ARDS

ARDS is the most severe form of Acute Lung Injury (ALI) characterized by diffuse alveolar damage as a result of complex interaction of inflammatory mediators and stimuli of direct lung injury; mortality continues to be high; despite significant research, ventilation with small tidal volumes is the only treatment that shows a reduction in mortality.

ANESTHETIC CONSIDERATIONS:

- Profound hypoxemic respiratory failure
- Complications related to etiology of ARDS
  - Sepsis / SIRS
  - Infection / aspiration
  - Trauma
  - Transfusions
- Multi-organ system failure
- Lung protective ventilation strategies
  - 6 mL/kg Vt, PEEP & FiO_2 to avoid hypoxemia, goal $P_{Plat} < 30$
- Other Therapies
  - HFOV – theoretical advantages, improved oxygenation, no long-term proven benefit
  - iNO – improved oxygenation 24-48 h, no long-term proven benefit
  - Prone – improved oxygenation, no long-term proven benefit, increased mild complications
  - Surfactant – transient improved oxygenation, no long-term proven benefit

ANESTHETIC GOALS:

- Maintain oxygenation and end organ perfusion
- Avoid further lung injury – lung-protective ventilation strategy
- Treat the underlying cause

HISTORY

- Precipitating events:
  - Infection – sepsis syndrome 40% → ARDS
  - Trauma – 25% → ARDS
  - Pulmonary aspiration
  - Other – massive transfusion, inhalational injury, near-drowning, pancreatitis, overdose
- Acuity of onset (usually 12-72 h after precipitating event)
- Degree of hypoxemia (PaO_2:FiO_2 ratio)
- Any evidence of cardiogenic pulmonary edema
- Review mode of ventilation, current vitals signs
- Review trends in vitals, oxygenation, organ system function

TABLE 2. CLINICAL DISORDERS ASSOCIATED WITH THE DEVELOPMENT OF THE ACUTE RESPIRATORY DISTRESS SYNDROME.

DIRECT LUNG INJURY	INDIRECT LUNG INJURY
Common causes	Common causes
Pneumonia	Sepsis
Aspiration of gastric contents	Severe trauma with shock and multiple transfusions
Less common causes	Less common causes
Pulmonary contusion	Cardiopulmonary bypass
Fat emboli	Drug overdose
Near-drowning	Acute pancreatitis
Inhalational injury	Transfusions of blood products
Reperfusion pulmonary edema after lung transplantation or pulmonary embolectomy	

PHYSICAL

- VITALS - HR, BP, SpO_2, Temp
- CVS - volume assessment, end organ perfusion (capillary refill, U/O, LOC etc.)
- HEENT - current ETT size, edema
- RESP - look for : crackles, cyanosis, pleural effusions, PTX

INVESTIGATIONS

- **Labs**
  - Review trends in CBC, lytes, BUN, Cr, coagulation profile, ABG (acidosis and oxygenation)
  - Crossmatch, antibody screen
- **Imaging**
  - CXR (bilateral pulmonary infiltrates, line / tube placement, pleural effusions, r/o complications - e.g. PTX)
  - ECHO (R/O pulmonary HTN)
  - CT – degree of lung involvement (usually depended lung more affected), can differentiate exudative vs. fibroproliferative changes

OPTIMIZATION

- Lung Protective Ventilation (ARDSnet ventilation)
  - Vt 6 mL/kg (Ideal Body Weight)
  - Plateau pressures < 30 cmH_2O
  - PEEP 5-15 cmH_2O
  - Titrate oxygenation to lowest FiO_2 possible (SpO_2 >88-90%, PaO_2 > 60 mmHg)
- Treat respiratory acidosis
  - $NaHCO_3$
- Fluid Management
  - Conservative fluid administration associated with more ventilator-free days, more ICU-free days (in 28 days)
  - No mortality difference at 60 days
- Steroids
  - No evidence to support improved outcome early in disease
  - There may be some benefit for severe ARDS, late in disease that is refractory to other treatments
- Other immune modulators
  - PGE_1, ketoconazole, lisophyllin, antioxidants etc. – demonstrate no improvement in severity, duration or mortality due to ARDS
- Nitric Oxide
  - Improves oxygenation and reduces PA pressures but no survival benefit

- HFOV
  - No proven mortality benefit
- Prone ventilation
  - No difference in length of ventilation, mortality at 28 and 90 days
  - Higher PaO_2:FiO_2, decreased incidence of VAP
  - Increased incidence of selective intubation, ETT obstruction, pressure sores

ANESTHETIC OPTIONS

- Is surgery necessary, given high mortality?
- Can it wait until / if ARDS has resolved?
- Only emergency surgery!
- Can present to OR for multiple reasons, commonly: tracheostomy, trauma related injuries, sepsis source control (intra-abdominal etc.), etc. (possibly for diagnosis with lung biopsy)
- Usually patients already intubated and sedated therefore GA +/- NMB is easy, consider local / regional if appropriate
- Transport from ICU to OR will require monitors, ventilation, available NMB, sedation, pressors

ANESTHETIC SETUP

- **Drugs**
  - Usual anesthetic emergency drugs
  - iNO if already in use
- **Equipment**
  - CAS + 5 lead
  - Art-line, CVC
  - +/- PAC, consider continuous mixed venous oximetry
  - ICU ventilator / HFOV
  - Continue FiO_2, PEEP
  - Chest tube available (risk of barotrauma)
  - Consider double lumen tube, but likely unable to tolerate OLV or rapid desaturation during placement of DLT

MANAGEMENT OF ANESTHESIA

- **Induction**
  - Likely already intubated, ventilated, lined
  - IV or IH induction
  - Hemodynamically neutral, maintain preload in face of PEEP and possibly high ventilation pressures
- **Maintenance**
  - Any that maintains goals for hemodynamics, ventilation, oxygenation
  - Monitor tidal volumes, airway pressures closely
  - Consider iNO, if significant intraoperative hypoxemia to help with oxygenation
- **Emergence**
  - Often return to ICU

DISPOSITION & MONITORING

- ICU
- Continue ETT and PPV

COMPLICATIONS

- Ventilator induced lung injury
  - Barotrauma
  - Volutrauma
  - Atelectotrauma
- Refractory hypoxemia

PATHOPHYSIOLOGY

- Diagnosis of ALI and ARDS based on 4 elements
  - Acute onset (12-72 hours)
  - Arterial hypoxemia
    - PaO_2:FiO_2 </= 200 (ALI)
    - PaO_2:FiO_2 </= 300 (ARDS)
  - Bilateral pulmonary infiltrates consistent with pulmonary edema
  - PAWP < 18 mmHg or lack of clinical evidence of left atrial hypertension
- ALI vs. ARDS distinction is useful as a predictor of mortality
  - ALI mortality 33%
  - ARDS mortality 58%
- Annual incidence (US) estimated at 75 per 100,000
- Incidence of ARDS from sepsis alone 54 per 100,000 per year
- European and Australian data estimate that 7% of all ICU admissions have ALI / ARDS
- 16% of patients on mechanical ventilation > 24 h have ALI / ARDS
- ⅔ of patients with ALI will progress to ARDS
- Risk Factors for increased mortality:
  - Presence of cancer
  - Ventilation with Vt greater than ARDS-net protective strategy
  - Degree of multi-organ dysfunction
  - Higher mean fluid balance

    - NB! Sepsis, septic shock and oxygenation at onset of ALI / ARDS were NOT independently associated with higher mortality rates in ICU patients
- Death in ARDS patients:
    - 33% die from the initial insult (diagnosis prior to onset of ARDS)
    - 66% die from complications arising at the time of ARDS or after
    - Only 15% die of respiratory failure (hypoxemia or acidosis refractory to treatment)
- Traditional ventilation with Vt 10-15 mL/kg, peak a/w pressure up to or > 50 cmH_2O
- Principles of treatment
    - Lung-protection ventilation strategies
        - Low tidal volumes 6 mL/kg ideal body weight
        - Permissive hypercapnia
        - Limit plateau pressures < 30 cmH_2O
        - PEEP (particularly in early ARDS, reduces alveolar flooding, improves gas exchange and improves lung compliance)
- Low tidal volumes compared to traditional ventilation strategies conferred decreased mortality, more ventilator-free days DESPITE the low Vt group having higher PEEP, higher FiO_2 and lower PaO_2:FiO_2 ratio
- Duration of ventilation in survivors
    - 10-12 days
    - 10% of patients require ventilation > 3 weeks
- In survivors of ARDS, PFTs returned to baseline within 6 months to 1 year, except for continued impairment of CO diffusing capacity vs. baseline
- At 1 year after ARDS, patients continue to exhibit muscle wasting and decreased functional capacity (no control group of ICU patients without ARDS)

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