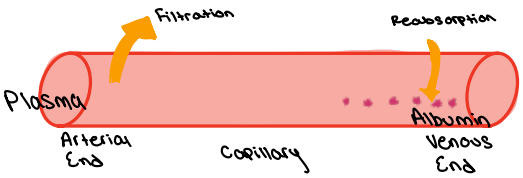
Vascular Injury: Hemorrhage, Thrombosis, and Tissue Response

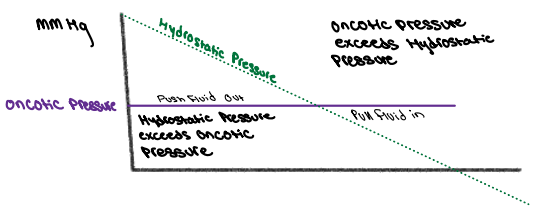
Learning Objective 1: Oncotic vs Hydrostatic Pressure

Oncotic: Pulls fluid in due to albumin

Hydrostatic pressure: Pushes fluid out due to blood pressure

↑ hydrostatic pressure or decrease in plasma osmotic pressure can lead to edema





Learning Objective 2: Basic Definitions

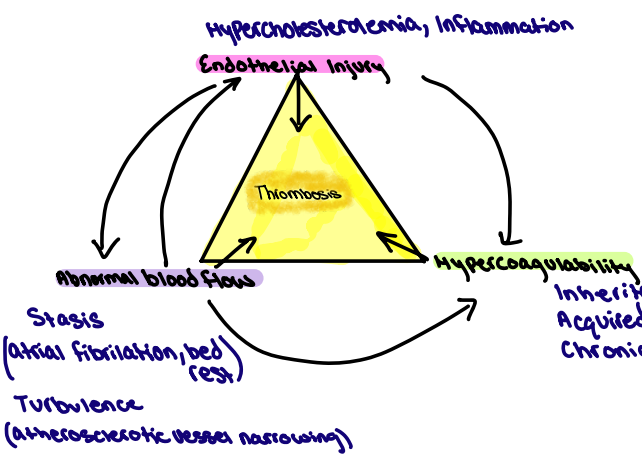
Ascites: Abdominal effusion

Pitting edema: Peripheral edema, you press in and the indent stays for a bit

Hydrothorax: Thoracic effusion (clear fluid)

Hemoabdomen: Blood in the abdomen

Learning Objective 3: Virchow's Triad



* Trinity of blood flow
  - Endothelium - smooth
  - Blood flow - laminar + fast
  - Coagulation factors - in balance

Disruption of any of these favors blood clotting (thrombosis)

Learning Objective 4: Petechiae, Ecchymoses, Hematoma

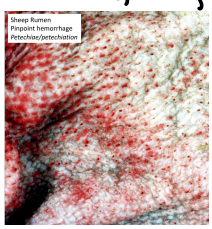
Petechiae

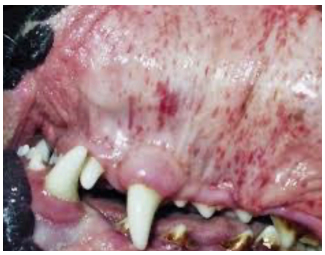
↳ Pinpoint hemorrhages

Present in skin/epithelia, oral cavity/mucous membranes, conjunctiva

Only 2 ways to get these pinpoint hemorrhages

1. Vasculitis
2. Thrombocytopenia



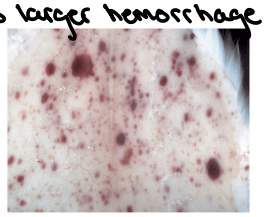


Ecchymoses

↳ Often looks like a blending of petechiae into larger hemorrhage

Causes: Vasculitis, Thrombocytopenia, Trauma, Bleeding disorders (clotting factors)

· Implies a more significant bleeding disorder than petechiae



igure 2-18 Ecchymotic Hemorrhages (Ecchymoses), Subcutis, abbit. Ecchymoses result from moderate injury to endothelial cells in the apillary beds. (Courtesy Dr. D.A. Mosier, College of Veterinary Medicine, ansas State University.)

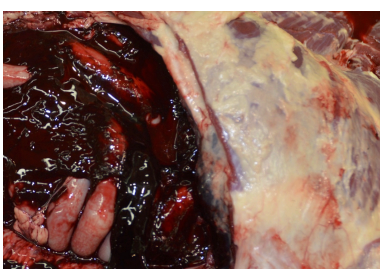
Hematoma

↳ Vessel rupture / damage

· Clotting factor depletion

· Coagulopathy

· NOT Thrombocytopenia



Learning Objective 5: Primary vs Secondary hemostatic plugs

Primary Hemostasis

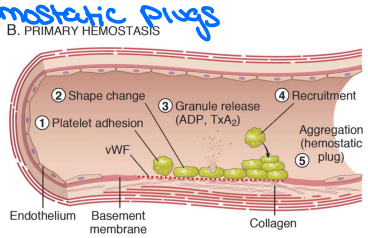
· Platelet Plug

LO 6 Dobermans *No Jug Stick

· Platelets are attracted to exposed collagen

· When aggregated, platelets become activated

· Secrete granules (ADP, TxA2) which cause more vasoconstriction → Grows plug



vWF: Von Willebrand's Factor. Mediates PLT adhesion!
TxA2= thromboxane A2

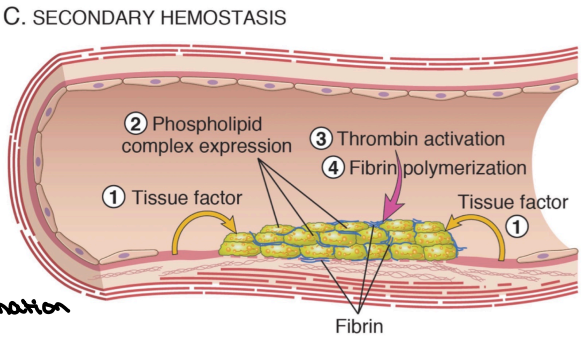
Dogs w/ deficiency in vWF have difficulty clotting. They may show signs of bleeding skin bruising, bleeding from the gums or nose, and excessive bleeding during surgery

Secondary Hemostasis

- Platelet plug already present
- This cements the plug
- With fibrin

Fibrin = queen of inflammation

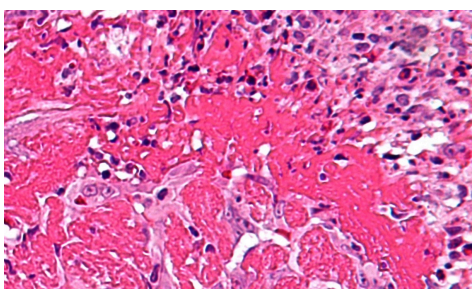
- fibrin is formed on the platelet membrane
- Then forms a supportive latticework for platelets



Learning Objective 7: Fibrin vs Fibrosis

Fibrin ≠ Fibrosis nor does Fibrin become Fibrosis

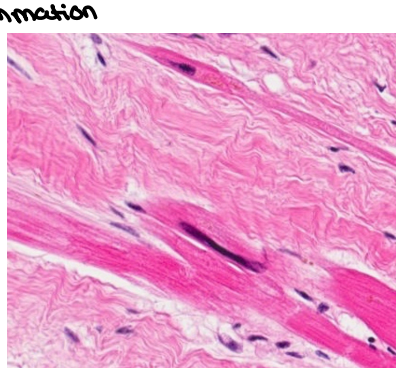
Fibrin/Fibrinous: Scab ACUTE inflammation



- Disorganized
- Acellular
- Protein exudation

Fibrous / Fibrosis: Scar CHRONIC inflammation

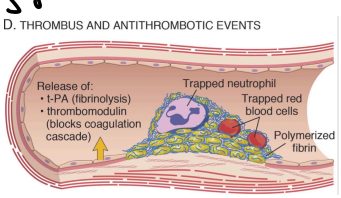
- organized
- Fibroblasts
- Collagen (dense regular/irregular)
- Contractile



Learning Objective 8: Thrombus Formation and Resolution

- Thrombosis occurs when the platelet + fibrin plug grows and it entraps WBC + RBC

-It can grow to occlude the lumen



Breakdown of Thrombus

- Fibrinolysis: Degradation of fibrin meshwork

- Allows "holes" to form in this large plug
- Endothelium will migrate into these holes

Recanalization: The restoration of blood flow through a thrombus

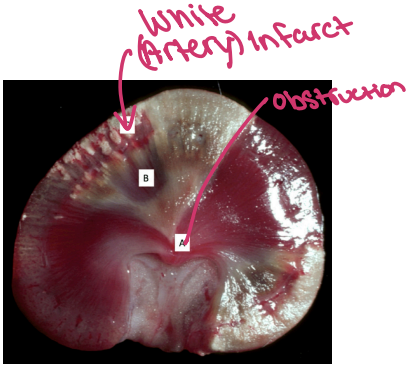
Learning Objective 9: White vs Red Infarct

Infarct: region of cell death caused by ischemia

2 major pathways

Arterial Obstruction: Blockade of blood delivery

Venous obstruction Blockade of blood drainage



White infarct: Arterial blockage → Tissue hypoxia → Necrosis → Pallor

Red infarct: Venous blockage → blood backup → blood stasis → Hypoxia → Necrosis

* Both are <u>Acute</u> regions of ischemia + tissue death only difference is if blood is present in lesion

Learning Objective 10: Chronic Infarct

- The ischemia causes tissue death
- Over time it is healed with fibrosis (chronicity)
- Fibrosis = scaring = contraction

Different than white infarct because it is chronic (scarred)