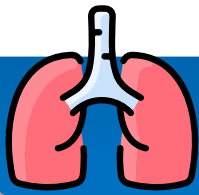




Round : Internal medicine

Group no : A4

Done By : Batool ALzubaidi



ARDS

I. PATHOPHYSIOLOGY OF ARDS

PULMONARY SHUNT

II. CAUSES OF ARDS

A. DIRECT LUNG INJURY

B. INDIRECT LUNG INJURY

III. CLASSIC FINDINGS OF ARDS

HYPOXEMIC RESPIRATORY FAILURE

COMPLICATIONS OF ARDS

A. HYPOXEMIC RESPIRATORY FAILURE WITH MULTI-ORGAN DYSFUNCTION

B. PULMONARY HYPERTENSION

C. VENTILATOR-ASSOCIATED PNEUMONIA (VAP)

D. VENTILATOR INDUCED LUNG INJURY (VILI)

IV. DIAGNOSTIC APPROACH TO ARDS

A. ASSESS FOR TYPE OF RESPIRATORY FAILURE

B. ASSESS FOR THE CAUSE OF TYPE I (HYPOXEMIC) RESPIRATORY FAILURE

V. TREATMENT OF ARDS

A. TREAT THE CAUSE OF ARDS

B. SUPPORTIVE CARE OF ARDS

00:33

I. PATHOPHYSIOLOGY OF ARDS

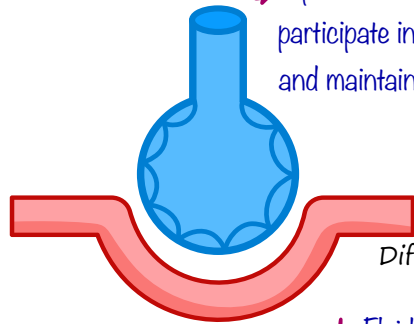
PULMONARY SHUNT

- o Lung Injury → Diffuse alveolar damage → Type I alveolar injury → ↑ Alveolar permeability → Severe Alveolar filling → No ventilation and poor gas exchange to alveoli → Severe hypoxemia

- o Lung Injury → Diffuse alveolar damage → Type II alveolar injury → ↓ Surfactant production → Severe alveolar collapse → No ventilation and poor gas exchange to alveoli → Severe hypoxemia

Normal

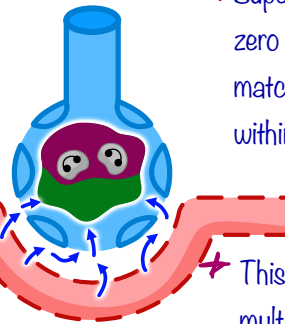
→ Squamous cells that participate in gas exchange and maintain a good barrier



⊕ Diffuse Alveolar Damage

→ Fluid can easily leak into the alveoli, it starts accumulating proteins, different types of immune cells

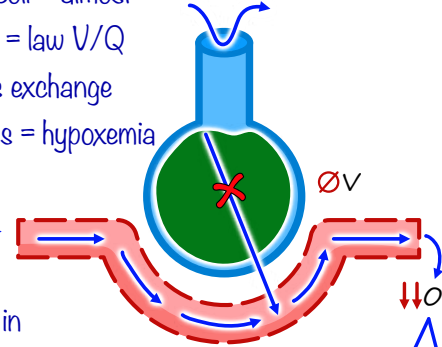
Type I Cell Injury



→ Super filled alveoli = almost zero ventilation = low V/Q match = no gas exchange within capillaries = hypoxemia

→ This occurs in multiple alveoli in both lungs

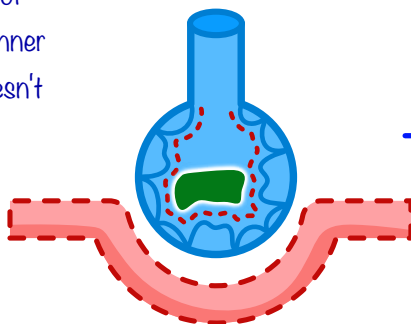
Pulmonary Shunt



→ They will feel very short of breath

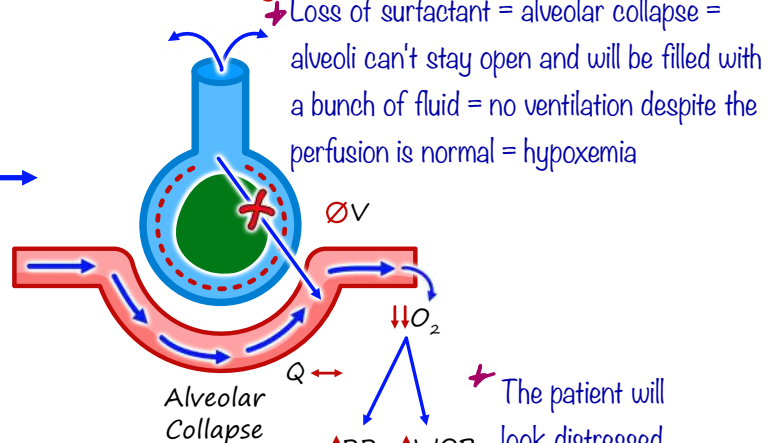
Type II Cell Injury

→ Cuboidal cells that are responsible for production of surfactant that coats the inner part of the alveoli so it doesn't collapse



→ The surface tension will start to rise & fluid will start to accumulate = alveolar collapse

Pulmonary Shunt



→ The patient will look distressed



II. CAUSES OF ARDS

A. DIRECT LUNG INJURY

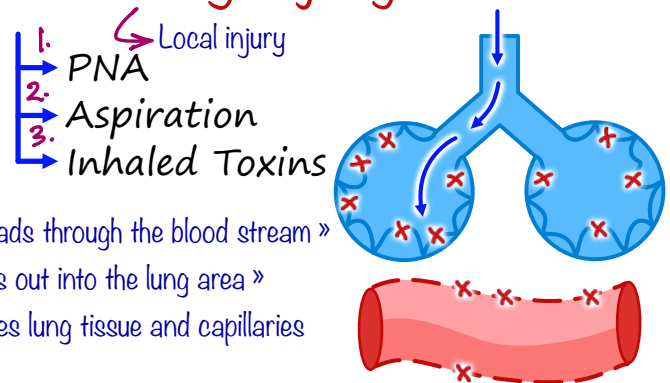
a) Pneumonia

- Severe bacteria or COVID-19 pneumonia → Diffuse lung inflammation → Diffuse alveolar damage
 - Assess for fever, productive cough, leukocytosis, and fevers

b) Aspiration

- Gastric content aspiration → Diffuse pneumonitis → diffuse alveolar damage
 - Assess for impaired level of consciousness that may be associated with aspiration

Direct Lung Injury



B. INDIRECT LUNG INJURY

a) Sepsis

- Systemic infection → Widespread systemic inflammation → Diffuse pulmonary capillary and alveolar cell injury
 - Assess for fever, leukocytosis, tachypnea, and hypotension (e.g. shock)
 - The most common cause of ARDS

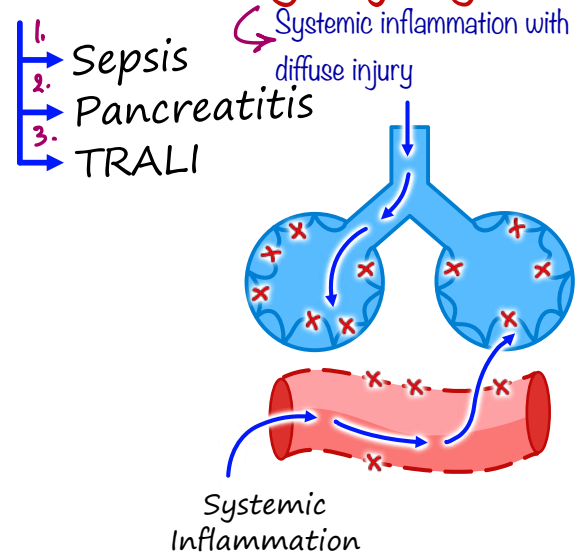
b) Pancreatitis

- Widespread systemic inflammation → Diffuse pulmonary capillary and alveolar cell injury
 - Assess for epigastric pain and ↑↑ Lipase (3 x ULN)

c) Transfusion-Associated Lung Injury (TRALI)

- Transfusion → Donor antibodies react against neutrophils → Widespread systemic inflammation → Diffuse pulmonary capillary and alveolar cell injury
 - Monitor within 6 hours post-transfusion

Indirect Lung Injury

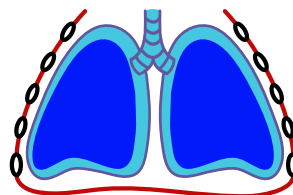


III. CLASSIC FINDINGS OF ARDS

Hypoxemic Respiratory Failure

- Dyspnea, Tachypnea, ↑ Work of breathing (WOB)
 - ↓ O₂ → Stimulates chemoreceptors → ↑ Respiratory drive to breathe
 - Hypoxia (SpO₂ < 90%) on pulse oximetry

Berlin Criteria



Acute Hypoxemia (< 1 wk) → SpO₂ < 90% or PaO₂ < 60 mmHg

Ratio (P/F < 300) → Determines the degree / severity of hypoxemia

Diffuse B/L Infiltrates

Swan (PCWP < 18) → Pulmonary capillary wedge pressure

→ Cardiogenic Pulmonary Edema

- PaO₂ = partial pressure of oxygen in arterial blood (from an ABG)
- FiO₂ = fraction of inspired oxygen (e.g., 21% = 0.21; 100% = 1.0)

Pulmonary capillary wedge pressure (PCWP)—also known as wedge pressure or pulmonary artery occlusion pressure (PAOP)—is an important hemodynamic measurement used to estimate left atrial pressure and indirectly assess left ventricular end-diastolic pressure (LVEDP).

How is it measured?

- It is obtained using a Swan-Ganz (pulmonary artery) catheter.
- The catheter is threaded through the right heart into the pulmonary artery.
- A balloon at the tip is inflated, "wedging" the catheter in a small pulmonary artery branch.
- The pressure measured at this point reflects back pressure from the left side of the heart.

Normal Value:

- 6–12 mmHg

Clinical Uses:

- Differentiate cardiogenic vs. non-cardiogenic pulmonary edema:
 - Cardiogenic (e.g., LV failure): PCWP > 18 mmHg
 - Non-cardiogenic (e.g., ARDS): PCWP ≤ 18 mmHg

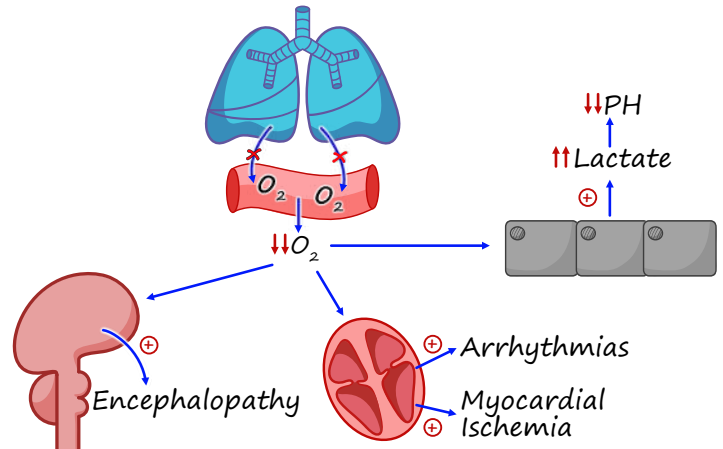


IV. COMPLICATIONS OF ARDS

A. Hypoxemic Respiratory Failure with Multi-Organ Dysfunction

- **Pathophysiology:**
 - Severe hypoxia → ↑Oxygen delivery to tissues → Organ ischemia → Organ dysfunction
- **Presentation:**
 - Hypoxia not responsive to oxygen supplementation (e.g. shunt physiology)
 - **Encephalopathy:**
 - Confusion → Stupor → Coma
 - **Arrhythmias:**
 - Atrial fibrillation
 - Ventricular tachycardia
 - **Myocardial Ischemia:**
 - Angina, ST changes, and a ↑Troponin
 - **Lactic Acidosis:**
 - ↑Lactate level
 - ABG with ↓pH and ↓HCO₃⁻

Multi-Organ Dysfunction

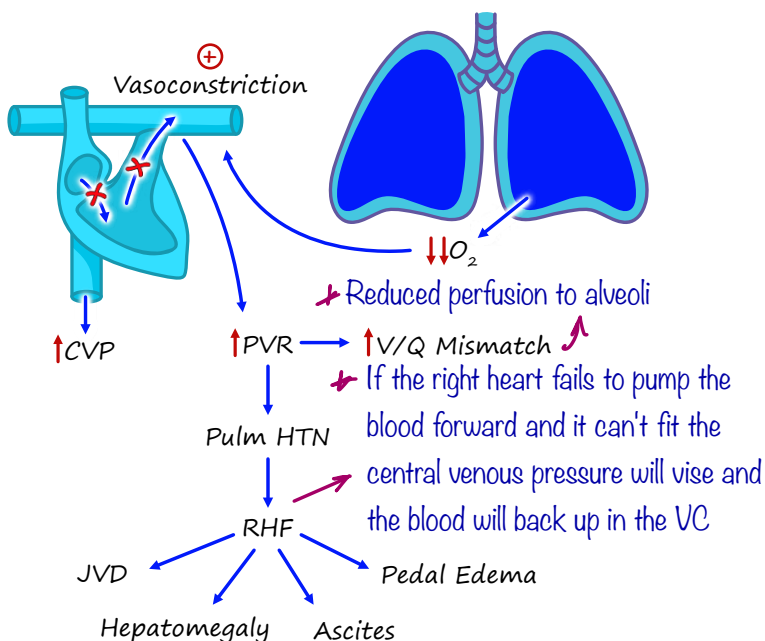


B. PULMONARY HYPERTENSION

12:24

- **Pathophysiology:**
 - Extremely severe acute hypoxemia → ↑Hypoxic pulmonary vasoconstriction → ↑PVR → ↑Pulmonary BP
- **Presentation:** Pulmonary vascular resistance
 - Dyspnea on exertion
 - ↑Risk of Right Heart Failure (e.g. Cor Pulmonale)
 - JVD
 - Ascites
 - Hepatomegaly
 - Lower extremity edema

Pulmonary Hypertension

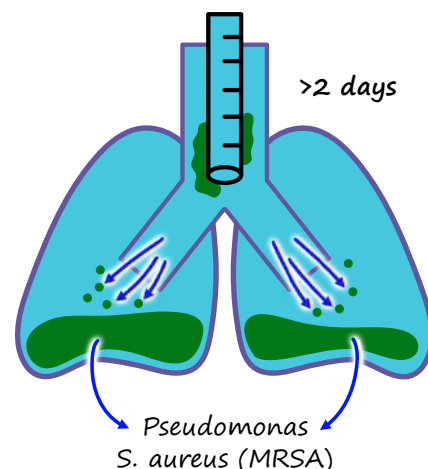


C. VENTILATOR-ASSOCIATED PNEUMONIA (VAP)

15:21

- **Pathophysiology:**
 - Severe ARDS that requires intubation → Prolonged intubation (> 48 hrs) → Risk of bacterial infection (e.g. MRSA or Pseudomonas) in the presence of ETT and microaspiration around ETT
- **Presentation:**
 - Fevers and Leukocytosis
 - ↑Purulent sputum Production
 - Worsening infiltrates on CXR
 - ↑Oxygen/ventilator requirements
 - Sputum culture (+) for MDR pathogen (e.g. Pseudomonas or MRSA)

Ventilator Associated PNA (VAP)



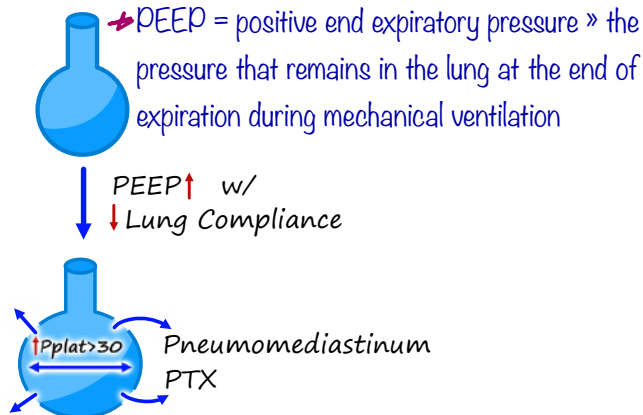
Tube is a foreign object to the body » it creates an opportunity for the bacteria to grow allowing it to spread to multiple parts of lung tissue which can lead to infection .. The longer the patient is intubated the higher risk of colonization and higher risk of infection



1. Barotrauma

- **Pathophysiology:**
 - ARDS with ↓ Lung compliance → Ventilator delivers **excessive** airway pressure via (↑ Plateau pressures and ↑ PEEP) → **↑ Alveolar rupture**
- **Presentation:**
 - ↑ Plateau pressures (> 30)
 - ↑ Risk of Pneumothorax and pneumomediastinum

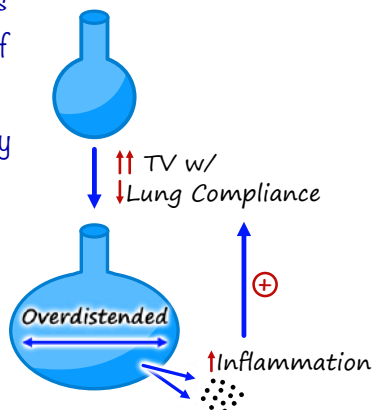
Barotrauma



2. Volutrauma

- **Pathophysiology:**
 - ARDS with ↓ lung compliance → Ventilator delivers **larger tidal volumes** → **Alveolar overdistention** → ↑ Alveolar rupture and inflammation (↑ Lung injury severity)
- **Presentation:**
 - ↑ Tidal volumes (> 6cc/kg of IBW)
 - ↑ Risk of Pneumothorax and pneumomediastinum

Volutrauma

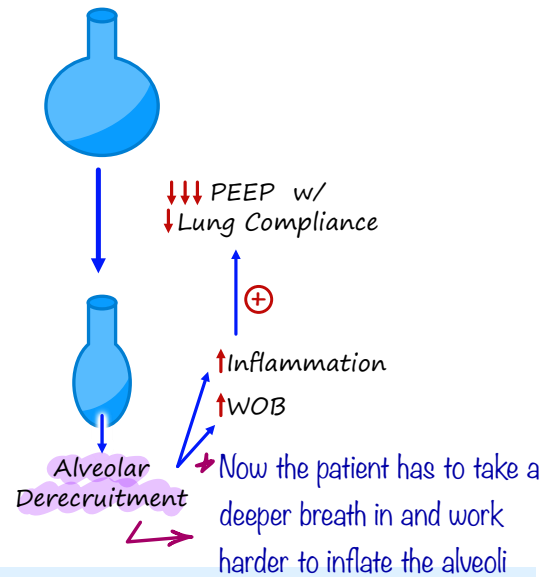


→ Overdistention causes injury to both types of lung cells » they start releasing inflammatory mediators increasing inflammation itself more alveolar edema and collapse » decreasing the compliance even more

3. Atelectrauma

- **Pathophysiology:**
 - ARDS with ↓ lung compliance → Ventilator delivers **very little PEEP** → alveoli minimally expand and quickly collapse → **↑ Inflammation** (which worsens lung injury)
- **Presentation:**
 - CXR with worsening atelectasis
 - ↑ FiO₂ requirements on ventilator

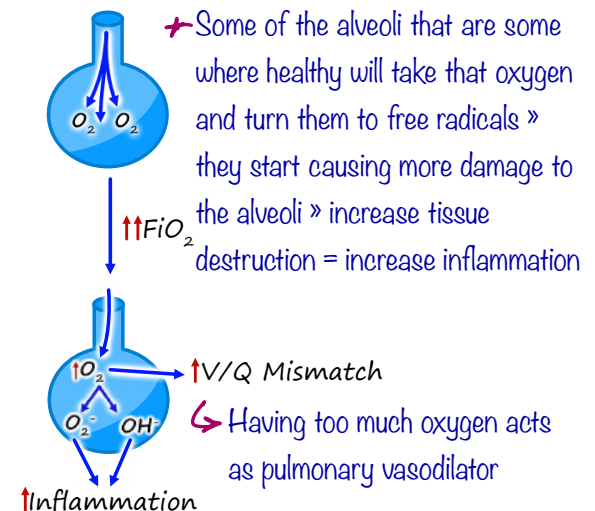
Atelectrauma

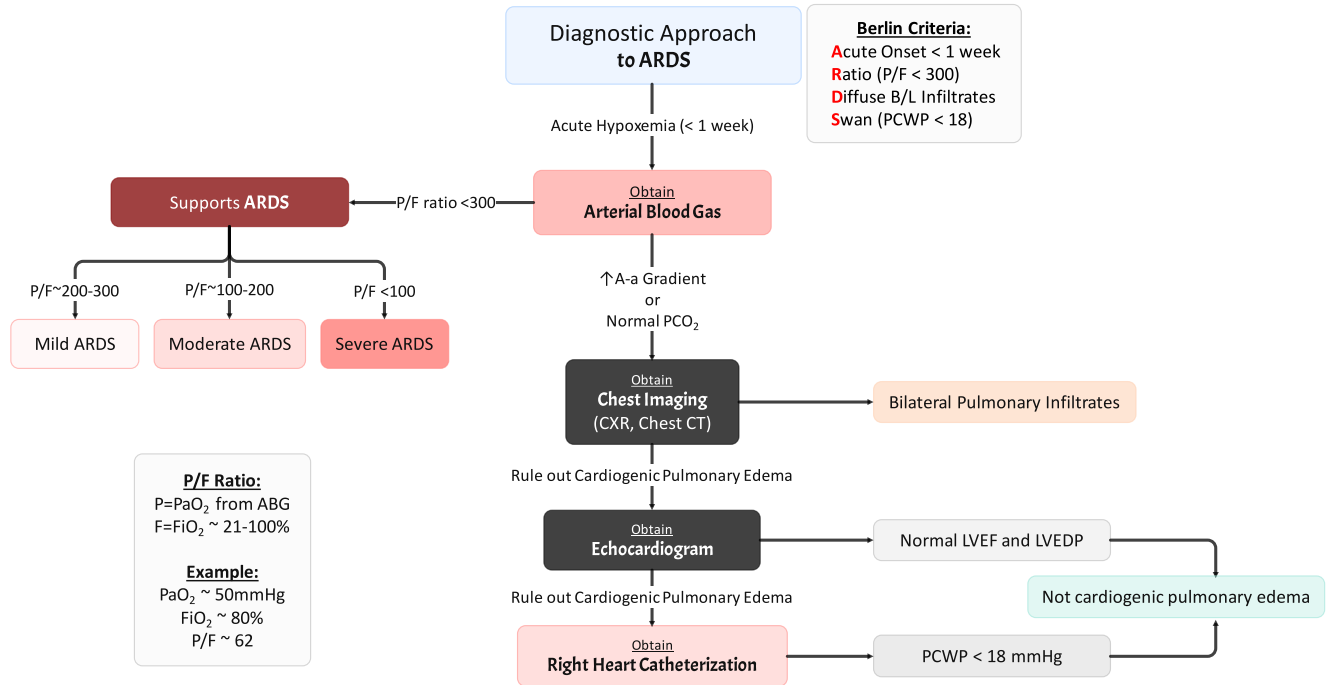


4. Hyperoxia

- **Pathophysiology:**
 - ARDS with ↓ lung compliance → Ventilator delivers **too much FiO₂** → Worsens V/Q mismatch and **↑ Inflammation** (↑ Lung injury severity)
- **Presentation:**
 - ABG with ↑ PaO₂ while on ↑ FiO₂

Hyperoxia

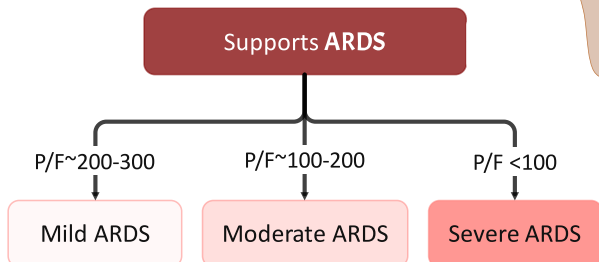
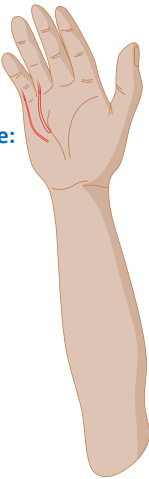




A. ASSESS FOR TYPE OF RESPIRATORY FAILURE

1. Obtain an ABG

- **Purpose:**
 - Differentiate between Type I and Type II respiratory failure
 - Assess the severity of ARDS via the P/F ratio
- **Abnormal Findings:**
 - **Type I (Hypoxemic) Respiratory Failure:**
 - PaO₂ < 60mmHg
 - Normal or Low PaCO₂
 - **Type II (Hypercapnic) Respiratory Failure:**
 - PaO₂ < 60mmHg
 - ↑ PaCO₂ (> 45mmHg)
 - **ARDS Severity via P/F Ratio:**
 - P/F ~ 200-300 → Mild ARDS
 - P/F ~ 100-200 → Moderate ARDS
 - P/F < 100 → Severe ARDS



2. Administer 100% FiO₂ Challenge

- **Purpose:**
 - Differentiate between V/Q mismatch and Shunt in Type I respiratory failure
- **Abnormal Findings:**
 - **V/Q Mismatch:**
 - Hypoxemia improves with oxygen supplementation
 - **Shunt:**
 - Hypoxemia DOES NOT improve with oxygen supplementation

P/F Ratio:
 P=PaO₂ from ABG
 F=FiO₂ ~ 21-100%

Example:
 PaO₂ ~ 50mmHg
 FiO₂ ~ 80%
 P/F ~ 62



B. ASSESS FOR THE CAUSE OF TYPE I (HYPOXEMIC) RESPIRATORY FAILURE

1. Obtain CXR or Chest CT

- **Purpose:**
 - To assess for the presence of pneumonia, pulmonary edema, atelectasis, alveolar hemorrhage, ARDS
 - To assess for the presence of PE (use CTPA study)
- **Abnormal Findings in ARDS:**
 - Diffuse infiltrates → ARDS
 - ✦ Diffuse bilateral opacities

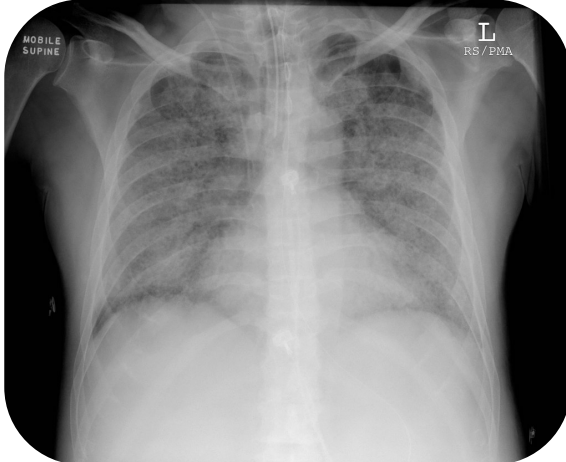


FIGURE 1. CXR SHOWING BILATERAL PULMONARY INFILTRATES

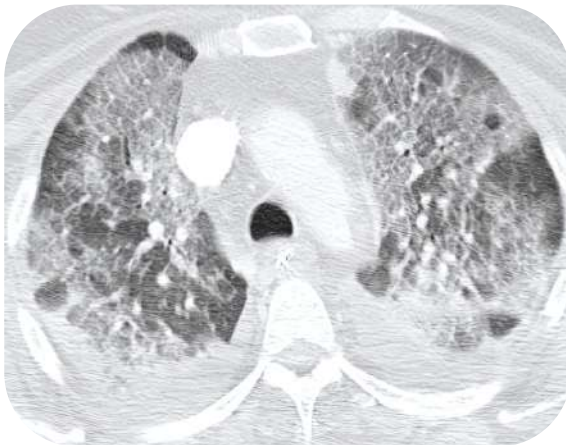


FIGURE 2. CT SHOWING BILATERAL PULMONARY INFILTRATES

2. Obtain an Echocardiogram

- **Purpose:**
 - Assess for intracardiac shunt (e.g. ASD, PFO, VSD)
 - Assess for evidence of cardiogenic pulmonary edema
- **Findings in ARDS:** Left ventricular ejection fraction
 - Normal LVEF or Normal Diastolic function helps support diffuse infiltrates are not from cardiogenic pulmonary edema and suggests ARDS

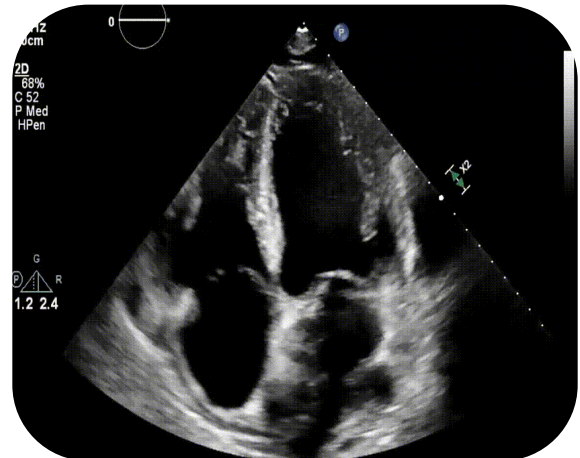


FIGURE 3. APICAL 4 CHAMBER VIEW SHOWING NORMAL LVEF & DIASTOLIC FUNCTION

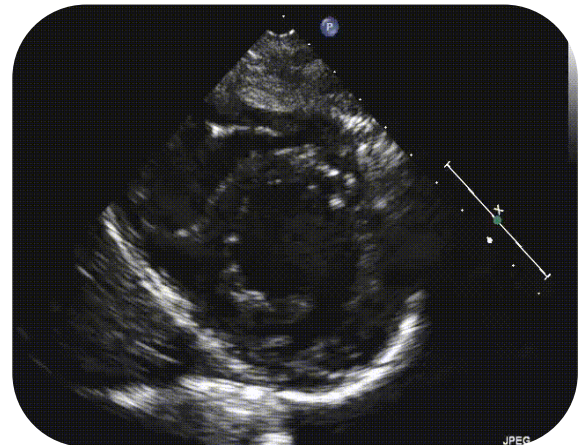
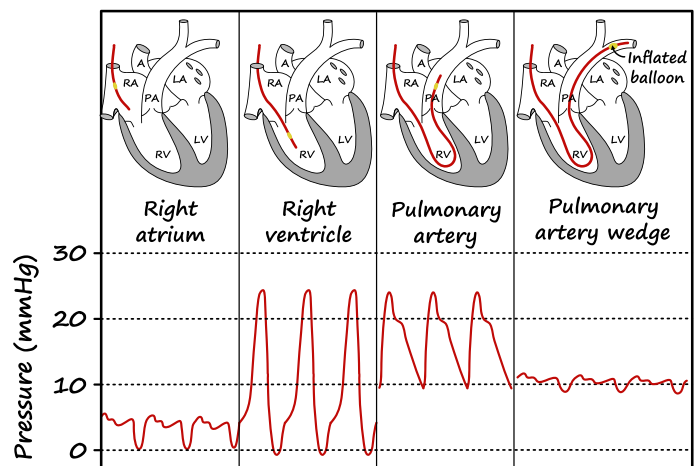
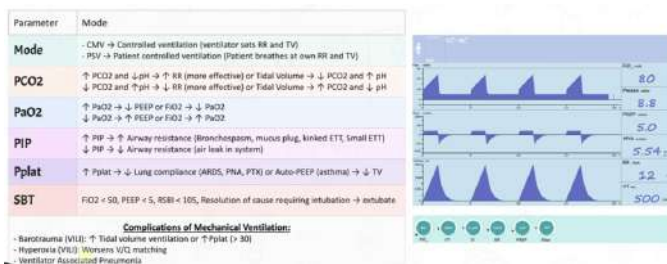


FIGURE 4. PARASTERNAL SHORT AXIS SHOWING NORMAL LVEF & DIASTOLIC FUNCTION

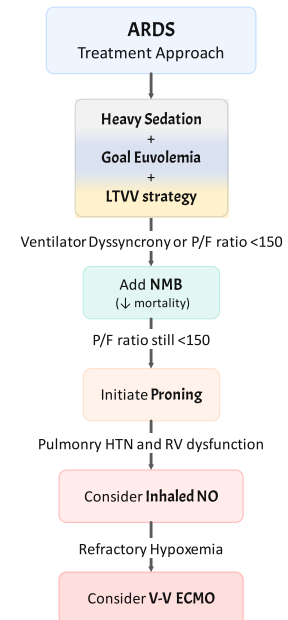
3. Obtain Right Heart Catheterization (RHC)

- **Purpose:**
 - Definitively assess for evidence of cardiogenic pulmonary edema
- **Findings in ARDS**
 - PCWP < 18 mmHg helps support diffuse infiltrates that are not from cardiogenic pulmonary edema and suggests ARDS



Treatment of ARDS

Type of Treatment	Goal	Additional Comments
Fluid Conservative Therapy	↓ Pulmonary Edema	Aim for Euvolemia
Mechanical Ventilation	↓ TV (< 6cc/kg) ↑ PEEP	↓ TV → ↓ VILI → ↓ mortality ↑ PEEP → ↓ Atelectrauma → ↓ mortality
Paralysis via NMB	↓ Ventilator Dyssynchrony	P/F Ratio < 150 → NMB (↓ mortality)
Sedation/Analgesia		Needed if on NMB
Prone	↓ Dependent atelectasis	P/F Ratio < 150 → Prone (↓ mortality)
Inhaled Pulmonary Vasodilators	↑ Oxygenation (refractory hypoxemia)	Good if Pulmonary HTN present
V-V ECMO		No good trials to suggest benefits



A. TREAT THE CAUSE OF ARDS

1. Pneumonia, Aspiration, and Sepsis

● **Treatment:**

- Antibiotics for bacterial infections
 - Vancomycin and Piperacillin-tazobactam empirically.
- Narrowly based on sputum/blood cultures

B. SUPPORTIVE CARE OF ARDS

1. Invasive Oxygen Supplementation

● **Types of Supplementation:**

- Intubation and mechanical ventilation

● **Indications:**

- Hypoxemia refractory to non-invasive ventilation (e.g. HFNC)

● **Modes of Ventilation:**

- Controlled mechanical ventilation (CMV)
 - Used in patients who require diaphragmatic rest, allowing the ventilator to do the work

● **Purpose:**

- Improve oxygenation

- Use of FiO₂
 - ↑ FiO₂ in hypoxia
- Use of PEEP
 - ↑ PEEP in hypoxia to reduce atelectrauma (VILI)

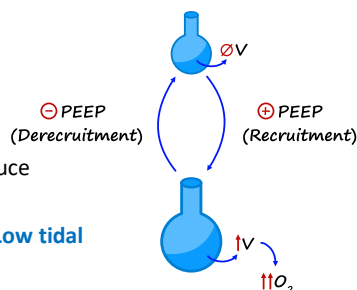
- Reduce risk of mortality via Low tidal volume ventilation (LTVV)

- Use of Tidal volume and RR
 - ↓ TV in ARDS to reduce volutrauma (VILI)
 - ↑ RR to allow clearance of CO₂ but can allow permissive hypercapnia

● **Monitoring:**

- Monitor Tidal Volumes (TV):
 - ↑ TV (> 6cc/kg of IBW) → ↑ Risk of volutrauma in ARDS
- Monitor Plateau pressures (Pplat):
 - ↑ Pplat (> 30) in ARDS → Assess for increased risk of barotrauma in ARDS
- Monitor the P/F ratio on ABG for the progression of ARDS

Effect of PEEP



2. Heavy Sedation and Analgesia

● **Types of Supplementation:**

- Sedation:

- Propofol infusion
- Midazolam infusion

- Analgesia:

- Fentanyl infusion
- Dilaudid infusion

● **Indications:**

- Worsening Hypoxemia while on LTVV causing ventilator dyssynchrony

● **Purpose:**

- Hypoxemia while on LTVV causing ventilator dyssynchrony and worsening P/F ratio
- These agents **inhibit the respiratory center**, facilitating mechanical ventilation by preventing the patient from resisting the ventilator's settings, particularly when they instinctively attempt to take deeper breaths than the machine permits

● **Monitoring:**

- Monitor the ventilator for any dyssynchrony and titrate the infusions to the goal of vent synchrony
 - Ventilator alarms signal **high breathing frequency** or **high tidal volumes** along with **hypoxia** indicates **dyssynchrony**
- Monitor the P/F ratio on ABG for the progression of ARDS



3. Paralysis

- **Types of Supplementation:**
 - **Neuromuscular Blockade (NMB):**
 - **Cisatracurium** infusion
 - Combined with sedation and analgesia
- **Indications:**
 - Worsening Hypoxemia while on LTVV → Refractory to heavy sedation and analgesia with P/F ratio < 150
- **Purpose:**
 - Hypoxemia while on LTVV and heavy sedation/analgesia causing ventilator dyssynchrony and worsening P/F ratio < 150
 - **NMBs paralyze the diaphragm and intercostals** → Allow the ventilator to do the work, preventing the patient from resisting the ventilator's settings, particularly when they instinctively attempt to take deeper breaths than the machine permits
- **Monitoring:**
 - Monitor the ventilator for any dyssynchrony and titrate the infusions to the goal of vent synchrony
 - Ventilator alarms signal high breathing frequency or high tidal volumes along with hypoxia indicates dyssynchrony
 - Monitor the P/F ratio on ABG for the progression of ARDS

4. Prone Positioning

- **Indications:**
 - **Worsening Hypoxemia** while on LTVV → Refractory to heavy sedation, analgesia, and NMB with P/F ratio < 150
- **Purpose:**
 - Hypoxemia while on LTVV and heavy sedation, analgesia, and NMB causing ventilator dyssynchrony and worsening P/F ratio < 150
 - Proning → ↓Compression of the posterior bases of lungs → ↓Dependent atelectasis → Improves V/Q matching
- **Monitoring:**
 - Monitor for reduction in FiO₂, PEEP, and improvement in oxygen saturation as well as improving CXR
 - Usually, **16 hours of lying prone and 8 hours of lying supine**
 - Monitor the P/F ratio on ABG for the progression of ARDS



Supine



Prone

5. Inhaled Pulmonary Vasodilators

- **Types of Supplementation:**
 - **Inhaled Nitric Oxide (iNO)**
- **Indications:**
 - **Worsening Hypoxemia** while on LTVV refractory to heavy sedation, analgesia, and NMB with P/F ratio < 150 and presence of pulmonary HTN or RV dysfunction on echocardiogram
- **Purpose:**
 - Worsening Hypoxemia while on LTVV refractory to heavy sedation, analgesia, NMB with P/F ratio < 150, and presence of pulmonary HTN or RV dysfunction on echocardiogram
 - iNO → Pulmonary vasodilation → Improves perfusion to well-ventilated alveoli → Improves V/Q matching with pulmonary vasodilation → **Less stress on the right heart**
- **Monitoring:**
 - Monitor **methemoglobin levels** to avoid methemoglobinemia with prolonged infusions
 - Monitor the P/F ratio on ABG for the progression of ARDS

6. Veno-Venous Extracorporeal Membrane Oxygenation (V-V ECMO)

- **Indications:**
 - **Refractory Hypoxemia** despite LTVV on heavy sedation, analgesia, NMB, and iNO with P/F ratio < 150
- **Purpose:**
 - Refractory Hypoxemia despite LTVV on heavy sedation, analgesia, NMB, and iNO with P/F ratio < 150
 - **V-V ECMO takes over the job of the lungs** by oxygenating the blood and removing CO₂ → This allows time for the lungs to heal from such an extensive injury
- **Monitoring:**
 - Monitor for reduction in FiO₂, PEEP, and improvement in oxygen saturation, as well as improving CXR
 - Monitor the P/F ratio on ABG for the progression of ARDS
 - Monitor for ECMO complications such as bleeding or circuit thrombosis

