open.michigan

Author(s): Aken Desai, Michael Mathis, 2008

Circulatory Derangements I

Tuesday, April 22, 2008
1:00 PM

- **Congestion**
  - **Active -** aterial 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, parastic 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 parastic 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, restritive
    - 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), hypercoagubility (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 occulsion, 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, vascoconstriction
    - **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 dislodges
    - **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**