Circulatory Derangements I

Tuesday, April 22, 2008
1:00 PM

- **Congestion**
  - **Active** - arterial dilation, "rubor"
  - **Passive** - venous outflow obstruction, congestion of heart failure usually
  - **Transient** --> resolves w/ removal of stimulus
  - **Long-term** --> results in fibrosis

- **Acute Pulmonary Congestion**
  - No inflammation --> acute
  - Passive b/c blood has accumulated in veins

- **Chronic Passive Congestion – Definition, Cause**
  - **Congestion** – from a *left ventricular failure*, can’t push blood out fast enough & *venous buildup*
  - **Pulmonary** --> fibrosis, *hemosiderin*-laden macrophages
  - Likely due to congestive heart failure (LV MI years ago)

- **Edema**
  - **Edema** – collection of fluid in interstitium (3rd space)
    - **Hydrostatic Pressure** – increase forces transudate to leak out
    - **Hypoproteinemia** – decrease in plasma proteins (proteinuria, malnourishment)
    - **Lymphatic Obstruction** – removal of lymph nodes, parasitic infections, etc.
  - **Pulmonary edema secondary to congestion** - you know it happened fast b/c there is no inflammation
    - Ruptured papillary muscle
    - Acute MI

- **Transudate vs. Exudate**
  - **Transudate** – protein-poor fluid, with & w/out inflammatory cells, denser; produced by:
  - **Exudate** – protein-rich fluid; oozes out of vessels due to inflammation

- **Consequences**
  - **Respiratory Tract** – edema of respiratory tract --> can’t breathe!
    - Also, as edema builds up in alveoli --> *decreased perfusion* of lung vessels --> hypoxic damage of capillary walls --> *protein leaks* into interstitium --> more edema, *snowball effect*
    - Edema of larynx can also become so great that *airway closed off*
  - **CNS** – cerebral edema causes IC pressure to build up, *veins compressed* – lethal
  - **Elephantiasis** - due to parasitic infection --> edema builds up --> leg swells

- **Cardiac Failure Causes** - heart is incapable of providing sufficient perfusion to meet tissue demands
  - **Myocardial Injury**
    - Ischemic changes - most common
    - Myocarditis - usually viral
    - Primary muscle disease - dilated, hypertrophic, restrictive
    - Systemic conditions - amyloidosis, hemochromatosis
  - **Load Problems**
    - **Valvular Stenosis** – regurgitant or stenotic valve
    - **Excessive Workload** – systemic hypertension (LV) or pulmonary hypertension (RV)
    - Abnormalities in conduction systems - arrhythmias
  - **Cardiac Compensation Mechanisms**
    - **Myocardial Hypertrophy** – heart muscle becomes larger and stronger to generate more pumping
      - Fibers *increase in size*, not more cells – as heart gets larger, *vasculature can’t accommodate*
      - Hypertrophied myocardium undergoes apoptosis, fibrosis, ventricle geometry changes
      - At risk for *sudden cardiac death*
    - **Frank-Starling Effect** – heart muscle dilates, larger volume and muscle stretches beyond ability to contract
    - **Increases in HR** - chambers don’t have enough time to fill

- **Cardiac Decompensation**
  - **Forward Failure** – decreased CO, *heart can’t pump blood to organs* --> causes:
    - **Left Heart Failure**
      - **Kidneys** – glomerular filtration rate plummets
      - **Liver** – Zone 3 areas suffer, as far from blood supply
      - **Colon** – mucosa underperfused, becomes ischemic
- **Backward Failure** – CO can’t keep up with input --> **blood supply before heart congested**
  - **Left Heart** – blood from lungs can’t get through LV fast enough, **passive congestion of lungs**
    - **Lungs** – dyspnea on exertion, orthopnea due to congested capillaries, paroxysmal nocturnal dyspnea
  - **Right Heart** – systemic blood supply can’t get thru RV fast, **central venous pressure increase**
    - **Liver** – sinusoids tend to get backed up from this (double whammy from forward failure)
    - **Edema** – fluid build-up leads to more in interstitium – can see pitting edemas in feet
    - Passive congestion of everything drained by a vein
    - Decreased perfusion of lungs
- **Renin System**: Heart Fails --> Decreased kidney perfusion --> Renin secretion --> Angiotensin activation --> Aldosterone released --> Kidney absorbs more sodium --> water follows --> increased vascular volume --> gives heart even more problems

- **Thrombosis**
  - **Thrombus** – blood clot formed during life (not post-mortem) and within vascular system (not clot out)
  - **Virchow’s Triad**: stasis (immobility), intimal damage (atherosclerosis), hypercoagulability (advanced carcinoma)
  - **Thrombosis** elicited by:
    - **Vessel wall disease** – include vasculitis (vessel inflammation), atherosclerosis (ulceration of endothelial lining, fissure of plaque induces clot)
      - **QUIZ**: look for plaque rupture (cholesterol crystals) inducing a clot
      - **Plaque stability** determine occlusion, not size --> **unstable** = thin roof, likely to rupture
      - **Organization**: 6-9 days is danger period b/c no collagen
      - **Timeline** of atherosclerotic thrombosis
        - Blows out; looks normal right after
        - 24h --> PMNs
        - 6-9d --> organization
    - **Flow abnormality** – stagnant flow can lead to thrombosis – bed-ridden, air travel, cast
    - **Hypercoagulability** – chemistry of blood more conducive to thrombosis
      - **Risks include acquired** – surgery, trauma, drugs, acquired antibodies, cancers
      - ...and also **genetic** – inherited forms of predisposition to clot
  - **Thrombus Formation**
    - **Platelet activation** – release thromboxane, attract more platelets, vasoconstriction
    - **Activation of Clotting System** – prothrombin --> thrombin; fibrinogen --> fibrin, clot forms!
    - **Lines of Zahn** – platelet ridges interrupted by pockets of coagulated blood, make lines
      - Can only occur if there is constant blood flow – must be alive for this, not post-mortem!
  - **Fate of Thrombi**
    - **Resolution** – Leukocytes chew up thrombus, dissolves and dislodge
    - **Organization/Recanalization** – inflammatory response 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
    - **Embolization** – thrombus breaks off, migrates, and lodges somewhere else
- **LAB**: Refer to Google Doc and Slides