RESPIRATORY DISEASES: PATHOPHYSIOLOGY, MANAGEMENT, AND CARE OF THE PATIENT

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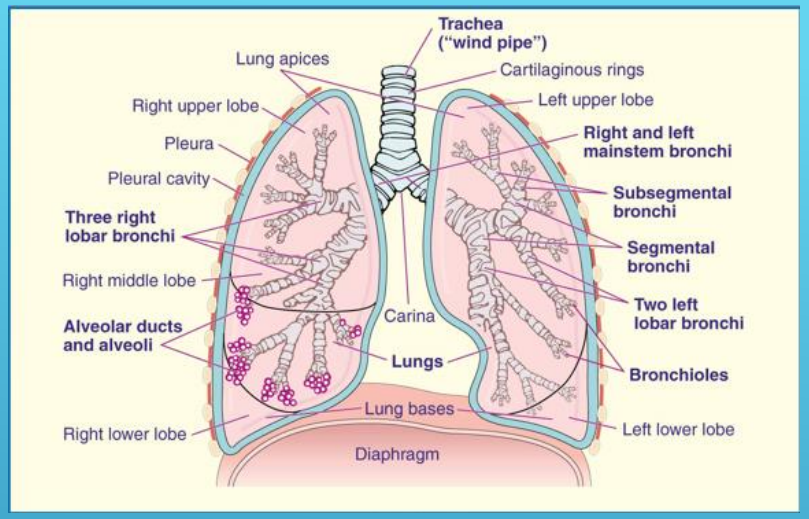
RESPIRATORY SYSTEM

- **Basic Function of Respiratory Functions:**
  - **Gas Exchange: supplies O2 and removes CO2**
  - **Arterial Blood Oxygenation: Maintain PaO2 and CaO2**
  - **Tissue Oxygenation: oxygen deliver and tissue utilization**



Basic lung function:

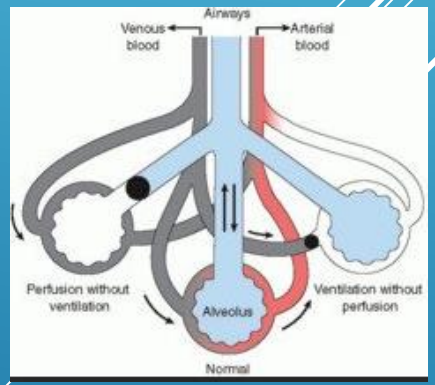
- When you breathe, air passes through your nose and mouth into your windpipe. The air then travels to your lungs' air sacs. These sacs are called alveoli
- Small blood vessels called capillaries run through the walls of the air sacs. When air reaches the air sacs, the oxygen in the air passes through the air sac walls into the blood in the capillaries.
- At the same time, carbon dioxide moves from the capillaries into the air sacs. This process is called gas exchange.

HYPOXIA

- Not to be confused with Hypoxemia which is low oxygen in the blood
- Hypoxia is a condition in which the body or region of the body is deprived of adequate oxygen supply at the tissue level.
- Without oxygen to our vital organs damage can occur in just minutes after symptoms start
- Can be general, affecting the whole body or local, affecting a region of the body

HYPOXIA

- 4 Common Causes seen in Acute Setting:
- Hypoventilation
  - occurs when ventilation is inadequate to perform needed gas exchange leading to increased carbon dioxide and respiratory acidosis due to diffusion oxygen from the alveolus to the pulmonary capillaries declines.
  - Precursor to hypoxia
  - Caused by: stroke affecting brain stem, medications, drugs, accidental/intentional overdose, opioids, hypocapnia, obesity
- VQ mismatch
  - Occurs when there is a problem with either ventilation (air going in and out of the lungs) or perfusion (oxygen and Co2 diffusion at the alveoli and the pulmonary arteries)
  - VQ ratios compare the amount of air reaching the alveoli to the amount of blood reaching the alveoli
  - Ventilation problems include: inadequate rate or tidal volume during respiration
  - Perfusion problems include: excess pulmonary dead space: emphysema, bronchitis, pneumonia, atelectasis, low pulmonary artery pressures, RVF, lack of haemoglobin availability

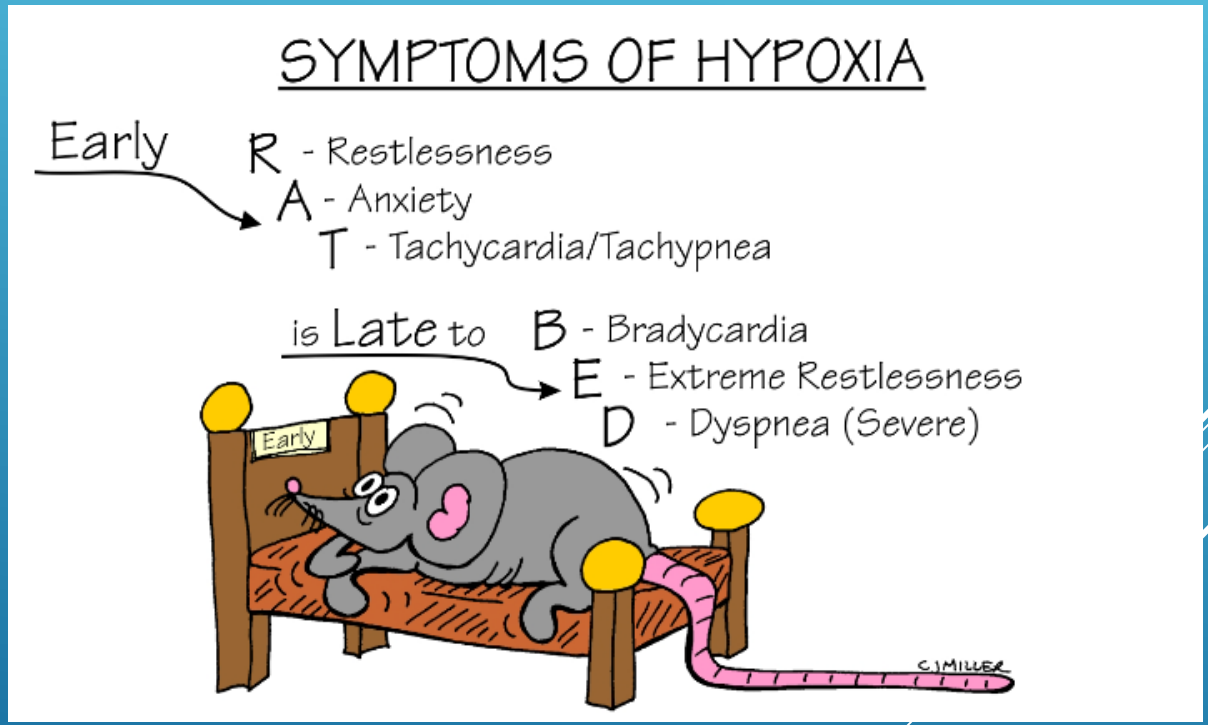


HYPOXIA

- Right to Left Shunt
  - Hypoxia is a well-recognized consequence of venous admixture resulting from right to left intracardiac shunting. Right to left shunting is usually associated with high pulmonary artery pressure or alteration in the direction of blood flow due to an anatomical abnormality of the thorax (PFO).
  - Blood passes from right side of the heart to the left without being oxygenated.
- Diffusion Limitation
  - Exists when the movement of oxygen from the alveolus to the pulmonary capillary is impaired.
  - Usually a consequence of alveolar and/or interstitial inflammation and fibrosis
  - Can be exercise induced or exacerbated
- Reduced inspired O2 tension
  - Impaired oxygen diffusion by decreased oxygen gradient from the alveolus to the artery.

HYPOXIA

- Clinical Presentation
  - Changes in color of your skin
  - Tachycardia
  - Slow heart rate
  - Cough
  - Rapid Breathing
  - Shortness of breath/Dyspnea
  - Restlessness
  - Anxiety
  - Confusion
  - Sweating
  - Wheezing

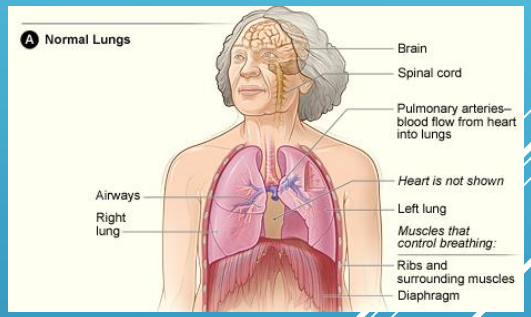


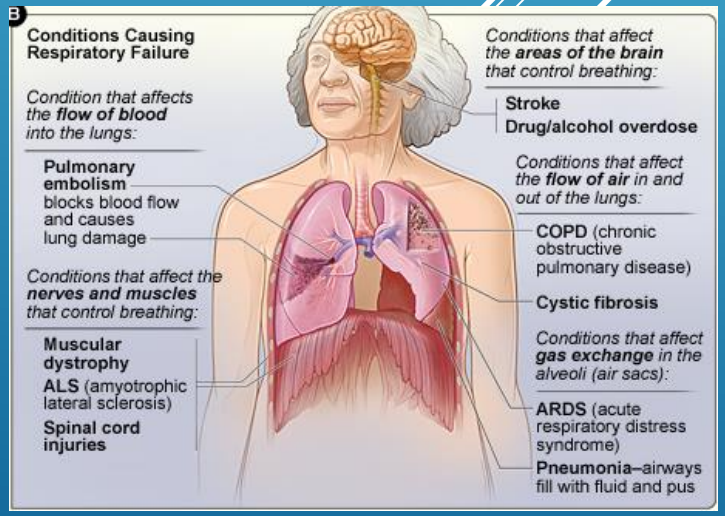
HYPOXIA

- Treatment/Management of Hypoxia
  - Oxygen: Supplemental Oxygen should be administered to patient to keep sats >90% or as ordered
  - Ventilatory support: Patients with severe respiratory distress and/or marked hemodynamic instability will need ventilatory support via HFNC, NIPPV (CPAP/BiPAP), or Intubation requiring mechanical ventilation based on severity of symptoms of hypoxia
  - Imaging and Laboratory values: CXR, ABG's, if ordered.
  - Treating cause
  - Close monitoring of patient including vitals (HR, BP, RR, SpO2) and assessments
  - When to Call the Dr
  - What to know when calling the Dr

RESPIRATORY FAILURE

- Definition:
  - Hypoxemic Respiratory Failure: Condition in which not enough oxygen passes from the lungs into the blood affecting the body's organs as the body's organs require oxygen-rich blood leading to lack of oxygen to the organs/tissues
  - Hypercapnic Respiratory Failure: Condition is which the lungs can't properly remove carbon dioxide from your blood leading to high carbon dioxide levels which can damage the organs.
  - Both types can occur simultaneously
  - Acute (short term) or Chronic (on going).
- Causes:
  - Conditions affecting nerves/muscles: muscular dystrophy, ALS, spinal cord injuries, stroke
  - Damage to tissues and ribs around lungs, injuries/trauma to chest
  - Drug or alcohol overdose
  - Lung diseases: COPD, pneumonia, ARDS, PE, CF
  - Lung injuries: inhaling harmful fumes, smoke





RESPIRATORY FAILURE

- Clinical Presentation
  - Can depend on underlying cause but common signs and symptoms include:
    - Shortness of breath
    - rapid breathing
    - Accessory muscle use
    - air hungry (feel like you can't breath in enough air)
    - cyanosis of skin, lips, fingernails
    - Confusion
    - Seizures
    - Sleepiness
    - loss of consciousness
    - arrhythmias or irregular heartbeats
    - tachycardia

RESPIRATORY FAILURE

- Treatment/Management of Respiratory Failure
  - Oxygen: Oxygen should be administered to patient with evidence of clinical significant desaturations to keep sats >90% or as ordered
  - Ventilatory support: Patients with severe respiratory distress and/or marked hemodynamic instability will need ventilatory support via HFNC, NIPPV (CPAP/BiPAP), or Intubation requiring mechanical ventilation based on severity of symptoms of respiratory failure
  - IV access: peripheral or central access to administer medications, IV fluids, vasopressors if required
  - Arterial access: based on clinical status and need for invasive hemodynamic monitoring
  - Imaging and Laboratory values: CXR, ABG's, if ordered.
  - Treating cause
  - Close monitoring of patient including vitals and assessments

COPD

- Chronic obstructive pulmonary disease (COPD) is a common respiratory condition characterized by airflow limitation.
- It affects more than 5 percent of the population and is associated with high morbidity and mortality.
- It is the third-ranked cause of death in the United States, killing more than 120,000 individuals each year.
- As a consequence of its high prevalence and chronicity, COPD causes high resource utilization with frequent clinician office visits, frequent hospitalizations due to acute exacerbations, and the need for chronic therapy (eg, supplemental oxygen therapy, medication)

COPD

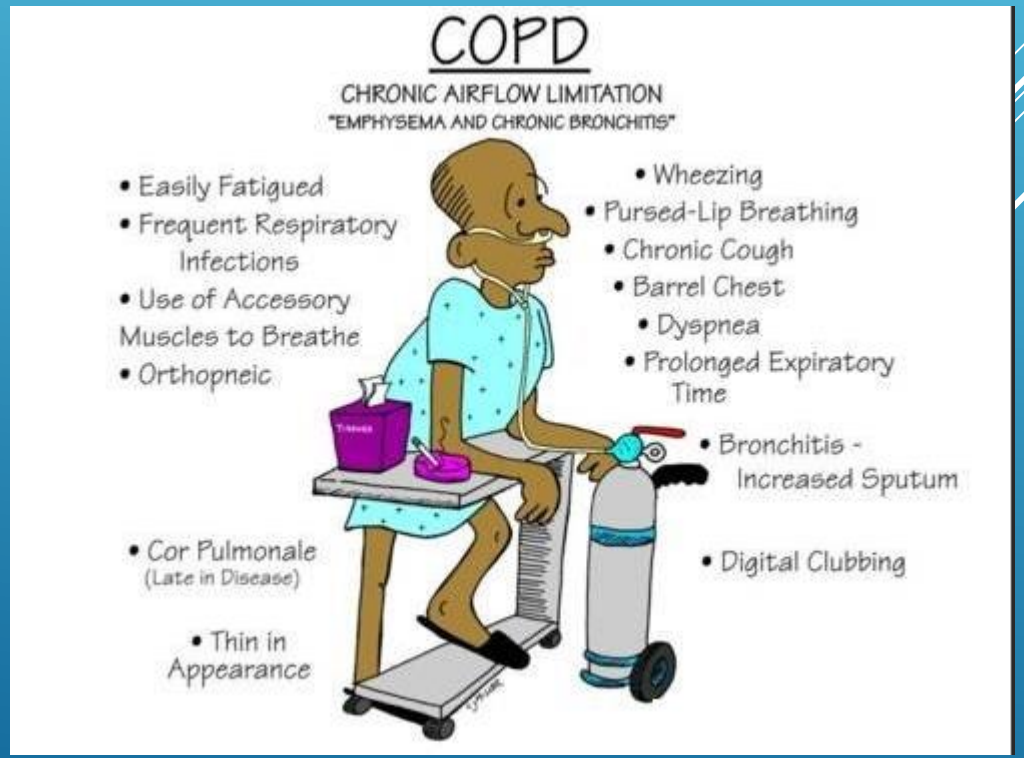
- COPD is a progressive lung disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.
- The chronic airflow limitation that characterizes COPD is caused by a mixture of small airways disease (eg, obstructive bronchiolitis) and parenchymal destruction (emphysema).
- Chronic inflammation causes structural changes, small airways narrowing, and destruction of lung parenchyma. A loss of small airways may contribute to airflow limitation and mucociliary dysfunction, a characteristic feature of the disease."
- CAUSES:
  - Smoking
  - exposure to fumes or organic or inorganic dusts.
  - exposures including passive smoke, biomass fuel use, air pollution, fumes, chemicals
  - Asthma increases risk
  - Genetics

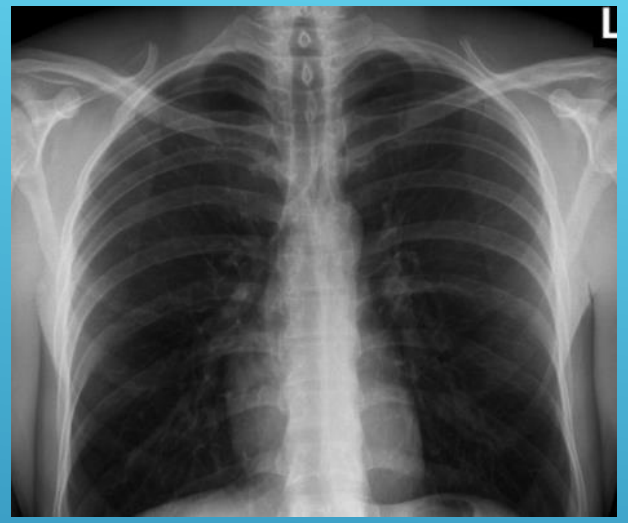
COPD

- **Chronic bronchitis** — Chronic bronchitis is defined as a chronic productive cough for three months in each of two successive years in a patient in whom other causes of chronic cough (eg, bronchiectasis) have been excluded. It may precede or follow development of airflow limitation
- **Emphysema** — Emphysema is a pathological term that describes some of the structural changes sometimes associated with COPD. These changes include abnormal and permanent enlargement of the airspaces distal to the terminal bronchioles that is accompanied by destruction of the airspace walls, without obvious fibrosis (ie, there is no fibrosis visible to the naked eye
- **Refractory (non-reversible) asthma-** Asthma is a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role. The chronic inflammation is associated with airway responsiveness that leads to recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning Patients with asthma whose airflow obstruction does not remit completely are considered to have).

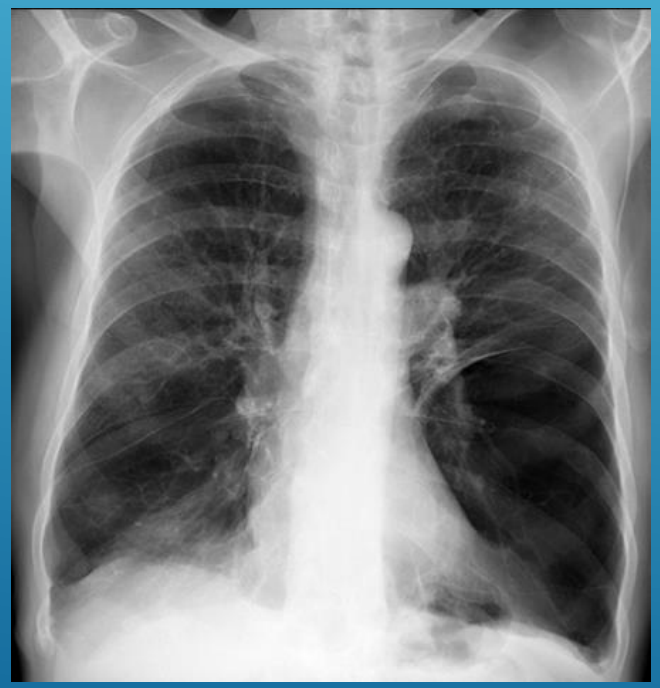
COPD

- **Clinical Presentation**
  - **3 cardinal symptoms of COPD include: dyspnea, chronic cough, sputum production**
  - **Other symptoms include:**
  - Frequent respiratory infections
  - Blueness of the lips or fingernail beds (cyanosis)
  - Fatigue
  - Wheezing

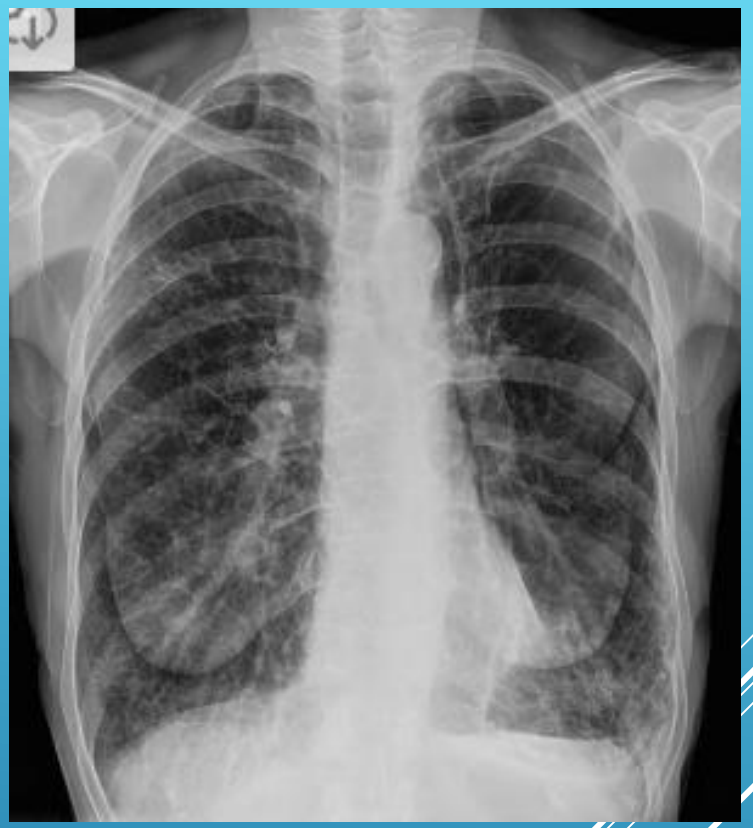




Normal CXR



COPD



COPD

COPD

- **Associations**
- **Respiratory infections.** People with COPD are more likely to catch colds, the flu and pneumonia. Any respiratory infection can make it much more difficult to breathe and could cause further damage to lung tissue. An annual flu vaccination and regular vaccination against pneumococcal pneumonia can prevent some infections.
- **Heart problems.** For reasons that aren't fully understood, COPD can increase your risk of heart disease, including heart attack. Quitting smoking may reduce this risk.
- **Lung cancer.** People with COPD have a higher risk of developing lung cancer. Quitting smoking may reduce this risk.
- **High blood pressure in lung arteries.** COPD may cause high blood pressure in the arteries that bring blood to your lungs (pulmonary hypertension).
- **Depression.** Difficulty breathing can keep you from doing activities that you enjoy. And dealing with serious illness can contribute to development of depression. Talk to your doctor if you feel sad or helpless or think that you may be experiencing depression.

COPD

- Treatment/management of COPD
  - Medication to prevent exacerbations
    - Bronchodilators
    - Anti-Inflammatory Drugs: Steroids
    - Combination inhalers/nebulizers
    - Antibiotics
    - Vaccinations: pneumococcal
  - Supplemental Oxygen
    - To support oxygen saturation and work of breathing
    - NC, HFNC, NIPPV (CPAP/BiPAP), intubation/mechanical ventilation my be required for exacerbations
  - Pulmonary rehab/Palliative care
  - Smoking Cessation
  - Surgery

ARDS

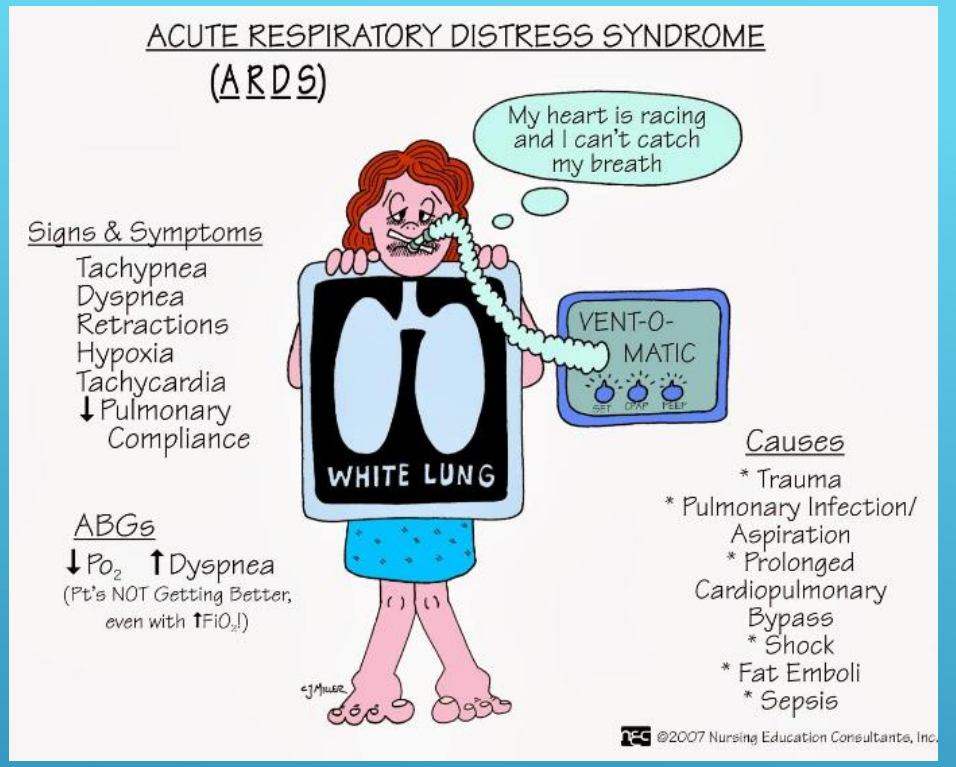
- Healthy lungs regulate the movement of fluid to maintain a small amount of interstitial fluid and dry alveoli. This is interrupted by lung injury, causing excess fluid in both the interstitium and alveoli. Consequences include impaired gas exchange, decreased compliance, and increased pulmonary arterial pressure
- Acute respiratory distress syndrome (ARDS) is a consequence of an alveolar injury producing diffuse alveolar damage. The injury causes release of pro-inflammatory cytokines such as tumor necrosis factor, interleukin. These cytokines recruit neutrophils to the lungs, where they become activated and release toxic mediators that damage the capillary endothelium and alveolar epithelium.
- Damage to the capillary endothelium allows protein to escape from the vascular space. The oncotic gradient that favors resorption of fluid is lost and fluid pours into the interstitium, overwhelming the lymphatics. The ability to upregulate alveolar fluid clearance may also be lost.
- Increase in interstitial fluid, combined with damage to the alveolar epithelium, causes the air spaces to fill with bloody, proteinaceous edema fluid and debris from degenerating cells. In addition, functional surfactant is lost, resulting in alveolar collapse

ARDS

- **Consequences** — Lung injury has numerous consequences including impairment of gas exchange, decreased lung compliance, and increased pulmonary arterial pressure.
  - **Impaired gas exchange** – Impaired gas exchange in ARDS is primarily due to ventilation-perfusion mismatching: physiologic shunting causes hypoxemia, while increased physiologic dead space impairs carbon dioxide elimination [
  - **Decreased lung compliance** – Decreased pulmonary compliance is one of the hallmarks of ARDS. It is a consequence of the stiffness of poorly or nonaerated lung, rather than the pressure-volume characteristics of residual functioning lung units [
  - **Pulmonary hypertension** – Pulmonary hypertension occurs in up to 25 percent of patients with ARDS who undergo mechanical ventilation. Causes include hypoxic vasoconstriction, vascular compression by positive airway pressure, parenchymal destruction, airway collapse, hypercarbia, and pulmonary vasoconstrictors.

ARDS

- Causes/Predisposing factors
  - Sepsis
  - Aspiration
  - Pneumonia
  - Severe Trauma
  - Massive Transfusion
  - Transfusion-related acute lung injury
  - Drugs and Alcohol, smoking
  - Prolong cardiopulmonary bypass
  - Thoracic surgery
  - Pneumonectomy
  - Obesity
  - Embolism
  - Acute pancreatitis
  - Blood type A
  - Near drowning

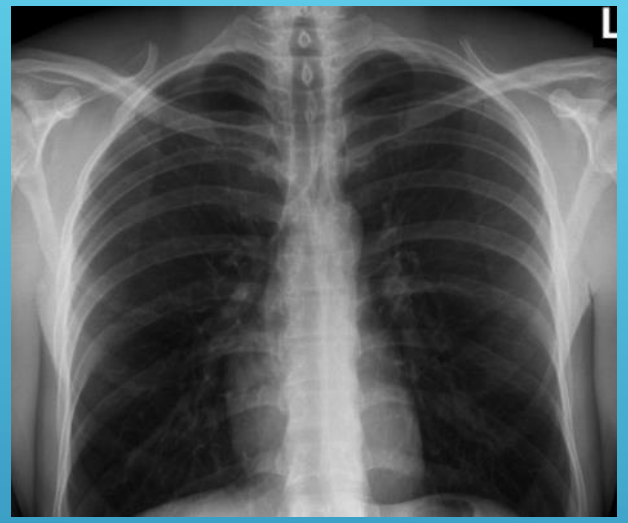


Signs and symptoms:

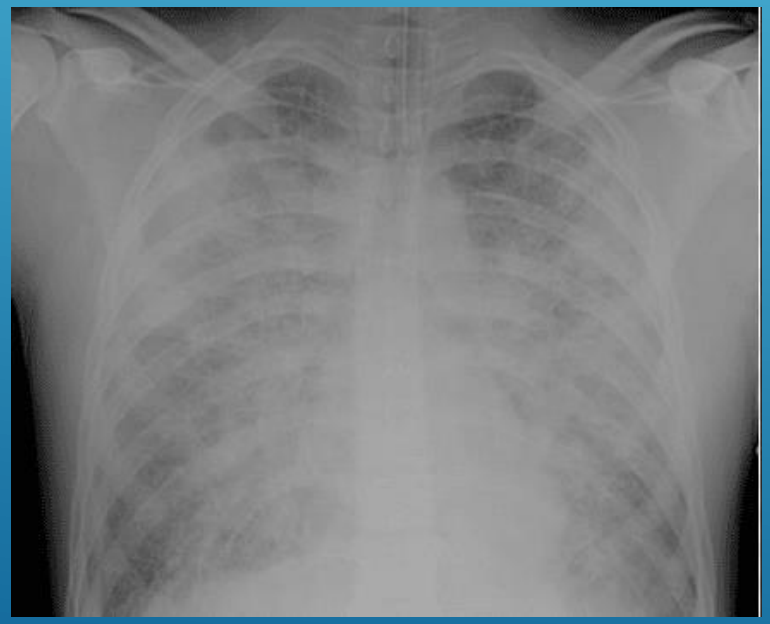
- Tachypnea
- Dyspnea
- Increased Work of Breathing
- Hypoxia
- Decreased PO2
- Decreased Pulmonary Compliance
- Tachycardia
- Hypotension
- Symptoms of what is causing ARDS

ARDS

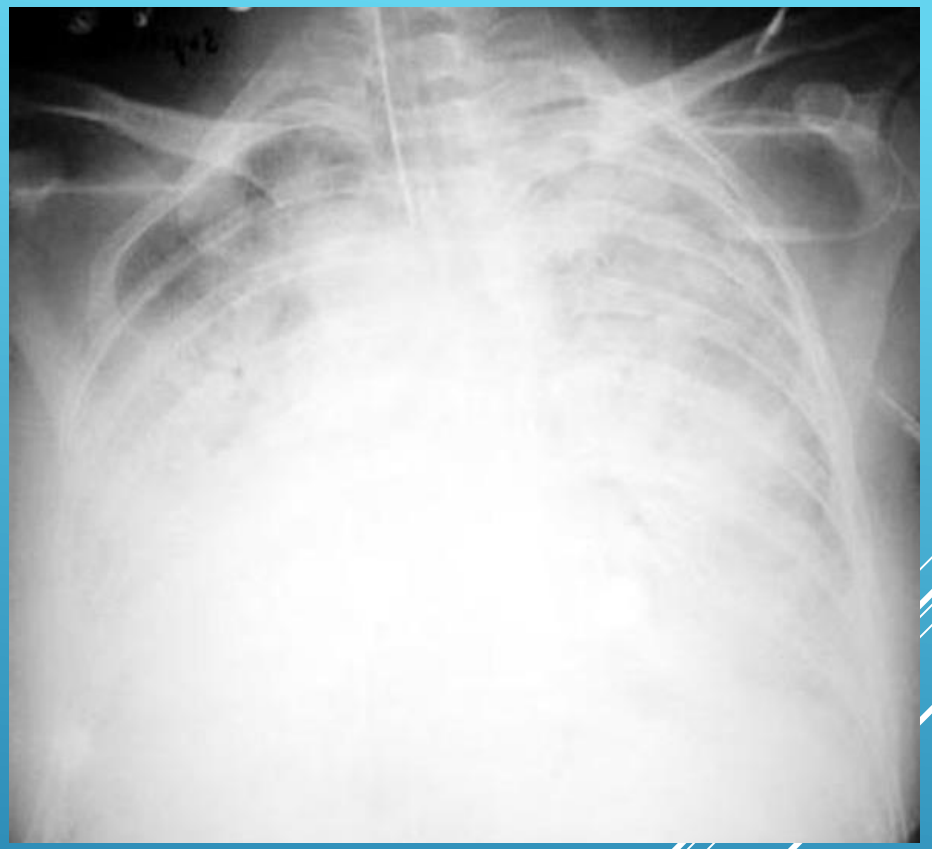
- **Clinical diagnosis (Berlin definition)** — ARDS can be diagnosed once cardiogenic pulmonary edema and alternative causes of acute hypoxemic respiratory failure and bilateral infiltrates have been excluded. The Berlin Definition of ARDS requires that all of the following criteria be present for diagnosis
  - Respiratory symptoms must have begun within one week of a known clinical insult, or must have new or worsening symptoms during the past week.
  - Bilateral opacities must be present on a chest radiograph or computed tomographic (CT) scan. These opacities must not be fully explained by pleural effusions, lobar collapse, lung collapse, or pulmonary nodules.
  - The patient's respiratory failure must not be fully explained by cardiac failure or fluid overload. An objective assessment (eg, echocardiography) to exclude hydrostatic pulmonary edema I
  - A moderate to severe impairment of oxygenation must be present, as defined by the ratio of arterial oxygen tension to fraction of inspired oxygen (PaO_2/FiO_2). The severity of the hypoxemia defines the severity of the ARDS



Normal CXR



ARDS



ARDS

ARDS

- **ARDS tends to progress through three relatively discrete pathologic stages**
- **Early exudative stage (DAD)** – The early exudative stage during the first 7 to 10 days is characterized by DAD. DAD is a nonspecific reaction to lung injury from a variety of causes. It is characterized by interstitial edema, acute and chronic inflammation, type II cell hyperplasia, and hyaline membrane formation Further details are provided separately.
- **Fibroproliferative stage** – After approximately 7 to 10 days, a proliferative stage develops, characterized by resolution of pulmonary edema, proliferation of type II alveolar cells, squamous metaplasia, interstitial infiltration by myofibroblasts, and early deposition of collagen. It is unknown how long this phase lasts but is probably in the realm of two to three weeks.
- ●**Fibrotic stage** – Some patients progress to a fibrotic stage, characterized by obliteration of normal lung architecture, fibrosis, and cyst formation. The degree of fibrosis ranges from minimal to severe.

ARDS

- Treatment/Management of the patient
  - **SUPPORTIVE CARE**
  - **Determine Cause and Treat the Cause**
  - Oxygen Supplementation
    - High Oxygen Concentration Administration
    - Intubation/Mechanical Ventilation
    - administer high PEEP
  - Fluids
  - Prone Positioning
  - Nutritional support
  - Medications
    - Antibiotics, VTE prophylaxis, sedatives, pressors, paralytics
  - ECMO

PNEUMONIA

- Pneumonia is a bacterial, viral, or fungal infection of one or both sides of the lungs that causes the air sacs, or alveoli, of the lungs to fill up with fluid or pus.
  - Can be community acquired, hospital acquired, or aspiration pneumonia
  - Associated with considerable morbidity and mortality especially among those patients who have pre-existing comorbidities

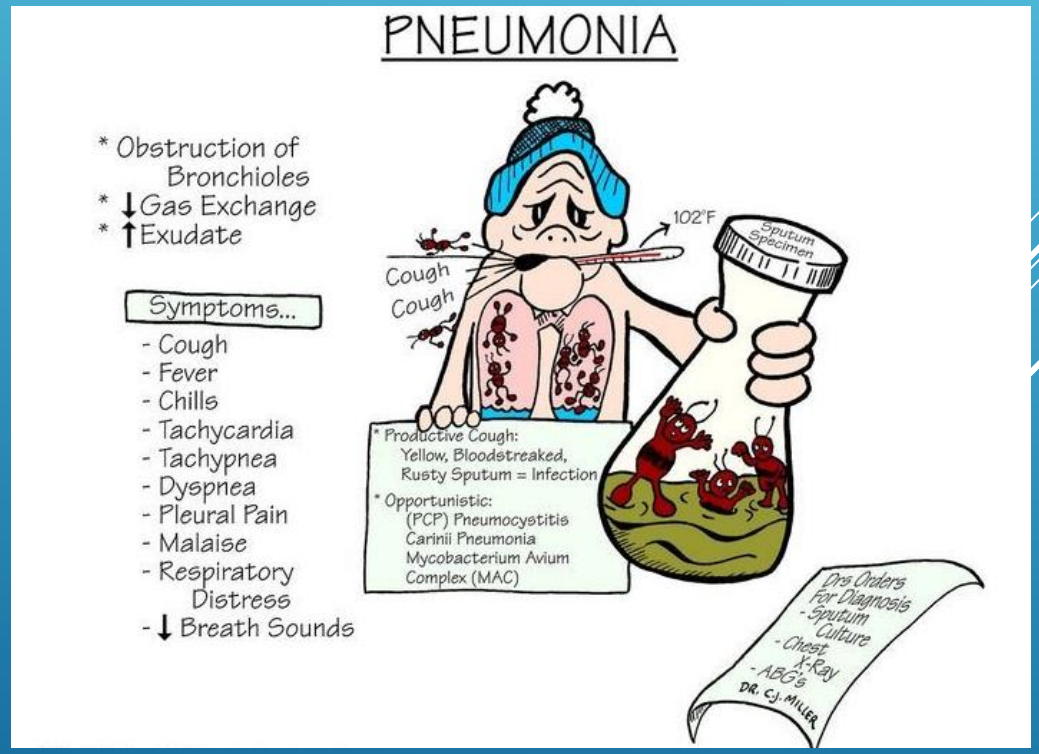
- The lungs are constantly exposed to particulate material and microbes that are present in the upper airways and, by microaspiration, enter the lower respiratory tract and cause infection.
  - Can be Lobar or widespread in the lungs
- oxygen may have trouble reaching your blood. If there is too little oxygen in your blood, your body cells can't work properly. Because of this and the risk of the infection spreading through the body, pneumonia can cause death.

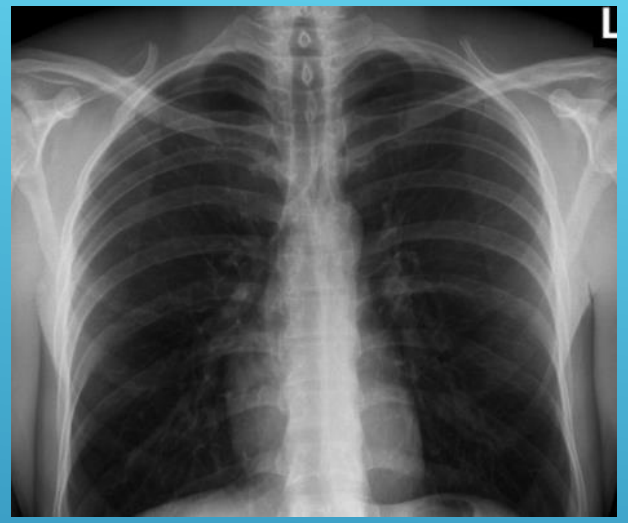
PNEUMONIA

- Risk Factors:
  - Older age (marked increase in pneumonia incidence among adults >65 years old)
  - Chronic lung diseases and/or other disorders that impair airway clearance (COPD, PE, Lung Cancer,
  - Conditions that increase risk of aspiration of stomach contents or upper airway secretions (seizure, stroke, anesthesia, drug or alcohol intoxication)
  - Immunocompromising conditions (DM, heart failure, immunosuppressive medications, HIV
  - Metabolic disorders (malnutrition, uremia, hypoxemia, acidosis
  - Lifestyle factors (smoking, alcohol consumption, toxic inhalations, homelessness
  - Intubation/Bronchoscopy
  - Viral Respiratory tract infections (influenza)

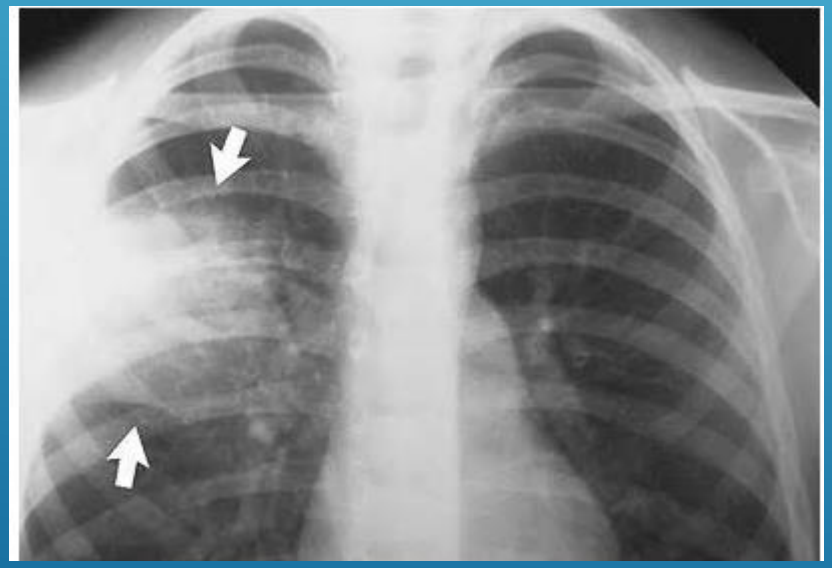
PNEUMONIA

- Clinical Presentation
  - Signs and Symptoms can include:
    - Cough: typically productive
    - Shortness of Breath
    - Low oxygen saturation
    - Fever
    - Chills
    - Sharp or stabbing chest pain
    - Loss of appetite
    - Fatigue
    - Confusion

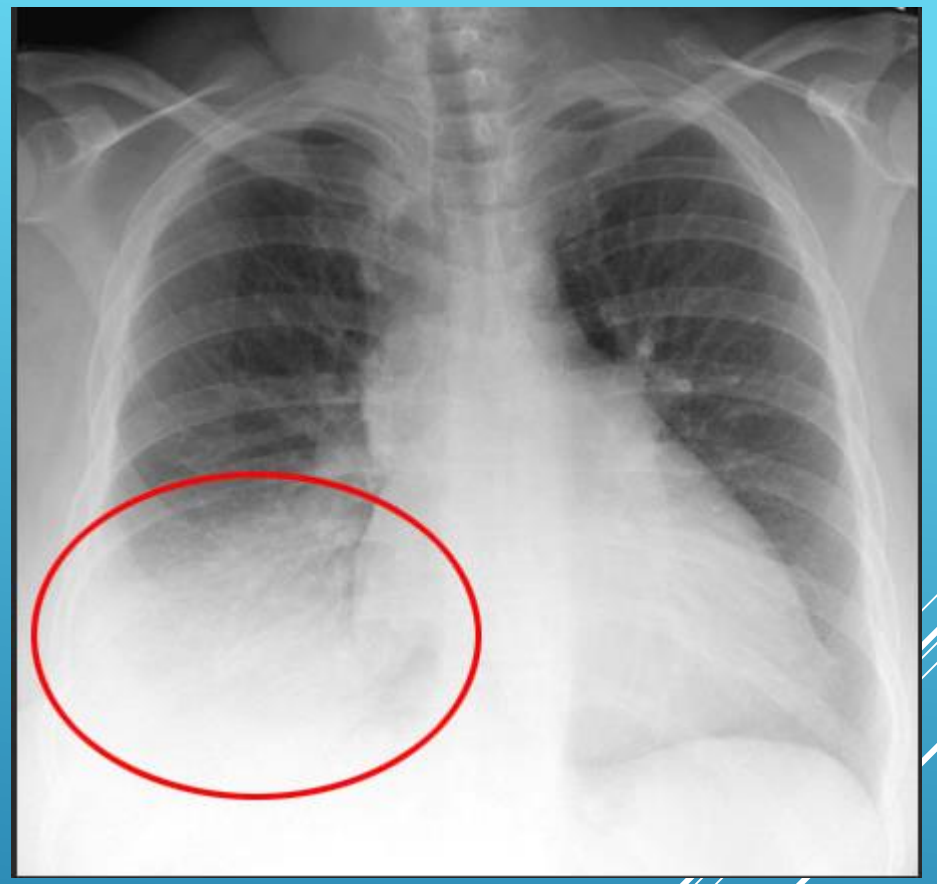




Normal CXR



Pneumonia In Right Middle Lobe



Pneumonia in Right Lower Lobe

PNEUMONIA

- Treatment/Management of the patient
  - Goal is to treat the infection and prevent complications
  - Treatment includes
    - Antibiotics
    - Supportive Care:
      - Oxygen supplementation
      - NC, HFNC, NIPPV, intubation/mechanical ventilation
      - IV fluids, vasopressors, fever reducers, etc.

RESPIRATORY/VENT SETTINGS AFFECTS ON HEMODYNAMICS (RESPIRATORY PUMP MECHANISM

- The use of a positive end-expiratory pressure (PEEP) can improve oxygenation and prevent atelectasis, although this method can cause important hemodynamic side effects
- These hemodynamic effects are due to increased airway pressure that is transferred to the intrapleural space, increasing the intrathoracic pressure, which decreases venous return to the heart. Cardiac output is significantly reduced with high PEEP levels which in turn precludes the improvement effects on blood oxygenation.
- **Positive pressure ventilation affects preload, afterload and ventricular compliance**. The net effect in most situations is a **decrease in cardiac output**

RESPIRATORY/VENT SETTINGS AFFECTS ON HEMODYNAMICS (RESPIRATORY PUMP MECHANISM

- **Effects on Preload:**
  - PEEP is essentially end-expiratory positive intrathoracic pressure. This influences the return of venous blood to the heart.
  - In a patient who is slightly underfilled, venous return is still adequate in the absence of PEEP, because the CVP is still higher than the (mostly) negative thoracic pressure.
  - with inspiration, that pressure becomes even more negative, and blood is actively sucked into the chest.
  - The PEEP is higher than the CVP, and venous return is impaired. On echo, the right atrium and IVC will appear collapsed.
  - This decreased preload will result in a decrease in cardiac output.

RESPIRATORY/VENT SETTINGS AFFECTS ON HEMODYNAMICS (RESPIRATORY PUMP MECHANISM

- **Effects on Afterload:**
  - Right Ventricular Afterload
    - In order to function normally, the right ventricle needs to generate enough pressure to overcome the sum of PA pressure and PEEP
    - Consequently, if the PEEP is too high, the right ventricle will begin to fail due to increased afterload
    - The fact that by this stage preload is also decreased doesn't help. The net effect is to cause greatly diminished cardiac output.
    - if there is a decrease in right-sided cardiac output, there will also be a decrease in left sided cardiac output
  - Left Ventricular Afterload
    - putting positive pressure on the intrathoracic aorta causes a pressure gradient to develop between this vascular compartment and the extrathoracic circulatory system (which is under atmospheric pressure). The resulting gradient favours the flow of blood into the systemic circulation, also decreasing LV workload.

RESPIRATORY/VENT SETTINGS AFFECTS ON HEMODYNAMICS (RESPIRATORY PUMP MECHANISM

- **Effects of PEEP on left ventricular compliance**
  - Left ventricular compliance is decreased by a leftward shift of the interventricular septum.
    PEEP, by causing a dilatation of the right ventricle (as a result of increased right ventricular afterload) supposedly causes the interventricular septum to bulge into the left ventricle during diastole (because the LV end-diastolic pressure ends up being less than the RV end-diastolic pressure).
  - As a result, left ventricular preload is decreased even more, and cardiac output decreases.

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