

CMC Certification Review

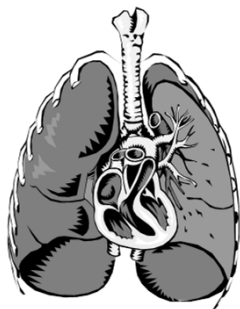
Pulmonary Physiology and Pathophysiology: Making the Link to Clinical Practice

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Physiology of Pulmonary System



- Ventilation and Perfusion
- Diffusion
- Relationship of Oxygen to Hemoglobin
- Oxygen Delivery to the Tissues
- Cellular Respiration

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Ventilation

- **Definition:** The movement of air between the atmosphere and alveoli and the distribution of air within the lungs to maintain appropriate concentrations of oxygen and carbon dioxide in the blood
- Process of ventilation occurs through inspiration and expiration
- Pressure difference between airway opening and alveoli
 - Result: Negative pressure breathing

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Ventilation

- Minute ventilation (V_E) = Total volume of air expired in one minute
 - RESPIRATORY RATE X TIDAL VOLUME
 - Normal minute ventilation = $12 \times 500 \text{ ml} = 6000\text{ml}$
 - Note: (hypoventilation can occur with normal respiratory rate)

Clinical Application: How do you correct ventilation problems?

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Alveolar Ventilation (V_A)

- $V_A = V_T -$ anatomical dead space
- Approximately 350 ml per breath

Anatomical dead space:

Walls are too thick for diffusion
Mixed venous blood not present

Approximately 1 cc per pound of ideal body weight

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Respiratory Anatomy

Conducting Airways

- Nose
- Pharynx
- Larynx
- Trachea
- Right and Left Bronchi
- Non-Respiratory Bronchi

Gas Exchange Airways

- Respiratory Bronchioles
- Alveolar Ducts
- Alveoli

V_A : Alveolar ventilation

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Alveolar Cells

- Type I (**make up 90% of alveolar surface area**)
 - Squamous epithelium
 - Adapted for gas exchange
 - Prevents fluid from entering alveoli
 - Easily injured
- Type II
 - Can generate into Type 1 cells
 - Produces surfactant (**allows alveoli to remain inflated at low distending pressures by decreasing surface tension, decreases work of breathing, detoxifies inhaled gases**)
 - Lipoprotein (phospholipid)
 - Hypoxemia / hypoxia may lead to decreased production or increased destruction
 - Metabolically active
- Alveolar Macrophages
 - Phagocytosis

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Ventilation

- Work of Breathing Affected by:
 - Compliance (elastic work of breathing)
 - Lungs distend most easily at low volumes
 - Compliance is opposite of elastic recoil
- Airway Resistance (flow resistance / resistive work of breathing)
 - Total resistance is comprised of tissue (20%) and airway resistance (80%)
 - Directly proportional to viscosity and length of tube / indirectly proportional to radius
 - Small airway resistance offset by numerous small airways (greatest resistance normally in medium bronchi)

Resistive work of breathing greatest during forced expiration.



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Assessment of Ventilation

- Efficiency and effectiveness of ventilation is measured by PCO_2 (inversely related to V_A)
 - $PCO_2 > 45$ mm Hg indicates alveolar hypoventilation *
 - $PCO_2 < 35$ mm Hg indicates alveolar hyperventilation

Note: Only one physiologic reason for increased $PaCO_2$.

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More on Ventilation

- Normal ventilation on room air results in an alveoli with a partial pressure of oxygen of approximately 100 mmHg.

Partial pressure of O_2 100 (104) mmHg

Inspired gas PIO_2 149 mm Hg.

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Untreated Alveolar Hypoventilation

Untreated alveolar hypoventilation will lead to hypoxemia. The hypoxemia is secondary to uncorrected alveolar hypoventilation.

In acute respiratory failure a blood gas is necessary to assess the PaCO₂ to determine if inadequate ventilation contributed to the hypoxemia.

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Conditions Altering Ventilation

- Non Pulmonary Conditions
- Pulmonary Conditions
 - Decreased Compliance
 - Decreased surfactant production
 - Atelectasis
 - Obesity / musculoskeletal disorders
 - Restrictive disorders (fibrosis, interstitial lung disease)
 - Increased Resistance
 - Narrowing of airways (secretions / bronchospasm)
 - Obstructive Disorders
 - Asthma
 - Emphysema
 - Bronchitis
 - Foreign body causes a fixed obstruction
 - Sleep apnea can be obstructive

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Perfusion

- Definition: The movement of blood through though the pulmonary capillaries



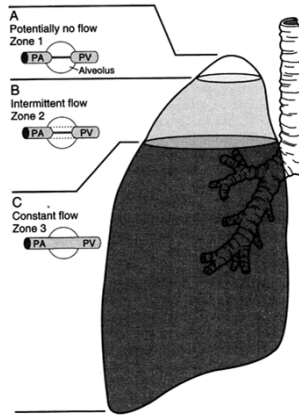
- Blood supply to lung
 - **Pulmonary blood flow**
 - Entire output of right ventricle
 - Mixed venous blood
 - Gas exchange with alveolar air into pulmonary capillaries
 - Bronchial blood flow
 - Left ventricle
 - Part of tracheal bronchial tree
 - Systemic arterial blood¹³

Perfusion Fun Facts

- Pulmonary capillaries are slightly smaller than average erythrocyte
- Gas exchange actually starts in smaller pulmonary arterial vessels that are not true capillaries (functional pulmonary capillaries)
- 280 billion capillaries supply 300 million alveoli
- Potential surface area for gas exchange is 50-100 m²
- Alveoli are completely enveloped in pulmonary capillaries
- At rest each red blood cell spends only about 0.75 seconds in the pulmonary capillary. Less time during exercise.

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Zones of Perfusion



Zone 1 increased in positive pressure ventilation and PEEP.

- Zone 1: May be no blood flow. (alveolar deadspace – no zone 1 in normal breathing)
 - Pulmonary artery systolic and diastolic pressures are < alveolar pressures
- Zone 2: Flow during systole.
- Zone 3: Flow during entire cardiac cycle.

Note: Zones are not static.

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Pulmonary Vascular Resistance

- Comparison with systemic vascular resistance
 - 1/10 of systemic vascular resistance
 - Pulmonary vascular resistance is evenly distributed between the pulmonary arteries, the pulmonary capillaries, and the pulmonary veins.
- Relationship to pulmonary artery pressures and cardiac output
 - Increase in cardiac output = Increase in PAP = Increased capillary recruitment = Decrease in PVR
- Relationship to lung volumes
 - High lung volumes pull pulmonary vessels open. Results in a decrease PVR.

- During positive pressure mechanical ventilation, both the alveolar and extra-alveolar vessels are compressed during lung inflation and PVR is increased.
- PEEP increases PVR further.

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Hypoxic Pulmonary Vasoconstriction

- Diverts blood away from poorly ventilated alveoli.
- Also occurs in response to more global hypoxia.
 - Increases pulmonary artery pressure and recruits pulmonary capillaries to improve ventilation and perfusion matching.
- Has limitations because of small amount of vascular smooth muscle in the pulmonary arteries.

- Hypoxic vasoconstriction greatly increases the workload of the right ventricle
- Increased pulmonary artery pressure may lead to pulmonary edema.

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Diffusion

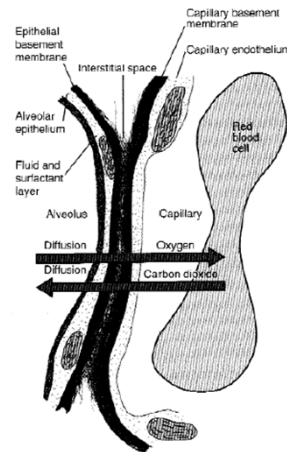
- Movement of gases between the alveoli, plasma, and red blood cells
- Net movement of molecules from an area where the particular gas exerts a high partial pressure to an area where it exerts a lower partial pressure
- Different gases each move according to their own partial pressure gradients

- ***Diffusion of oxygen from alveoli to capillary determines the patient's oxygenation status***

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Determinants of Diffusion

- **Surface Area:** negatively affected by any type of pulmonary resection; tumor, emphysema, V/Q mismatching
- **Driving pressure:** negatively affected by low inspired fraction of O₂ (smoke inhalation) or by low barometric pressure (high altitudes)
 - Barometric pressure is the sum of the pressures of all the gases it contains
- **Thickness of alveolar capillary membrane (< 1 RBC):** negatively affected by pulmonary edema or fibrosis



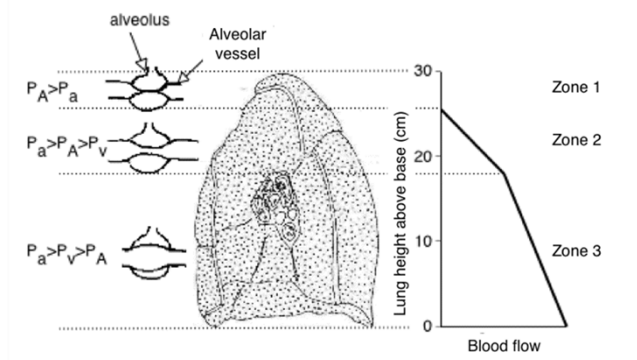
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Assessment of Diffusion

- Assessed by PaO₂ and oxygen saturation (SaO₂)
- *Clinical Application: CO₂ is 20 times more diffusible than O₂ - so a diffusion problem causing hypoxemia does not result in the same problem with CO₂ retention (hypercapnia)*
- *Clinical Application: How do you correct a diffusion problem?*

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Ventilation and Perfusion Ratios



Alveoli in upper regions have greater volume and are less compliant. Alveoli in lower parts of lung have a greater change in volume during inspiration and are considered better ventilated.

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Ventilation / Perfusion Ratio (V/Q)

■ Ventilation (V)

– Alveolar minute ventilation = 4 to 6L

■ Perfusion (Q)

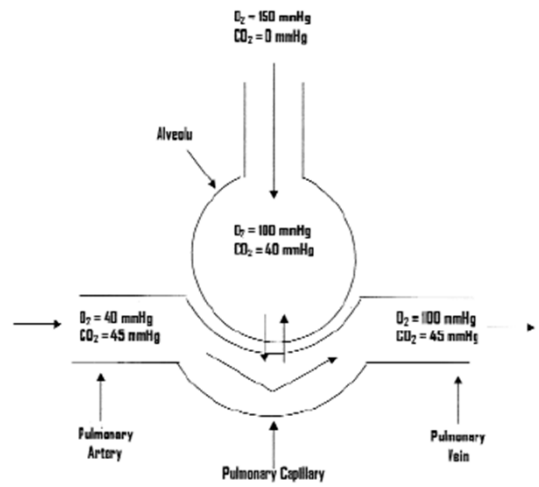
– Normal cardiac output = 5 L

Normal ventilation / perfusion ratio
(V/Q ratio) = 4/5, 5/5., or 6/5
0.8 to 1.2

Ventilation and perfusion must be matched at the alveolar capillary level

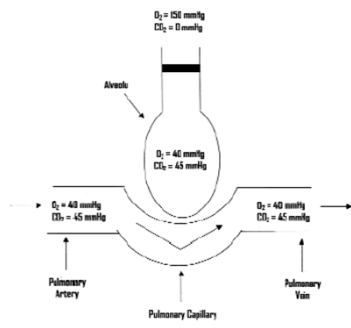
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**Normal V/Q Ratio
4/5**

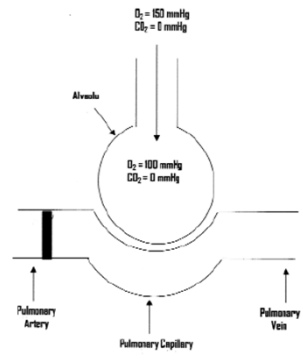


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**Decreased ventilation
to perfusion ratio
 $V/Q = < 4/5$
(Intrapulmonary Shunting)**



**Increased V/Q
Ratio
 $4 / < 5$
(Dead Space)**



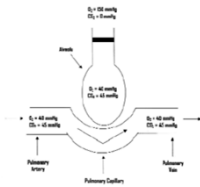
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Ventilation / Perfusion

- ▼ In ventilation perfusion ratio

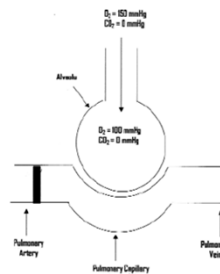
- Alveolar PO₂ will fall
- Alveolar PCO₂ will rise

- Gases in alveoli equilibrate with venous blood.



- ▲ In ventilation perfusion ratio

- Alveolar PO₂ will rise
- Alveolar PCO₂ will fall



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Decreased V/Q Ratio: Intrapulmonary Shunting

- Intrapulmonary shunt occurs when there is significant alveolar hypoventilation in relation to normal perfusion (Example: Poorly ventilated alveoli in ARDS)

- V/Q ratio < 0.8

Result

- Poorly oxygenated blood returns to left side of heart resulting in low PaO₂ and SaO₂ (oxygenation problem)
- KEY Assessment Finding compared to simple diffusion problem: Response to O₂ therapy

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Causes of Decreased V/Q Ratio

- Causes of non uniform ventilation
 - Uneven resistance to airflow
 - Collapsed airways (Emphysema)
 - Bronchoconstriction (Asthma)
 - Inflammation (Bronchitis)
 - Non-uniform compliance throughout the lung
 - Fibrosis
 - Pulmonary vascular congestion
 - Atelectasis

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Increased V/Q Ratio: Alveolar Dead Space

- Alveolar dead space:
When ventilation is
greater than perfusion
- V/Q ratio > 0.8
- Causes of non uniform
perfusion:
 - **Pulmonary Emboli**
 - Compression of pulmonary
capillaries (high alveolar
pressures)
 - Tumors
 - Collapse of alveoli /
pneumothorax
 - Shock (pulmonary vascular
hypotension)

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Assessing Oxygenation

- *Clinical Application: Cannot assess PaO₂ (arterial) without considering alveolar oxygenation content (PAO₂)*
 - Increase in FIO₂ will increase PAO₂
 - Increase in PACO₂ will decrease PAO₂

Note: With normal diffusion the majority of oxygen in the alveoli should diffuse across the alveolar capillary membrane.

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Importance of FIO₂

Normal arterial oxygen content of 80 - 100 mm Hg is only normal when the FIO₂ is .21

Expected PaO₂ based on FIO₂
(FIO₂ % x 6) – PaCO₂

Example: FIO₂ of 100% or 1.0 with PaCO₂ 40 mm Hg
(100 x 6) – 40 = 560 mm Hg

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PaO₂ and FIO₂ Ratio

- An assessment and trending tool
- PaO₂/ FIO₂ ratio:
 - Normal > 300
 - Acute lung injury < 300
 - ARDS < or= 200

PaO₂ less than 60 mmHg with an FIO₂ of 0.5 (50%) represents a clinically significant shunt.
(return of poorly oxygenated blood to the left side of the heart).

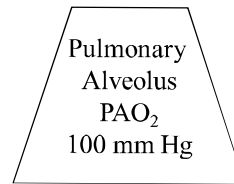
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Linking Knowledge to Practice with PaO₂ / FIO₂ Ratios

PaO ₂	FIO ₂	Ratio	Treatment / Notes
55	21%	261	Admit; respiratory distress
60	100%	60	Worsening; NRB Mask
210	100%	210	Post intubation ABG, antibiotics
190	60%	316	Continued treatment, FIO ₂ decreased
150	40%	375	Clinical improvement, FIO ₂ decreased

A – a Gradient ($PAO_2 - PaO_2$)

- Provides an index regarding diffusion as cause of hypoxemia.
- A large A-a gradient generally indicates that the lung is the site of dysfunction.
- Normal A-a Gradient = 5 to 15 mm Hg



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Hypoxemia

- Causes
 - Untreated alveolar hypoventilation
 - Diffusion abnormality
 - Ventilation and perfusion mismatching
 - Significant decreased V/Q ratio = intrapulmonary shunting
- Assessment Clues
 - PaCO₂
 - PaO₂ / SpO₂
 - PaO₂ / FIO₂ ratio
 - *A-a gradient*

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Hypoxia and Hypoxemia

■ Hypoxemia

- Insufficient oxygenation of the blood
- Mild: PaO₂ < 80 mm Hg or SaO₂ 95%
- Moderate: PaO₂ < 60 or mmHg or SaO₂ 90%
- Severe: PaO₂ < 40 mmHg or SaO₂ 75%

■ Hypoxia

- Insufficient oxygenation of tissues
- Determined by cardiac index, Hgb, SaO₂, cellular demand, patency of vessels

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Oxygen Toxicity

■ Complications of O₂

- Absorption atelectasis
- Decreased hypoxic drive

■ Signs and symptoms of oxygen toxicity

- Dyspnea
- Decreased lung compliance
- Retrosternal pain
- Parasthesia in the extremities

■ To reduce risk of oxygen toxicity:

- 100% no more than 24 hours
- 60% no more than 2-3 days
- Use 40% if therapy for longer term therapy

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Mechanical Ventilation

Indications

- Respiratory failure
 - Hypercapnic
 - Hypoxemic
- Excessive work of breathing
 - Tachypnea
 - Accessory muscle use
 - Tachycardia
 - Diaphoresis
- Protection of airway

Goals

- Achieve adequate ventilation
- Achieve adequate oxygenation
- Provide decreased work of breathing, patient comfort and synchrony with the ventilator
- Protect the lungs from further injury

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Non Invasive Positive Pressure Ventilation

- Continuous Positive Airway Pressure
 - Pressure at end expiration
 - Similar to PEEP
- Biphasic Positive Airway Pressure
 - Combines CPAP with pressure support
- Contraindications
 - Decreased level of consciousness
 - Increased gastrointestinal bleeding
 - Hemodynamic instability
 - Progressive decline in respiratory status

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Mechanical Ventilation Breaths

- **Volume cycled:**
 - Preset tidal volume

- **Time cycled:**
 - Delivered at constant pressure for preset time

- **Flow cycled:**
 - Pressure support breath. Constant pressure during inspiration.

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Modes of Ventilation

- **Assist Control Mode (AC)**
 - Volume targeted (volume cycled)
 - Pressure targeted (time cycled)
- **Synchronized Intermittent Mandatory Ventilation (SIMV)**
 - Same breath options as assist control
- **Adaptive Support Ventilation**

- **Airway Pressure Release Ventilation (APRV)**
 - Open lung strategy
- **High Frequency Oscillator Ventilation**
 - Open lung strategy

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■ Assist Control

- Minimal respiratory rate is set. Set number of breaths delivered at the preset parameters.
- Allows the patient to assist. Maintains control of patient breaths once initiated.
- Differs from (CMV) where no spontaneous breaths are allowed.
- Is effective in decreasing the work of breathing when used with appropriate sedation.

■ SIMV

- Delivers a set number of ventilator breaths at preset parameters.
- Also allows the patient to initiate breaths above the preset rate.
- Patient initiated breaths in SIMV are patient dependent and not guaranteed to achieve ventilator set parameters.
- Pressure support is often used during spontaneous breaths.
- The primary disadvantage of SIMV is the increased work of breathing in the patient with respiratory distress.

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Adaptive Support Ventilation

- Dual control
 - Pressure limited
 - Time cycled
- Breath to breath
- Pressure limit of spontaneous and mandatory breaths continuously adjusted
- Other names based on commercial ventilators

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Open Lung Strategies: Focus on Mean Airway Pressure

■ APRV

- Spontaneous breathing / tiny tidal volume
- Release time allows removal of CO₂
- P High (20 -30 cmH₂O) and P low (0) (pressure)
- T high (4-6 seconds) and T low (0.8 seconds) (time)

■ High frequency oscillation

- Not jet ventilation
- Constant mean airway pressure
- TV 1-3ml/kg
- Delivers and removes gas: 1/3 time delivery in and 2/3 time delivery out
- Usually set starting at 5 to 6 HZ (60 oscillations / HZ)
- Chest wiggle
- JVD: Tamponade effect

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Initial Ventilator Settings: Acute Respiratory Failure

- Most common initial mode of ventilation used in critical care for respiratory failure is AC with volume cycled breathes.
- Tidal volume: (VT): Usually set at 8 – 10 ml/kg of ideal body weight.
- Respiratory Rate: Usually set at 12-16 breaths per minute.
- Fraction of Inspired Oxygen (FIO₂): Started at 1.0 or 100%. Weaning as quickly as possible to .4 or 40% while maintaining an oxygen saturation of 92-94%.
- PEEP: Usually started at 5 cm of H₂O. PEEP is titrated up as needed to achieve adequate oxygenation. > 15 cm H₂O of PEEP is rarely needed.

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Adjuncts to Mechanical Ventilation

- PEEP: Positive end expiratory pressure; similar to CPAP
- PSV: Pressure support ventilation; positive pressure during inspiration; during spontaneous breaths with SIMV or during non invasive mechanical ventilation

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More on PEEP

- PEEP is used to improve oxygenation by increasing mean airway pressures and increasing the driving pressure of oxygen across the alveolar capillary membrane.
- Prevents derecruitment, low levels do not recruit
- PEEP and PAOP
- Potential complications:
 - Barotrauma
 - Decreased cardiac output
 - Regional hypoperfusion

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■ Other Ventilator Settings

- **Peak Flow** (gas flow): speed and method of VT delivery, velocity of air flow in liters per minute
- **Sensitivity**: determines patient's effort to initiate an assisted breathe
- **I:E ratio** (inspiratory to expiratory ratio): Typically set at 1:2 (can be altered to facilitate gas exchange and prevent auto peep)

■ Cuff Pressures

- Should not exceed capillary filling pressures of trachea
 - < 25 cm H₂O or < 20 mmHg
- Adequate seal for positive pressure ventilation and PEEP
- Prevents aspiration of large particles but not liquids
- **Low pressure / high volume cuffs used**
- Leak in cuff or pilot balloon valve requires replacement
- Routine measurement of cuff pressures

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Measured Parameters

- Mean Airway Pressure: Constant airway opening pressure
 - PEEP
 - CPAP
 - Pressure Support
- I:E Ratio
 - Increased inspiratory time increases mean airway pressure
 - Development of auto PEEP

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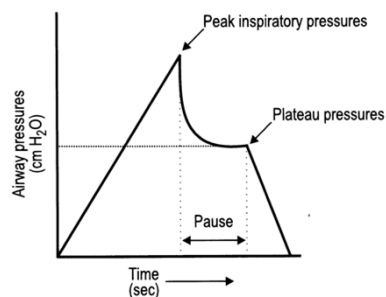
Measured Parameters

■ Peak Inspiratory Pressure

- Accounts for airway resistance and lung compliance

■ Inspiratory Plateau Pressure

- Takes resistance out of equation



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Supporting Ventilation: Improving Resistance and Compliance

■ *Interventions To Decrease Airway Resistance*

- Bronchodilators (albuterol) or steroids for bronchospasm
- Repositioning and suctioning to mobilize and aspirate secretions
- Decrease endotracheal tube resistance.
 - > 8 mm
 - Short tubes

■ *Interventions to Improve Lung Compliance*

- *Deep breath and hold*
- *Incentive spirometry (10 breaths per hour)*
- Prevent abdominal distention
- Treating underlying cause:
 - Thoracentesis or chest tube for pleural effusion
 - Diuretics for pulmonary edema
 - Antibiotics for pneumonia

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■ Hemodynamic Effects of Mechanical Ventilation

- Decreased venous return
- Pulmonary capillary compression and increased right ventricular afterload
- Decreased right ventricular stroke volume
- Decreased left ventricular afterload

■ Hypotension with Mechanical Ventilation

- Conversion to positive pressure ventilation.
 - Assure adequate circulating fluid volume
- Tension Pneumothorax
 - Chest tube required
- Development of auto PEEP
 - Increase expiration time

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Complications of Mechanical Ventilation

- **Barotrauma**
 - caused by excessive pressure
- **Volutrauma**
 - caused by excessive volume
- **Atelectrauma**
 - caused by low volume resulting in repetitive opening and closing of distal lung units
- **Biotrauma**
 - caused by biochemical mediators released in response to mechanical ventilation as opposed to a mechanical complication

Lung Protective Strategies

- Low tidal volume (6 ml / kg) with permissive hypercapnea
- Maintain plateau pressure ≤ 30 mm Hg

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Benefits of Sedation During Mechanical Ventilation

- Reduce anxiety
- Amnesia, particularly during use of neuromuscular blocking agents
- Prevent recall of unpleasant experience
- Decrease level of stress hormones
- Reduce tissue oxygen consumption
- Improve ventilator synchrony

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Prevention of VAP

- Hand hygiene
- Brushing of teeth, gums, and tongue
- HOB elevated 30 to 40 degrees
- Suction only when necessary (not routine)
- Routine installation of NS not recommended
- Adequate endotracheal tube cuff pressure
 - Maintain at < 20 mm Hg or < 25 cm H₂O to not exceed capillary filling pressure of trachea.
 - Low pressure high volume cuffs typically used.
 - Inflate to assure no or minimal leak during inspiration.
 - Need for increasing air may be due to tracheal dilation or leak in cuff or pilot balloon valve (tube must be replaced if leak present).
 - Cuff pressures measured routinely every 8-12 hours and with any change in tube position.

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Prevention of VAP

- Cover yankauer catheters when not in use
- Ventilator circuit changes only when soiled, or weekly
- Subglottic suctioning prior to repositioning or deflating cuff
- Hold tube feedings if residuals > 150 cc
- Discontinue NG tubes as soon as possible
- Extubate as soon as possible
- Avoid nasal intubation
- Stress ulcer prophylaxis with sucralfate rather than H2 blockers or proton pump inhibitors (potential advantage)
- Avoid overuse of antibiotics

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Basic Ventilator Changes: Review

- | | |
|-------------------------------|------------------------------|
| ■ To Change PaCO ₂ | ■ To change PaO ₂ |
| – Change Rate | – Change FIO ₂ |
| – Change Tidal Volume | – Change PEEP |

Ventilation
Problem

Oxygenation
Problem

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Ventilator Weaning

■ Spontaneous breathing trial

- Short period of time
- T – Piece with CPAP or CPAP and PSV
- If patient has not been on ventilator very long
- Quickly need to extubate if patient tolerates

■ IMV

- Decreasing rate
- Adding pressure support
- If patient has been on ventilator for several days and has deconditioning of respiratory muscles

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Minimum Weaning Parameters

- Spontaneous respiratory rate < 30 breaths per minute
- Spontaneous tidal volume: > 5ml/kg
- Vital capacity: > 10 ml/kg, ideally 15ml/kg
- Minute ventilation: < 10L
- Negative inspiratory pressure: < -25 to -30 cm H₂O
- FIO₂: < 0.50
- PaO₂ / FIO₂ ratio > 200

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Tracheostomy

■ Indications

- Facilitate removal of secretions
- Decrease dead space
- Bypass upper airway obstruction
- Prevent or limit aspiration with cuffed tube
- Patient comfort for prolonged mechanical ventilation

■ Benefits

- Decrease laryngeal damage, swallowing dysfunction, and glottic trauma
- Decrease in airway resistance
- Improved ability to suction lower airways
- Decreases risk of sinusitis
- Improved patient comfort and mobility

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Bedside Respiratory Monitoring: SpO₂ (Pulse Oximetry)

- Used to estimate oxyhemoglobin. The SpO₂ generally correlates with the SaO₂ + or - 2%.
- The goal equal to or greater than 92-94% in most patients.
 - Higher in African Americans
- Requires the presence of a pleth wave detecting an accurate pulse.
 - Patients receiving administration of high fat content such as with propofol or TPN can have a falsely high SpO₂.
- Several factors can interfere with the accuracy
 - Hemoglobin < 5 g/dL or hematocrit < 15%.
 - Abnormal hemoglobin, such as carboxyhemoglobin or methemoglobin.
 - SpO₂ below 70%.
 - State of low blood flow, such as with hypotension or vasoconstriction.
 - IV dyes, fingernail polish, and some skin pigmentations

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Acid – Base Balance

■ pH

- Indirect measurement of hydrogen ion concentration
- Reflection of balance between carbonic acid and bicarbonate (base)
- Inversely proportional to hydrogen ion concentration (acids donate H⁺ ions)
 - ▲H⁺ concentration = ▼pH, more acid
 - ▼H⁺ concentration = ▲pH, less acid
 - pH <6.8 or > 7.8 is incompatible with life

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Acid - Base Regulation

■ Respiratory System

- Responds within minutes – fast but weak
- Regulates the excretion or retention of carbonic acid
 - If pH is down: increase rate and depth of respiration to blow off PCO₂
 - If pH is up: decrease rate and depth of respiration to retain PCO₂

■ Renal System

- Responds within 48 hours – slow but powerful
- Regulates excretion or retention of bicarbonate and the excretion of hydrogen and non-volatile acids
 - If pH is down: kidney retains bicarbonate
 - If pH is up: kidney excretes bicarbonate

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ABG Analysis

- Evaluate ventilation: PaCO₂
- Evaluate acid-base status: pH
- Evaluate source of abnormal pH:
respiratory or metabolic
- Evaluate oxygenation: PaO₂, SaO₂

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ABG Analysis: Parameters

- pH
 - Normal 7.35-7.45
 - < 7.35 Acidosis (Acidemia)
 - >7.45 Alkalosis (Alkalemia)
- PaCO₂
 - Normal 35-45 mm Hg
 - < 35 alkalosis or respiratory compensation for metabolic acidosis
 - >45 acidosis or respiratory compensation for metabolic alkalosis

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ABG Analysis: Parameters

- HCO_3
 - Normal 22-26 mEq/L
 - < 22 metabolic acidosis or metabolic compensation for respiratory alkalosis
 - > 26 metabolic alkalosis or metabolic compensation for respiratory acidosis
- Base Excess (BE)
 - Normal +2 to -2
 - < -2 (base deficit) metabolic acidosis or metabolic compensation for respiratory alkalosis
 - > +2 metabolic alkalosis or metabolic compensation for respiratory acidosis

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ABG Analysis: Parameters

- Pao_2
 - Normal 80-100 mm Hg
 - >100 hyperoxemia
 - < 80 mild hypoxemia
 - < 60 moderate hypoxemia
 - < 40 severe hypoxemia
- Sao_2
 - Normal 95% or >
 - < 95% mild desaturation of HGB
 - < 90% moderate desaturation of HGB
 - <75% severe desaturation of HGB

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Compensation

An acidosis or alkalosis for which there has been compensation causes the pH to return to the normal range while leaning toward the initial disorder. The body never overcompensates. A non leaning pH with two abnormal indicators suggests a mixed disorder (one alkalotic and one acidotic process).

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Anion Gap

- The anion gap is used to help determine the cause of the patient's metabolic acidosis.
- **Anion Gap = $\text{Na}^+ - [\text{Cl}^- + \text{HCO}_3^-]$**
- A normal anion gap is 12 (+ or - 4) mEq/L.
- An increased anion gap typically indicates an increased concentration of anions other than Cl^- and HCO_3^- .
 - Lactic acidosis
 - Ketoacidosis
 - Renal retention of anions

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■ Respiratory Acidosis

- Depression of respiratory control centers
- Neuromuscular disorders
- Chest wall restriction
- Lung restriction
- Airway obstruction
- Pulmonary parenchymal disease

■ Respiratory Alkalosis

- Central nervous system disorders
- Drugs
- Hormones
- Bacteremia
- High altitude
- Over mechanical ventilation
- Acute asthma
- Pulmonary embolism

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■ Metabolic Acidosis

- Ingested toxic substances
- Loss of bicarbonate ions
- Lactic acidosis
- Ketoacidosis
- Renal failure

■ Metabolic Alkalosis

- Loss of hydrogen ions
 - Vomiting
 - Diuretics
 - Steroids
- Excess bicarbonate

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ABG Analysis Practice

■ pH	7.30
■ PaCO ₂	54
■ HCO ₃	26
■ PaO ₂	64

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ABG Analysis Practice

■ pH	7.48
■ PaCO ₂	30
■ HCO ₃	24
■ PaO ₂	96

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ABG Analysis Practice

■ pH	7.30
■ PaCO ₂	40
■ HCO ₃	18
■ PaO ₂	85

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ABG Analysis Practice

■ pH	7.50
■ PaCO ₂	40
■ HCO ₃	33
■ PaO ₂	92

74

ABG Analysis Practice

■ pH	7.35
■ PaCO ₂	54
■ HCO ₃	30
■ PaO ₂	55

75

ABG Analysis Practice

■ pH	7.21
■ PaCO ₂	60
■ HCO ₃	20
■ PaO ₂	48

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ABG Analysis Practice

■ pH	7.54
■ PaCO ₂	25
■ HCO ₃	30
■ PaO ₂	95

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Acute Respiratory Failure

Failure of the respiratory system to provide for the exchange of oxygen and carbon dioxide between the environment and tissues in quantities sufficient to sustain life

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Acute Respiratory Failure

■ Type I: Hypoxemic Normocapnic

- Low PaO₂
- Normal PaCO₂
- Widened A-a gradient

**Oxygenation
Failure**

■ Type II: Hypoxemic Hypercapnic

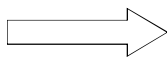
- Low PaO₂
- High PaCO₂
- Normal A-a gradient

**Ventilatory
Failure**

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Acute Respiratory Failure: Causes

- Type I (oxygenation failure)
- Pathophysiology: Decreased V/Q ratio (shunting), diffusion defect
 - Pneumonia
 - Pulmonary edema
 - Pleural effusion
 - ARDS



Alveolar / capillary
exchange impacted

- Type II (acute ventilatory failure)
- Pathophysiology: Hypoventilation
 - CNS depressant drugs
 - Spinal cord injury
 - Chest trauma
 - Acute exacerbation of COPD

**Oxygen Therapy and
PCO₂ goals
in COPD**

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Pulmonary Pharmacology

- Bronchodilators
 - Anticholinergics are the first-line medication in maintenance therapy.
 - ipratropium (Atrovent).
 - Beta-agonists can be added
 - Short acting
 - racemic albuterol (Ventolin, Proventil, Accuneb).
 - levalbuterol (Xopenex).
 - metaproterenol (Alupent).
 - pirbuterol (Exirel, Maxair).
 - Long acting
 - salmeterol (Serevent).
 - formoterol (Foradil, Oxeze).
 - Theophylline is a long acting weak bronchodilator.

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Practice Example

- Patient history: COPD (CO₂) retainer
- Initial presentation: Tachypneic with SaO₂ of 78%

- Cause of exacerbation ?
- Initial interventions?

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Case Example

- ABG
 - 7.29
 - PaCO₂ 60
 - HCO₃ 30
 - PaO₂ 48
- Treatment options?
- Goals for ABG values?

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Pulmonary Embolism

- Obstruction of blood flow to one or more arteries of the lung by a thrombus (other emboli – fat, air, amniotic fluid) lodged in a pulmonary vessel
- Lower lobes frequently affected due to increased perfusion

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Risk Factors for PE

- Stasis of blood
 - Prolonged immobilization after surgical procedures
 - Plaster casts
 - Venous obstruction
 - Heart failure / Shock / Hypovolemia
 - Varicose veins
 - Obesity
- Hypercoagulability
 - Polycythemia vera
 - Sickle cell disease
 - Malignancy
 - Pregnancy
 - Recent trauma
 - Oral contraceptives
- Injury to the vascular endothelium
 - Central venous and arterial catheters
 - Phlebitis

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Pulmonary Embolism: Pathophysiology

- > 90% of thrombus develop in deep veins of iliofemoral system
 - Can also originate in the right side of the heart, pelvic veins, and axillary or subclavian veins.
 - Another source is around indwelling catheters.
- Thrombus formation leads to platelet adhesiveness and release of serotonin (vasoconstrictor).
- Dislodgement of thrombus
 - Intravascular pressure changes (standing, massaging legs, fluid challenge, valsalva maneuver).
 - Natural clot dissolution (7-10 days after development).

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Pulmonary Embolism: Pathophysiology

- Clot lodges in pulmonary vessels
- Ventilation continues but perfusion decreases
 - Increase in alveolar dead space
 - Alveolar CO₂ decreases (alveolar shrinking). Allows for more inspired air into the perfused alveoli.
- Overperfusion of uninvolved lung results in a decreased V/Q ratio in other areas
- Decreased blood flow damages type II pneumocytes, which results in a decrease in surfactant production. (atelectasis)
- Pulmonary edema can develop as secondary complication
- Hypoxemia can occur due to ventilation perfusion mismatching
- Increased PVR can lead to pulmonary hypertension and potential acute cor pulmonale
- Cardiogenic shock can occur as the result of right-ventricular failure

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Pulmonary Embolus: Clinical Presentation

- Large to massive when 50% of pulmonary artery bed is occluded
 - Impending doom
 - Hypoxemia
 - Syncope
 - Sign and symptoms of right heart strain or right-ventricular failure
 - Signs of right-ventricular strain on ECG.
 - Sudden shock
 - Pulseless electrical activity
- Medium-sized emboli
 - Dyspnea
 - Substernal chest discomfort/pleuritic chest pain
 - Many non-specific signs
 - Tachypnea
 - Tachycardia
 - Rales
 - Accentuated 2nd heart sound

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Pulmonary Infarction

- Pulmonary infarction is infrequent
- More common
 - Large embolus
 - Pre-existing lung disease
- Results in alveoli filling with RBCs and inflammatory cells
- Complicated by infection
 - Abscess
- Signs and Symptoms
 - Pleuritic chest pain
 - Dyspnea
 - Hemoptysis
 - Cough
 - Pleural friction rub

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Pulmonary Embolus: Treatment

- Prevent thrombus formation
 - Compression stockings that provide a 30-40 mm Hg or higher gradient
 - Low molecular weight heparin
- Heparin is the treatment of choice for reducing mortality in PE
 - Initiated prior to a confirmed diagnosis
 - Slows or prevents clot progression and decreases risk of further emboli
- Fibrinolytic therapy
 - Indicated in patients with hypotension (even if resolved), hypoxemia, or evidence of right-ventricular strain
 - Troponin levels can also be used to guide decision-making in patients with sub-massive PE
- Pulmonary embolectomy is a surgical option when fibrinolytic therapy is contraindicated.

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Pulmonary Embolus: Treatment

- Oxygen is indicated, even in the absence of hypoxemia
- Pulmonary vasodilators to help reduce pulmonary vascular resistance
- Treat right-ventricular failure with fluids and inotropes
- **Obstructive Shock**
- Warfarin
 - 3 to 6 months if there is identifiable reversible risk factor
 - Minimum of 6 six months if there is no identifiable risk factor
 - Long term with recurrent PE or in patients with ongoing risk factors
- Surgical interruption of inferior vena cava with a filter
 - Patients with contraindication to anticoagulants.
 - Recurrent thromboembolism despite anticoagulant.
 - Survivor of massive PE

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Special Considerations Air Emboli

- Large volume of air into venous system
- **Risk Factors**
 - Dialysis
 - Pulmonary artery catheters
 - Surgical procedures
 - CABG
- **Symptoms**
 - Dyspnea, chest pain, agitation, confusion, cough
- **Treatment**
 - Prevent
 - 100% oxygen
 - Left lateral / trendelenburg
 - Positive pressure ventilation
 - Hyperbaric oxygen
 - Aspiration of air

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Acute Respiratory Distress Syndrome

A syndrome of acute respiratory failure characterized by non-cardiac pulmonary edema and manifested by refractory hypoxemia. ARDS does not include mild or early acute lung injury, but rather involves severe and diffused lung injury.

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Risk Factors in ARDS

- Sepsis (most common)
- Transfusion
- Aspiration
- Trauma
- Massive transfusion
- Pancreatitis

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Acute Respiratory Distress Syndrome: Etiology

- | | |
|----------------------|------------------------|
| ■ Direct lung injury | ■ Indirect lung injury |
| – Chest trauma | – Sepsis |
| – Near drowning | – Shock |
| – Smoke inhalation | – Multi system trauma |
| – Pneumonia | – Burns |
| – Pulmonary embolism | – CABG |
| | – Head injury |

Time from injury of alveolar capillary membrane to onset of symptoms is 12-48 hours.

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ARDS: Pathophysiology

- Stimulation of **inflammatory** and immune systems
- Release of toxic substances, causing **micro vascular injury**
- Pulmonary capillary membranes are damaged
 - **Increase in capillary permeability**
- Cells and fluids leak into interstitium and alveolar spaces
 - **Pulmonary edema**
- Impaired production and dysfunction of surfactant
 - **Alveolar collapse and massive atelectasis**
- **Intrapulmonary shunting**
- Hypoxic vasoconstriction
- **Decreased the compliance of lung**
 - High-peak inspiratory pressures to ventilate the lungs.
- **Potential development of pulmonary fibrosis in chronic phase**
 - Endothelium, epithelium, interstitial space expand.
 - Protein exudate inside the alveoli produces a hyaline membrane.

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Acute Respiratory Distress Syndrome: Diagnosis

- Predisposing condition
- $\text{PaO}_2/\text{FIO}_2$ ratio < 200
- Chest x-ray: Diffuse bilateral infiltrates (Chest CT may also be used)
- Decreased static compliance of lungs
- PAOP < 18 mm Hg or no evidence of increased left-atrial pressure
- No evidence of COPD
- No other explanation for above

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ARDS: Treatment

- Optimal ventilation / oxygenation
- Avoid over hydration
- No routine use of steroids

- Pulmonary vasodilators
 - Nitric oxide by inhalation (dilates vessels to only ventilated alveoli)

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Mechanical Ventilator Management Strategies for ARDS

- Lower tidal volume ventilation
 - Permissive hypercapnia
- Maintain plateau pressure < 30 mmHg

- Uninterrupted PEEP
- Avoidance of auto PEEP

- Airway pressure release ventilation
- High frequency ventilation (Oscillatory)
- Independent lung ventilation
- ECMO

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Case Example

- 65 year old female; 85 kg
- Post witnessed cardiac arrest
- Initial PaO₂ / FIO₂ ratio 102

- Initial diagnosis?

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Case Example

- Ventilator settings:
 - AC
 - Rate 12
 - TV 700 ml
 - FIO₂ 80%
 - PEEP 5 cm
- 2nd ABG
 - pH - 7.33
 - PaCO₂ - 40 mmHg
 - HCO₃ - 14
 - PaO₂ - 92
 - PaO₂ / FIO₂ = 115
- Ventilator adjustment?
- Other treatment considerations?

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Pulmonary Hypertension

- PPH (primary pulmonary hypertension)
- IPAH (idiopathic pulmonary arterial hypertension)
- Rare disease
- Perhaps caused by insult to endothelium in patient with susceptibility to pulmonary vascular injury
 - Vascular scarring
 - Endothelial dysfunction
 - Intimal and medial smooth muscle proliferation

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Associated Conditions

- Portal hypertension
- Connective tissue diseases
 - Associated
- Anorexigens
- Alpha adrenergic stimulants (i.e. cocaine / amphetamines)
- HIV

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- IPAH has no cure
- Untreated leads to right sided heart failure and death
- New drugs have improved survival rates
 - Prostacyclin analogues
 - Endothelin receptor antagonists
- APAH occurs more frequently than IPAH
- 15-20% have familial component
- Females are affected more than males
 - Women of child bearing age more often affected.

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Presentation

- Average time from symptom onset to diagnosis is 2 years
- Most common symptoms in one study:
 - Dyspnea (60%)
 - Weakness (19%)
 - Recurrent syncope (13%)
- Possible Physical finding:
 - Increased pulmonic component of 2nd heart sound
 - Palpable 2nd heart sound
 - Murmurs of pulmonic and tricuspid regurgitation
 - Right ventricular heave
 - JVD
 - Large V waves
 - Other signs of right heart failure
 - Normal lungs

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Treatment

- **General**
 - Anticoagulation (Warfarin)
 - Digoxin
 - Diuretics
 - Oxygen with hypoxemia
 - May be of no benefit in left to right shunt
- **Conventional Pulmonary Vasodilators**
 - Calcium channel blockers
 - Nifedipine / diltiazem
 - Only used in patients who are responders to acute vasodilator testing (25%)
 - IV adenosine or inhaled nitric oxide
 - High doses are used
 - Can have rebound pulmonary hypertension when withdrawn
 - Only in patients without overt right sided heart failure

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Approved Pulmonary Vasodilators for PPH

- **Epoprostenol (Flolan)**
 - IV - Parental
 - Prostacyclin analogue (prostanoid)
- **Treprostinil (Remodulin)**
 - IV / SQ – Parental
 - Prostacyclin analogue (prostanoid)
- **Iloprost (Ventavis)**
 - Nebulized inhalation
 - Prostacyclin analogue (prostanoid)

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Approved Pulmonary Vasodilators for PPH

- Bosentan (Tracleer)
 - Oral
 - Endothelin antagonist (ERA)
- Ambrisentan (Letairis)
 - Oral
 - Endothelin antagonist (ERA)
- Sildenafil (Revatio)
 - Phosphodiesterase (type 5) enzyme inhibitor
 - Oral

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Other Treatment Options

- Single or double lung transplant (cardiac transplant may or may not be needed)
- Atrial septostomy (palliative)
 - Creates right to left shunt
- Cardiopulmonary rehab for mild symptom limited aerobic activity

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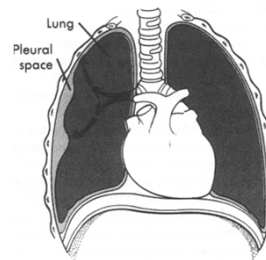
Closed (Simple) Pneumothorax

- Air enters the intrapleural space through the lung causing partial or total collapse of the lung
- **Possible etiology**
 - Blunt trauma (lung laceration by rib fracture)
 - Positive pressure ventilation (rupture of weak alveoli, bleb or bullous)

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Closed (Simple) Pneumothorax

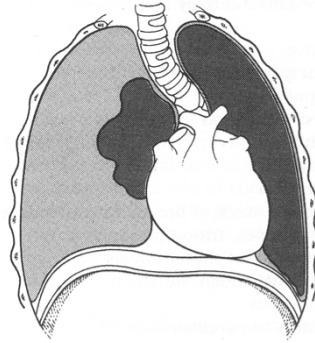
- **Pathophysiology**
 - Disruption of normal negative intrapleural pressure
 - Lung collapse
 - Decreased surface area for gas exchange
 - Acute respiratory failure
- **Signs and Symptoms**
 - Chest Pain, dyspnea, asymmetrical chest excursion, diminished to absent breath sounds on affected side, dramatic increases in peak inspiratory pressures on a mechanical ventilator
- **Treatment**
 - Oxygen
 - Position
 - Analgesics
 - Chest Tube Criteria



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Tension Pneumothorax

- Accumulation of air into the pleural space without a means of escape causes complete lung collapse and potential mediastinal shift
- **Etiology**
 - Blunt trauma
 - Positive pressure mechanical ventilation
 - Clamped or clotted water seal drainage system
 - Airtight dressing on open pneumothorax



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Tension Pneumothorax

- **Pathophysiology**
 - Air rushes in-cannot escape pleural space
 - Creates positive pressure in pleural space
 - Ipsilateral lung collapse
 - Mediastinal shift \Rightarrow contralateral lung compression \Rightarrow potential tearing of thoracic aorta
 - Can also compress heart \Rightarrow decrease RV filling \Rightarrow shock

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Tension Pneumothorax

Signs and Symptoms

- Similar to closed pneumothorax
- If mediastinal shift:
 - Tracheal shift away from affected side
 - JVD
 - Hypotension

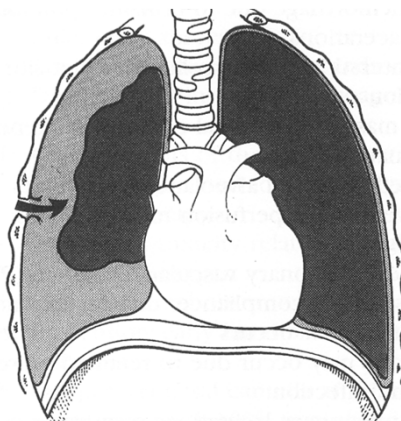
Treatment

- Oxygen (100%)
- Emergency decompression
 - 2nd anterior space mid clavicular
 - Large bore needle / flutter valve
- Chest Tube
- Other as with closed pneumo

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Open Pneumothorax

- Air enters the pleural space through the chest wall
- **Etiology**
 - Penetrating Trauma



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Open Pneumothorax

■ Pathophysiology and Signs and Symptoms

- Equilibrium between intrathoracic and atmospheric pressures
- Patient condition depends on size of opening compared to trachea
- The affected lung collapses during inspiration
- May cause a tension pneumothorax
- Subcutaneous emphysema usually present

■ Treatment

- Similar to closed pneumothorax
- Closure of open wound with petroleum jelly gauze
 - End expiration
 - Modification for tension pneumothorax
- Chest tube and water seal drainage

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