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Circulatory Derangements II

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1:00 PM

- **Fate of Venous Thrombi**
  - **Resolution –** Leukocytes chew up thrombus, dissolves and dislodges
  - **Embolization** – thrombus breaks off, becomes thromboemboli, migrates, and lodges in lungs
    - Can be so small that they are **asymptomatic**, or so large that they cause **sudden death**
  - **Organization/Recanalization** – inflammatory reponse to clot incites formation of granulation tissue
    - Vessel can "seal up" with a gran. tissue scar if resolution/organization happen concurrently
    - Organization advances around 1 week after clot forms
  - **Phlebolith –** "venous stone" – clot calcifies and forms a solid (atherosclerosis often?)
  - **Propagation** – additional Lines of Zahn form, clot grows
- **Fate of Arterial Thrombi**
  - Can come from heart valves, wall of heart (**mural** thrombus), aorta (usually **atherosclerosis**)
  - **Occulsion** – thrombus clogs artery
  - **Embolism** – movement of something (air, fat, amniotic fluid, cholesterol, foreign body, tumor, etc.)
    - **Embolus** – the "something", most often a **thrombus,** being moved
  - All emboli result in **infarction**
- **Thrombus vs. Embolus vs. Post-mortem Clot**
  - **Thrombus** – has **Lines of Zahn**, is a **cast of blood vessel**, and is **attached to vessel wall** (can organize)
  - **Embolus –** has **Lines of Zahn**, does **not fit vessel**, but can still **attach to vessel wall** (and organize)
  - **Post-mortem Clot –** has ***No* Lines of Zahn**, forms **in layers as blood settles**, and looks like **"chicken fat"** or **"currant jelly"**
- **Venous Emboli** – usually come from veins of lower extremity; thrombi in pulmonary artery extremely rare
  - Thromboembolus reaches pulmonary artery
  - Sudden death
  - Lung looks histologically normal
  - If not dead, pt experiences extreme SOB and chest pain
- **Arterial Emboli**
  - Occur on **left side** of circulation, usually coming from **aorta or heart**
  - **Symptoms** – always appear "**downstream"** from origin
  - **Atherosclerotic Plaque Embolus** – always **arterial,** two sources:
    - Can be **thrombotic embolus** from a clot from a plaque fissure
    - Can be **atherosclerotic debris** breaking off from the plaque itself – see crystals
    - Diabetes: atherosclerotic emboli travels to small vessels of foot --> toes die; neuropathic pt can't feel anything
  - **Iatrogenic factors –** from surgery
- **Cardiogenic Emboli**
  - 20% of ischemic strokes caused by cardiogenic emboli (other 80% are due to atherosclerosis in the cartoids
  - **Chamber** – number of sources:
    - Can be **mural** thrombus (from **infarction, myocarditis, valve disease)**
    - Can be **tumors** (*atrial myxoma* – can break off, or form thrombus on it to break off)
    - Can be from chronic **atrial fibrillation** – stagnant blood forming thrombus
  - **Valves** – emboli have a number of sources:
    - Can be **septic** (clouds of bacteria & PMNs, macrophages, from **endocarditis**)
    - Can be **marantic** (hypercoagulability, brought on by cancer in elderly)
    - Can be from **calcified valves** (rheumatic heart disease, on mitral valve)
  - Emboli may block coronary artery opening --> MI
- **Paradoxical Embolus** – thromboembolus from right side of circulation bypassing lungs; come through patent atrial septal defect to enter left heart --> the only way this can happen
- ***QUIZ:*** Will be given infarct result, must work backwards to figure out where it comes from
  - **Thrombi --> Emboli --> Occlusion --> Infarction**
  - An **arterial occlusion** – causes **coagulative necrosis**
- **Infarcts**
  - **Hemorrhagic "Red" Infarct –** partial occlusion leads to infarction but still **some blood flow**
    - **Liver and lung especially b/c of dual blood supply**
  - **Pale Infarct –** full occlusion leads to infarction and **no blood flow**

- **Myocardial Infarct Stages**
  - **0-4 hours** – not much, some waviness in fibers at border of infarct, staining defect
  - **4-12 hours** – some dark mottling, begin to see **coagulative necrosis, edema, hemorrhage**
  - **12-24 hours –** dark mottling, **coagulative necrosis**
  - **1-3 days –** mottled, yellow center, **coagulative necrosis --> karyolysis, PMN infiltration**
  - **3-7 days** – hyperemic border w/ yellow center, **disintegration of dead myocytes**, PMNs die, macrophages
  - **7-14 days** – **demolition phase** – very yellow-tan, **phagocytosis** by macrophages, **granulation tissue**
    - **Demolition phase** most vulnerable to rupture (deheniscince), very acellular
  - **2-8 weeks** – gray-white scar, **increased collagen deposition; angiogenesis**
  - **> 2 months** – scar complete, **dense collagen**
- **Central Hemorrhagic Necrosis, Dead Bowel**
  - **QUIZ**: **CHN** - See liver slide, indicates **heart failure**
  - **Dead Bowel** – embolus to blood supply to bowel, causing ischemic colitis, caused by **left side embolus**