Acute Respiratory Distress Syndrome (ARDS) and Acute Lung Injury (ALI)

- Mechanism

Systemic/Pulmonary Inflammatory Toxins Damage Pulmonary Endothelium/Epithelium
↓
Increased Alveolar Capillary Permeability
↓
CHF → Increased PCWP → Alveolar Edema + Inflammatory Material (not seen in CHF)
↓
Fibrin Hyaline Membrane Formation
↓
Scarring (after 3wks)

- Etiology
  - Direct Pulmonic Inflammation
    - Pneumonia (most common)
    - Aspiration
    - Near Drowning
    - Inhalational Injury
    - Lung Contusion
  - Indirect Systemic Inflammation
    - Sepsis (second most common)
    - Shock
    - DIC
    - Pancreatitis
    - Multiorgan Failure
    - Trauma/Burns
    - Bone Fractures
    - TRALI
    - Prolonged Cardiopulmonary Bypass
    - Intracranial HTN
  - NB given pneumonia and sepsis are the most common causes search for infection even doing a Bronch/BAL if everything is negative so far
- Diagnosis
  - 1994 American European Consensus Conference Criteria (need all 4/4, lung Bx helpful but not required)
    (1) Acute Onset (usually w/in 1d)
    (2) Bilateral (no need to be diffuse as CT shows heterogenous disease) Infiltrates on CXR
    (3) No Evidence of CHF defined by Swan-Ganz PCWP <18mm of Hg but also suggestive if no CM, Cephalization, etc
    (4) Hypoxemia Refractory to Oxygen Therapy defined by PaO_2/FiO_2 <200 (ARDS) <300 (ALI) 2/2 Increased Shunting (refractory hypoxemia)
        a. Blood does not see air because of the edema hence massive shunting resulting in refractory hypoxemia (after a while secondary pulmonary HTN can develop)
  - Other not part of the criteria
    - Predisposing Condition
    - Decreased Compliance (difficulty to ventilate)
      - High Peak Airway Pressure: V_T/P_{Plat} – PEEP <50cm H_2O
    - Fever, Tachypnea, Tachycardia, Hypotension, Leukocytosis, AMS
    - BAL would reveal neutrophilic dense, proteinaceous exudates in distal airways
- Treatment
  - Lung Protective Ventilation
    - Because the alveoli are so delicate you want to avoid Ventilatory Induced Lung Injury (VILI) allowing for permissive hypercapnea
      - Barotrauma (keep P_{plat} <30 cm H_2O)
      - Volutrauma (keep V_T <6mL/kg based on PBW not ABW)
        - THIS IS THE MOST THING ONE CAN DO TO HELP PTS WITH ARDS
        - Follow peak airway pressure b/c if >40cm H2O then you might want even lower tidal volumes
        - There will be permissive hypercapnea
      - VILI itself looks like ARDS and can cause Biotrauma in which VILI causes release of cytokines which in turn damage other distant organs
      - Approach: Start w/ 8mL/kg and decrease to 6mL/kg by 1mL/kg Qhrs then measure Pplat if <30 then good but if >30 decrease TV in 1mL/kg increments Q1hr until Pplat <30 or when 4mL/kg reached then monitor acidosis, if pH 7.15-7.30 then increase RR until pH >7.30 or RR 35pbm, if >35bpm needed to keep pH >7.30 then consider increasing TV slightly and giving bicarb
    - Because hypoxemia is so refractory you want to oxygenate while avoiding hypotension from high PEEP and oxygen toxicity from high FiO2

- PEEP (keep <15cm H_2O) the repetitive airway opening/closing itself can cause VILI, hence increase PEEP to stent airways open
- FiO2 (keep <60%)
- NB consider reversing I:E ratio
- NB Prone Ventilation which in theory decompresses the lung by mobilizing secretions, pulling the heart away, et al transiently improves oxygenation but no survival benefit
- NB consider High Frequency Jet Ventilation/Oscillation
- NB Extra-Corporal Membrane Oxygenation (ECMO) is controversial
- NB Nitric Oxide transiently improves oxygenation but no survival benefit

    - Fluid Balance by monitoring CVP w/ a central line or PCWP w/ a Swan Ganz
        - CVP (keep ~5cm H_2O) as low as possible to have the lowest PCWP but still adequate to maintain CO
        - Diuretic therapy is not helpful b/c it is not edema but inflammatory infiltrate that is filling the alveoli
    - Immunomodulating Nutritional Formulas (gamma linoleic acid, anti-oxidants, fish oil)
        - In theory they shift cytokine production from a pro- to an anti-inflammatory profile
        - Decreases duration of MV but had no survival benefit
    - Experimental Drugs
        - Steroids (methylprednisolone 2-3mg/kg/day) inhibit fibrosis
            - First 7 Days: no benefit b/c the fibrinoproliferative stage has not occurred yet
            - 7-13 Days: questionable benefit b/c active part of the fibrinoproliferative stage is occurring during this period
            - After 13 Days: no benefit b/c scarring is already complete
        - Surfactant Replacement have shown no benefit at all
        - Anti-Inflammatories (Pentoxyfylline, Ketoconazole, Prostaglandin-E1, NAC, Anti-TNF, Anti-IL1) have shown no benefit at all
        - Selective Pulmonary Vasoconstrictors (Almitrine)
    - Other
        - Avoid transfusions b/c they may also contribute to ARDS
- Complications
    - VILI
    - Scarring w/ permanent pulmonary function abnormalities
    - Infection
    - Multi-Organ Failure
- Prognosis
    - 40% overall mortality
        - 10% from pulmonary problems esp Infection
        - 90% from extrapulmonary problems esp Multi-Organ Failure
- DDx
    - Hemodynamic Pulmonary Edema
    - Diffuse Alveolar Hemorrhage
    - Acute Interstitial Pneumonitis (Hamman-Rich Syndrome)
    - Lymphangitis Spread of Cancer
- NB
    - Neonatal Respiratory Distress Syndrome (NRDS) aka Hyaline Membrane Disease
        - Similar to ARDS/ALI
        - Occurs after premature (<35wks) delivery when surfactant levels are low
        - Other complications: PDA, intraventricular brain hemorrhage, necrotizing enterocolitis