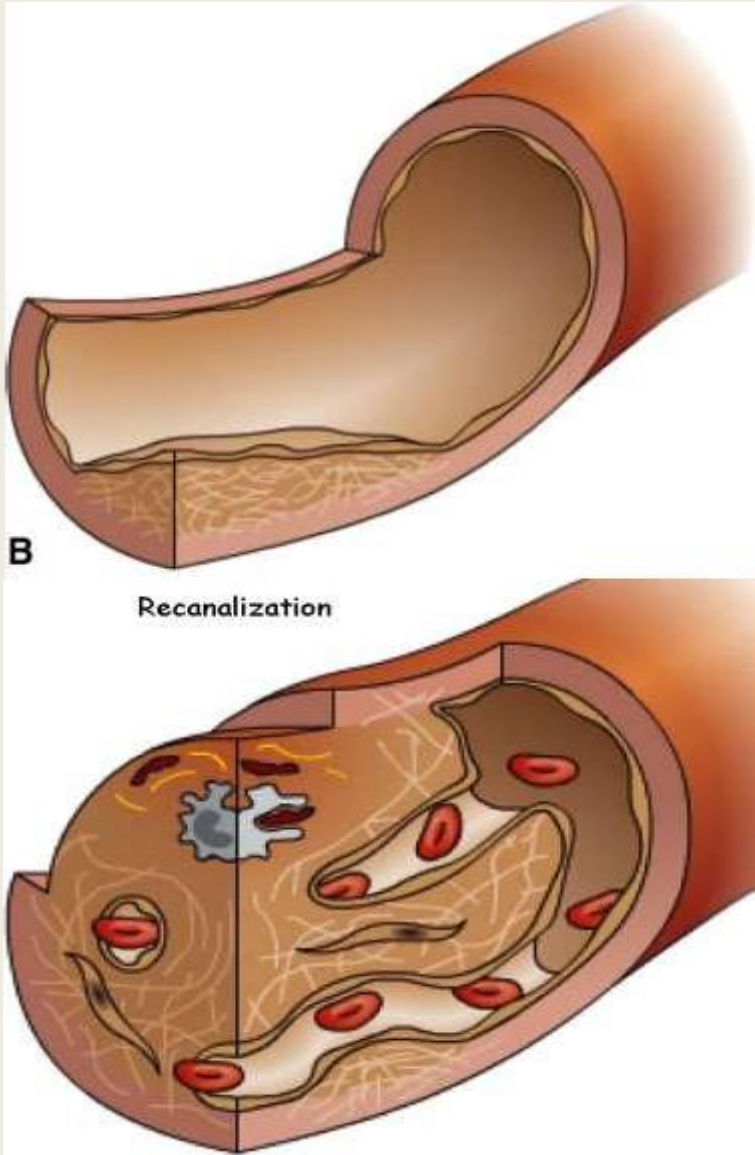
- Propagation
- Emboli formation
- Abscessation
- Dissolution
- Organization and re-canalization
- Calcification



Organization / Recanalization

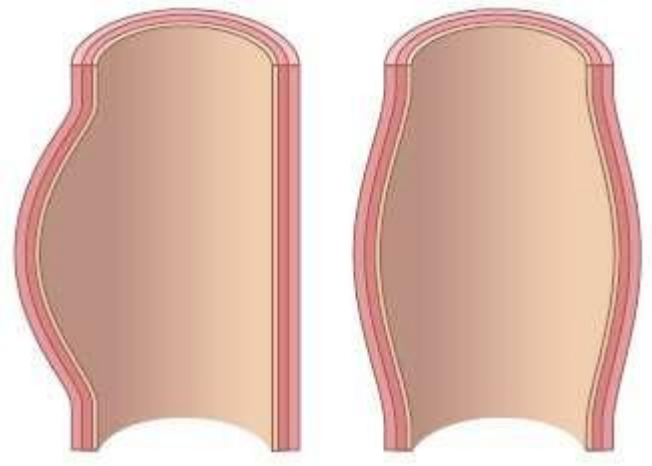


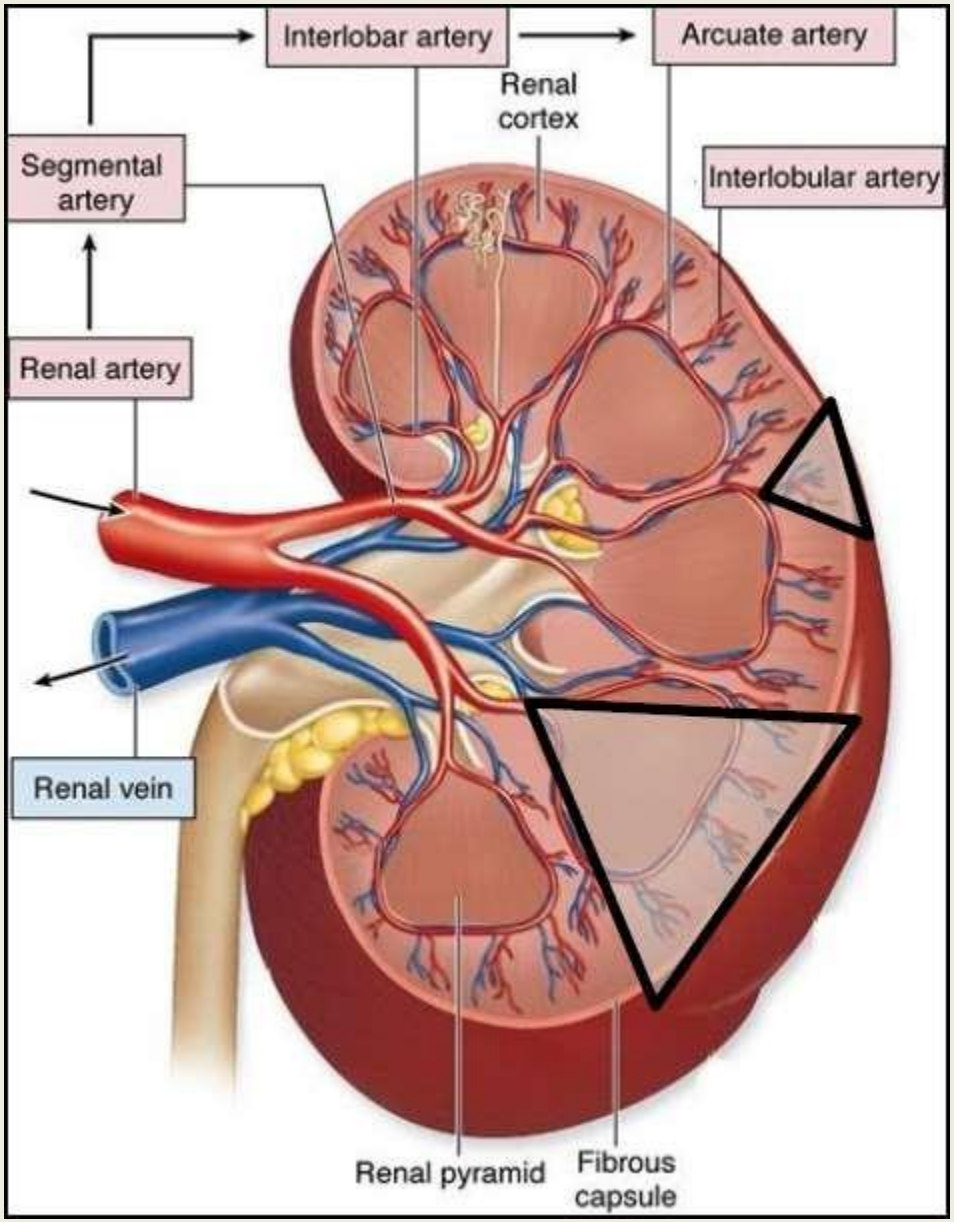
Occlusive mural thrombus, recanalization, cat. In occlusive and large thrombi, the healing process may occur by fibrosis and the invasion and growth of endothelial- lined vascular channels through the fibrosed area (recanalization). Note the vascular channel, horizontally in the middle of the thrombus. This provides alternate routes for blood flow to reestablish through or around the original thrombus



Significance (Effect of thrombus)

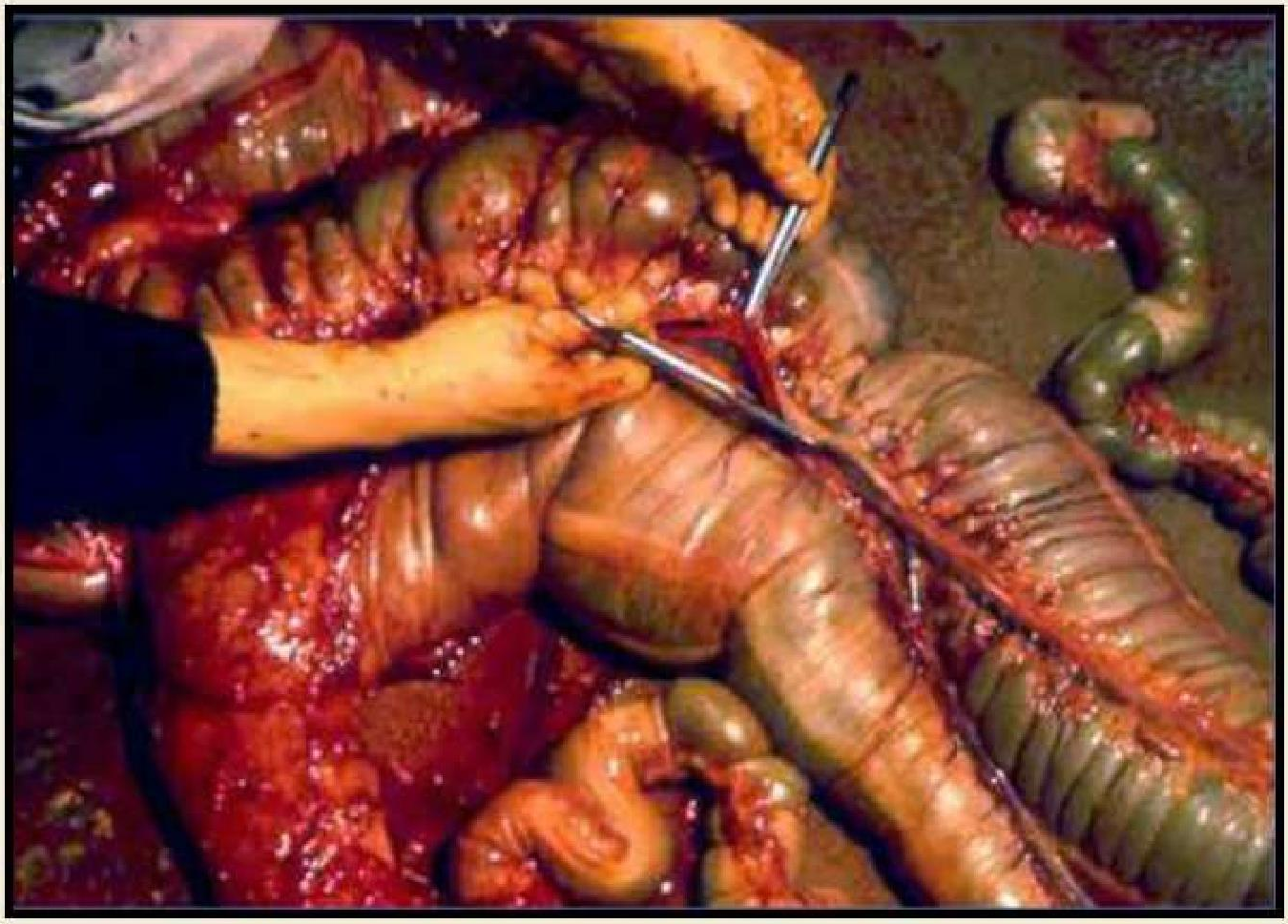
- Negligible effects
- Harmful effects
  - Infarction
  - Emboli
  - Oedema
  - Aneurysm





Blood Clot

- can refer to thrombus or post-mortem blood clot (so be specific)
- PM blood clot usually not associated with pathological change & not attached to wall



Chicken-Fat Clot

- gelatinous, yellow, post-mortem blood clot
- due to rapid rbc sedimentation rate
- mostly horses, pigs

Erythrocytes settle due to gravity in a post-mortem blood clot similar to blood in a test tube; giving "chicken fat" appearance to the upper part of the clot.

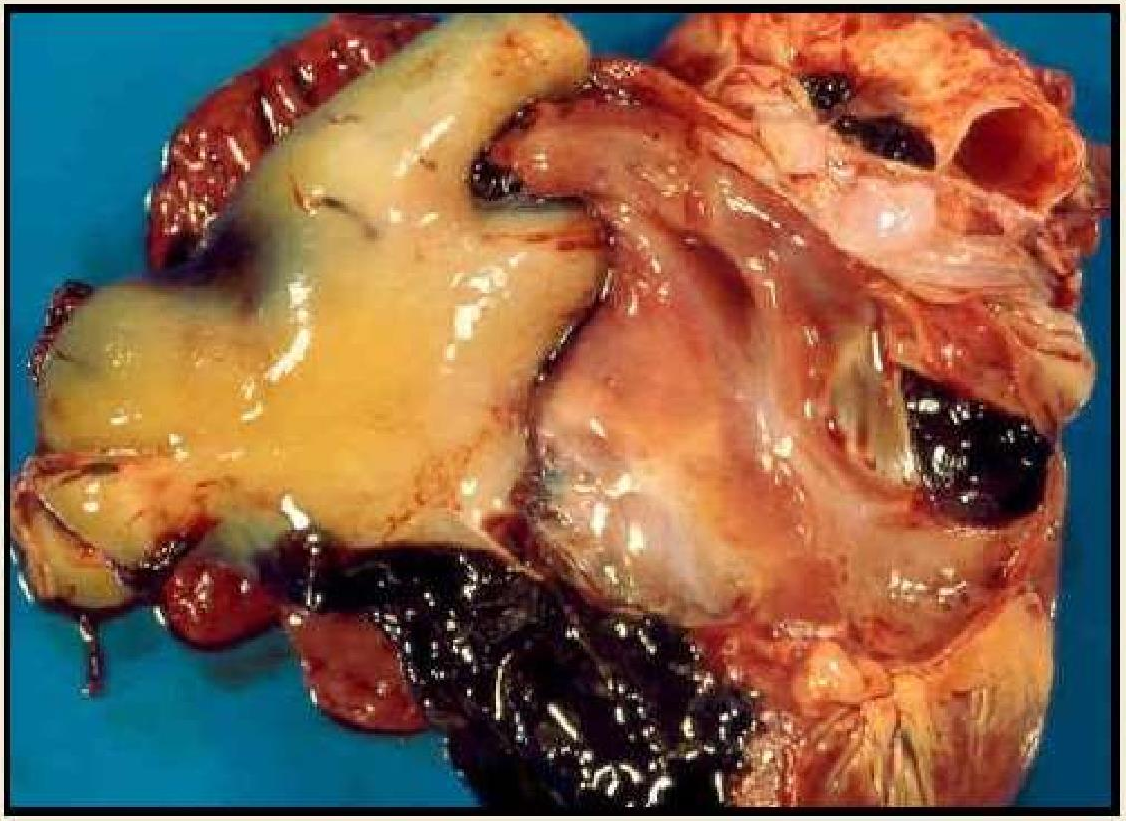
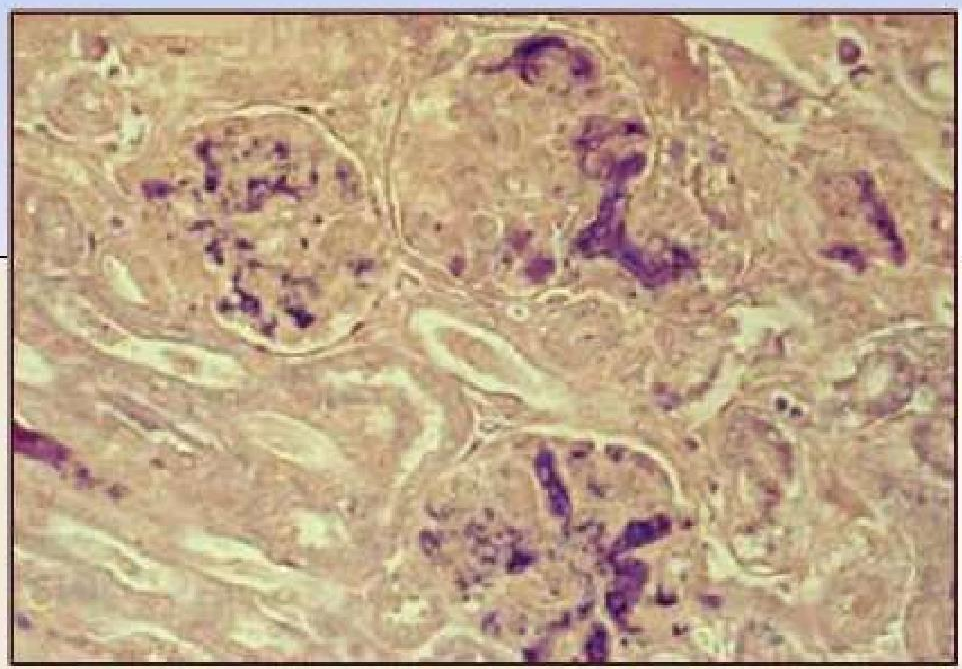


Table 11. Comparison of a thrombus and a postmortem clot

Thrombus	Postmortem clot
1. Granular, dry and rough	1. A rubbery, gelatinous coagulum
2. White or pale in colour	2. Intense red or yellow in colour
3. Attached to the vessel wall	3. Not attached to the vessel wall
4. Stratified in structure	4. Uniform in structure
5. Composed mainly of platelets	5. Composed mainly of fibrin
6. Vascular endothelium below the thrombus is damaged and rough	6. Vascular endothelium below the clot is undamaged, smooth, and glistening
7. Forms in a flowing stream of blood	7. Forms in a stagnant column of blood
8. Forms in the living animal	8. Forms in the dead animal
9. May be partially organized	9. No indication of organization
10. Caused by endothelial injury	10. Initiated by thromboplastin (tissue factor)

Disseminated Intravascular Coagulation (DIC)

- sudden onset of widespread fibrin thrombi in microcirculation
- often results in consumption of coagulation factors ***(consumptive coagulopathy)***
- causes:
  - **Severe burns**
  - **Heatstroke**
  - **Systemic viral disease**
  - **Shock (toxemia / septicemia)**
  - **Widespread metastatic tumors**
  - **Heartworm disease**
  - **Many other causes**



Note fibrin thrombi with glomerular capillaries (PTAH) stain