CCRN Review: Pulmonary

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Please make sure all phones and pagers are switched to mute or vibrate!
Objectives

At the end of this presentation, the participants will:

- Apply knowledge of pulmonary physiology and arterial blood gases to collaboratively manage acute and chronic pulmonary disorders, with and without mechanical ventilation.

- Differentiate acute hypoxic pulmonary failures (Pulmonary Embolism, ARDs, Pneumonia, Airleaks) and determine collaborative management strategies for each.

- Describe collaborative interventions for managing patients with airway disorders (COPD, Asthma, Emphysema).

- Relate nursing interventions for thoracic traumas/surgeries and pulmonary hypertension.
Test Plan

- Acute Lung Injury
  - ARDS
- Acute Pulmonary Embolus
- Acute Respiratory Failure
- Acute Respiratory Infections
  - Pneumonia
  - Bronchiolitis
- Air-leak Syndromes
- Aspiration Pneumonia
- COPD, Asthma, Chronic Bronchitis, Emphysema
- Pulmonary Hypertension
- Status Asthmaticus
- Thoracic Surgery
- Thoracic Trauma
  - Fractured Ribs
  - Lung Contusions
  - Tracheal Perforation
Review of Pulmonary Anatomy

- The transfer of inhaled oxygen and exhaled carbon dioxide occurs at the alveoli.
- Each alveoli is surrounded by a capillary bed that reaches the lungs from the pulmonary arteries.
Respiration is the process by which O2 is transferred from the air to the tissues and CO2 is excreted in the expired air.

Respiration involves a 3 Step Process:

- Ventilation
- Diffusion
- Transport
Control of Breathing

- **Respiratory pacemaker** is located in medulla
  - Generates rhythmic cycle
  - Breathing is spontaneous, but becomes irregular if input from the pons is disrupted.

- **Chemoreceptors**
  - Oxygen receptors are located in carotid / aortic bodies
    - PaO2 must be <60 to activate
  - Carbon Dioxide receptors located in the medulla are the main respiratory regulators.
    - PaCO2 > 70-80 can depress CNS
Work of Breathing

- The amount of effort required to maintain a given level of ventilation.

Determined by:

- **Lung Compliance** - Measure of elasticity of the lungs and thorax.
- **Airway Resistance** - The opposition to gas flow in the airways. Mainly due to diameter of airways.
  - Small changes in diameter produce large changes in resistance.
  - Autonomic nervous system and inflammatory mediators affect resistance:
    - Parasympathetic
    - Sympathetic
    - Histamine
Oxygen Transport

- Oxygen is carried in the blood in two ways:
  - Bound to hemoglobin in RBC’s (SaO2)
  - Dissolved in plasma (PaO2)
- Oxyhemoglobin dissociation curve
  - Shows the relationship between O2 saturation and PaO2.
  - Describes the ability of hemoglobin to bind to O2
Carbon Dioxide Transport

- Carried in the blood in three ways:
  - Dissolved in the plasma (PaCO2)
  - Chemically combined with hemoglobin
  - As bicarbonate through a conversion reaction:
    - \[ \text{CO}_2 + \text{H}_2\text{O} \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{H} + \text{HCO}_3^- \]

**KEY CONCEPT:** The amount of CO2 in the plasma determines the acidity of the blood.
Normal Diffusion

- The exchange of O2 and CO2 between the alveoli and capillaries normally occurs so that gases move from areas of higher concentration to lower concentration.

- Diffusion impairment can result from:
  - Thickening of alveolar capillary membrane
  - Reduction in alveolar capillary membrane surface area
If Ventilation/Perfusion (V/Q) mismatch occurs, the body compensates:

A) If capillary perfusion is decreased, the bronchioles constrict to limit air flow to that area.

B) If alveoli are not oxygenated, the arterioles to the area constrict to shunt blood away from the non-ventilated area.

To estimate the amount of shunt through the lungs, divide the patient PaO2 by the FiO2.
Definitions:
Shunt, Hypoxia, Hypoxemia

- **Shunt** – The amount of blood circulating through the lungs that *does not* participate in gas exchange
  - To estimate the amount of shunt through the lungs, divide the patient PaO2 by the FiO2
    - Normal: > 300
    - 20% shunt: 200

- **Hypoxia**: Decrease in the tissue oxygenation.
  - Oxygen therapy alone may not correct.

- **Hypoxemia**: Decrease in arterial blood oxygen tension (PaO2).
  - A good PaO2 does not guarantee tissue oxygenation.
  - Organs most susceptible: Brain, heart, kidneys, adrenals, liver, retina
Arterial Blood Gases
Arterial Blood Gases

- Arterial Blood Gases are used to determine both the acid-base status and the arterial oxygenation status of the body.
- Results must be interpreted in conjunction with the patient’s clinical picture.
- ABG interpretation is the systematic evaluation of individual test results.
Acid - Base Balance

- The body pH must remain within normal limits or the body will die.

- The respiratory and metabolic systems work together to maintain balance
  - The respiratory system begins to make adjustments immediately when there are imbalances.
  - The metabolic system may take days to adjust to imbalances.
pH

- The pH of blood is a measurement of the concentration of hydrogen ions in the plasma.
- Normal range: 7.35 – 7.45 (mean 7.40)
  - If a patient’s pH is below 7.35, the patient is experiencing *acidosis*.
  - If a patient’s pH is above 7.45, the patient is experiencing *alkalosis*.
Determination of pH

- In the blood, carbon dioxide (CO2) combines with water (H20) to form carbonic acid (H2CO3) according to the following:
  \[ \text{CO2 + H2O} \rightleftharpoons \text{H2CO3} \]

- In the kidneys, this acid is broken down to bicarbonate (HCO3) :
  \[ \text{H2CO3} \rightarrow \text{H} + \text{HC03} \]

Carbon dioxide concentration determines the amount of acid in the blood

Bicarbonate concentration determines the amount of base in the blood
Respiratory Component: CO2

- The CO2 level of the blood is controlled by the respiratory system.

  **Normal range is 35-45 mmHg**

- When the PaCO2 is below 40, there is LESS CO2 to form acid.
  - This occurs when the patient hyperventilates or blows off CO2.
  - The patient becomes alkalotic

- When the PaCo2 is above 40, there is MORE CO2 to form acid.
  - This occurs when the patient is hypoventilated.
  - The patient becomes acidotic.
The amount of bicarbonate ion, HCO3, is controlled by the kidney.

**Normal range is 22 – 26 mEq/l**

- When HCO3 is above 24, there is *MORE* base.
  - This occurs when the kidneys retain more bicarbonate ion
  - The patient becomes alkalotic

- When HCO3 is below 24, there is *LESS* base.
  - This occurs when bicarbonate ion is excreted by the kidney or lost through other sources
  - The patient becomes acidic
Steps of ABG Interpretation

- **Step I – Determine oxygenation**
  - PaO2 is the partial pressure of oxygen dissolved in arterial blood. It reflects only 3% of the total oxygen in the blood.
    - **Normal level**: 80 – 100 mmHg.
  - SaO2 is the measure of oxygen bound to hemoglobin.
    - **Normal SaO2**: 95% or greater on room air
  - **Special Considerations:**
    - Normal PaO2 is decreased in the elderly and neonates.

**Panic PaO2 at any age**: Below 40
**Step II**
Determine whether the pH is on the acid or base side of:

7.35  7.4  7.45

**Step III**
Determine if the CO2 is on the acid or base side of:

35  40  45

**Step IV**
Determine if the bicarb is on the acid or base side of:

22  24  26
Step V: Match it!

The component that matches the PH is the system controlling the ABG!

Acidosis:
- If CO2 is elevated, the pH is under *respiratory control*
- If HCO3 is low, the pH is under *metabolic control*

Alkalosis:
- If CO2 is low, the pH is under *respiratory control*
- If HCO3 is elevated, the pH is under *metabolic control*

If both systems match the pH, the patient is having problems with both systems!
Step VI: Determine If Compensation Has Started

- The metabolic and respiratory systems compensate to control pH.

- If the pH is normal, but PaCO2 and HCO3 are abnormal, the body is compensating for something.
  
  pH 7.35 - 7.40 – Recovering **ACIDOSIS**
  pH 7.40 - 7.45 – Recovering **ALKALOSIS**

- Compensation:
  - Partial compensation
  - Complete compensation

The body NEVER overcompensates!
PRACTICE ABG 1:

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<td>7.35</td>
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<tr>
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<tr>
<td>ACID</td>
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</tr>
<tr>
<td>BASE</td>
<td>7.35</td>
<td>40</td>
<td>24</td>
</tr>
</tbody>
</table>
PRACTICE ABG 2:

| pH   | 7.24 |
| PaCO2 | 60   |
| HCO3  | 30   |

7.35  7.4  7.45

ACID   BASE   ACID

35  40  45
BASE   ACID   BASE

22  24  26
PRACTICE ABG 3:

<table>
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<td>HCO3</td>
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</tbody>
</table>

ACID

BASE

ACID

BASE

7.35

35

22

7.4

40

24

7.45

45

26
PRACTICE ABG 4:

pH: 7.44
pCO2: 29
Bicarb: 19

7.35 ACID 7.4 BASE 7.45
35 BASE 40 ACID 45
22 ACID 24 BASE 26
PRACTICE ABG 5:

pH: 7.32
pCO2: 50
Bicarb: 24
PRACTICE ABG 6:

pH: 7.25
pCO2: 50
Bicarb: 18

7.35  ACID  7.4  BASE  7.45
35  BASE  40  ACID  45
22  ACID  24  BASE  26
Managing Acute Hypoxic Pulmonary Failure
Acute Respiratory Failure

- A rapid onset of respiratory impairment, which is acute enough to cause morbidity or mortality if untreated. Can be caused by a number of problems.

- Defined by:
  - PaO2 below 60 mmHg
  - PaCO2 above 50 mmHg

- 4 categories of causes:
  - Impaired ventilation
  - Impaired gas exchange
  - Ventilation / Perfusion (V/Q) mismatch
  - Airway obstruction

*Despite the cause, acute respiratory failure worsens due to anxiety!*
General Treatment Principles

- **Assure airway patency**
  - Airway adjuncts or suctioning if the patient is having difficulty managing secretions
  - Initiate aggressive pulmonary hygiene

- **Provide supplemental oxygen.**
  - Non invasive ventilation is usually preferred if acceptable PaO2 can be achieved

- **Improve ventilation**
  - May need to administer medications such as bronchodilators or mucolytics

- **Correct the underlying cause**

- **Reduce anxiety**
Visualizing Oxygen Delivery

- **SvO₂ = 75%**
- **SaO₂ = 100%**
- Arterial Oxygen Content (≈ SaO₂)

Venous Return

Venous Oxygen Content (≈ SvO₂)

Oxygen Consumption

Oxygen Delivery

The Cell

LOADING STATION

Oxygen Diffusion

Tissue Demands
Mechanical Ventilation
Lung Volumes

To decrease CO₂, work here.

To increase O₂, work here.
Ventilator Terminology

- **Tidal Volume**: The amount of air moving in and out of the lung with each normal breath.
  - Usually 10 cc/kg

- **FiO2**: Fraction of inspired oxygen.
  - Room air is 21%
  - Can deliver up to 100%

- **PIP**: Peak Inspiratory Pressure.
  - The highest pressure allowed before the ventilator alarms for excess pressure

- **PEEP**: Positive End Expiratory Pressure.
Positive End Expiratory Pressure (PEEP)

- Increases volume at end-expiration
- Prevents/Decreases alveolar collapse
- Physiologic PEEP is 5 cm H2O
- Levels > 5 cm H2O are usually used to recruit collapsed alveoli resulting in increased ventilation
  - Results in increased oxygenation
- Lower levels of PEEP may be used in the acute asthmatic or COPD patient due to hyperinflation.
- Complications of higher PEEP levels include:
  - Barotrauma
  - Decreased preload
  - May increase ICP
  - Increased afterload
Non-Invasive Ventilation

Continuous Positive Airway Pressure (CPAP)
- Also called spontaneous mode
- Used in treatment of sleep apnea in adults.
- Can be used as a step in weaning from mechanical ventilator.
- *Entire work of breathing is patient generated.*

BiLevel Positive Airway Pressure (BiPAP)
- CPAP with inspiratory pressure
- Decreases work of breathing
- Improves gas exchange
Ventilation Decision Tree
(Woodruff, D., 2002)

Airway Patent?

Yes

Mask

Therapy < 48 hours?

CPAP

No

Intubate

Therapy > 48 hours?

Is WOB increased?

BiPAP

Mechanical Ventilation
**Modes of Mechanical Ventilation**

- **AC: Assist Control**
  - Every breath is supported by a ventilator breath
  - Used when patient should have no metabolic work
    - Post arrest
    - Pulmonary edema
    - ARDS
    - Anxiety

- **SIMV: Intermittent Mandatory Ventilation**
  - Patient is able to initiate breaths between ventilator breaths
    - Machine breaths are synchronized to patient pattern
  - Used as a weaning mode in some patients
  - Minimizes barotrauma and hemodynamic effects
Ventilator Modes (con’t)

**Volume controlled**
- Machine is set to deliver a set volume
- Pressures generated by each breath will vary (PIP)
- Set pressure limit where machine alarms
- Most commonly used mode in adults

**Pressure Controlled**
- Machine is set to deliver until certain airway pressure is reached
- Volumes of each breath will vary
- Will alarm if minimal volume is not delivered
- Most commonly used modes in pediatrics
- May be used for patients with ARDS

**Pressure Support (PSV)**
- Each patient breath is supported by the ventilator during inhalation
- Overcomes resistance of tubing
- Used for weaning
Ventilatory Adjuncts

- **Aerosol treatments**
  - Bronchodilators
    - Any patient can have bronchoconstriction
    - Helps mobilize secretions
  - Mucolytics
    - Hydrate patient
    - Hydrate airway
    - Then use a mucolytic

- **Nitric Oxide**
  - Pulmonary vasodilator
  - Increases oxygenation
  - Not shown to improve overall mortality

- **Helium**
  - Promotes oxygen transport to alveoli
  - Used in asthma and COPD to improve oxygenation
Ventilatory Adjuncts (con’t)

- **Prone positioning**
  - Redistributes lung fluid
  - Relieves heart weight on lower lobes
  - Improves oxygenation
  - Decreases CO2
  - Complications can be avoided by:
    - Limiting time to less than 2 hours
    - Adequate staff to prone

- **Rotational beds**
  - If cannot move to chair, use chair position of bed
  - Turn and position every 2 hours
  - Rotational therapy for high risk patients

- **Vibration and percussion**
  - Helps mobilize secretions
  - VEST therapy or percussion mode on bed
Pulmonary Embolism
Fat Embolism
Pulmonary Embolism

- An obstruction to blood flow to one or more of the arteries of the lung.
  - Most thrombi develop in deep veins of upper extremities (above knee)
  - In PE, the deep vein thrombus (DVT) has been dislodged and moved into the pulmonary vessel.

- Virchow’s Triad (Risk Factors):
  - Hypercoagulability
  - Alteration to vessel wall
  - Venous stasis

- Factors contributing to dislodgement of thrombi:
  - Intravascular pressure changes
Pulmonary Embolus (con’t)

- Clot moves into pulmonary vessel. Ventilation continues but perfusion is decreased

- No gas exchange, so alveolar CO2 decreases
  - Results in bronchoconstriction to affected alveoli

- Cessation of blood flow damages pneumocytes
  - Production of surfactant decreases
  - Atelectasis occurs and work of breathing increases
Presentation / Diagnostic Findings

- **ABG**: Decreased PaO2, SaO2;
  - pH= elevated, then decreased
- **ECG**: Tall peaked P waves, atrial dysrhythmias, sinus tachycardia, S1, Q3, T3
- **V/Q scan / Spiral CT**: Shows perfusion defect with normal ventilation. Similar sensitivity and specificity.
- **Pulmonary Angiography**: “Gold Standard”
- **Labs**: D-dimer
- **Common symptoms**:
  - Tachypnea
  - Dyspnea
  - Chest pain
  - + Homan’s sign
  - Restless, apprehension

If the embolus is large the *presenting* symptom may be PEA!
Treatment

- Prevention of DVT is the key!
- Provide supplemental oxygen/circulatory/ventilatory support
- Thrombolytic therapy may be used in massive PE
- Heparin – Prevents further clot formation
- Inferior vena cava filter – May be inserted in high risk patients to catch future clots
- Pulmonary embolectomy – A very high risk interventional procedure
- Pulmonary vasodilators have been used in some cases.
Fat Embolus Syndrome

- Patients at increased risk:
  - Long bone fracture
  - Hip replacements
- Onset 24 – 48 hours after event
- Present with ARDS-type syndrome:
  - Pulmonary edema
  - Hypoxia
  - Axillary / subconjunctival petechiae
  - CNS disturbances
  - May see: Tachycardia, fever, drop in platelets, fat globules in urine, retina, sputum
- Treatment is same as treatment for PE.
Acute Respiratory Distress Syndrome
Acute Respiratory Distress Syndrome

- Acute respiratory failure in adults characterized by pulmonary edema manifested by right to left shunting through collapsed or fluid-filled alveoli.

- Specific findings:
  - Oxygenation – PaO2 / FiO2 < 200 regardless of PEEP levels
  - Chest x-ray – Bilateral infiltrates seen on frontal chest x-ray
  - No elevated pulmonary pressures
ARDS Lungs

Normal Anatomy

- Normal gas exchange across thin alveolar walls allowing the uptake of fresh oxygen and the release of carbon dioxide

Cut-section through Alveoli at Terminus of Bronchi

- Fluid releasing from capillaries filling the alveolar space and preventing gas exchange
Predisposing Factors

- Direct Pulmonary Injury due to:
  - Aspiration of gastric contents
  - Pulmonary contusion
  - Near drowning
  - Smoke inhalation
  - Pneumonia
  - Barotrauma from mechanical ventilator

- Indirect injury caused by inflammatory mediator release. Mediator release may be triggered by:
  - Sepsis or Multiple organ dysfunction syndrome (MODS)
  - Shock
  - Pancreatitis
  - Trauma
  - DIC
  - Multiple transfusions

Risk of ARDS increases if patient has more than one risk factor:
- One risk factor = 25% chance of ARDS
- Two risk factors = 42% chance of ARDS
- Three risk factors = 85% chance of ARDS
Pathophysiology of ARDS

- Diffuse injury to the alveoli – capillary membrane
- Increased lung permeability

Flooding of alveoli causes injury to Type II pneumocytes
  - Results in decreased surfactant production
  - Decreased surfactant causes increased alveolar surface tension
  - Increased alveolar surface tension causes atelectasis

- Now blood begins to “shunt” through the lungs without passing by alveoli that are ventilated
- Lungs become “stiff” or less compliant due to hypoxemic pulmonary vasoconstriction
- Refractory hypoxemia worsens
Clinical Manifestations

- **Latent:**
  - Beginning a-c membrane changes; PaO2/FiO2

- **Acute Interstitial:**
  - Alveolar edema and decreased lung compliance
  - Dyspnea, restless on room air, anxious
  - Lung sounds = ____________
  - Oxygen saturation is decreased
  - Patient begins to hyperventilate
    - ABG will demonstrate respiratory
    - Chest x-ray will be unchanged at this phase
Clinical Manifestations:
Acute Intra-alveolar/Chronic Phase

- When the shunt reaches the 20% level, the patient will have extreme dyspnea.

- ABG = Respiratory Acidosis with REFRACTORY HYPOXEMIA

- Chest x-ray shows diffuse infiltrates throughout the lung fields (“white out”)

- Post mortem exam reveals lung tissue that is congested, heavy and wet

- If the patient survives, may develop pulmonary fibrosis:
  - Form hyaline membranes
  - Thickening of alveolar septum
  - Loss of functional alveoli
  - Slow recovery
  - Death often results from infection.
Evidence-Based Multidisciplinary Plan of Care

- Goals of ARDS Therapy:
  - Prevent further injury
  - Maintain adequate pulmonary oxygenation
  - Optimize oxygen delivery to the tissues using the six P’s
ARDS - Prevention

- Initiate nursing care that reduces bacterial colonization and risk of aspiration
  - Handwashing
  - Elevate head of bed at least 30 degrees
  - Oral Care

- Consider therapy to block injury at the alveoli-capillary interface (controversial):
  - Nitric oxide
  - Xigris
  - Corticosteroids
  - Monoclonal antibodies
  - Non steroidal anti-inflammatories
ARDs - PEEP

- Improves oxygenation by re-expanding alveoli that are unstable or collapsed due to lack of surfactant.

- Goal: “Keep the lung open” or “recruit” more alveoli

- Studies have shown that higher levels of PEEP (14 – 16 cm H20) are necessary.
  - Allow elevated CO2 as long as pH is > 7.2

- Nitric Oxide
ARDS - Pumps and Pipes

- Adjust fluids and medications to maximize oxygen delivery to the cells
  - Use SVO2 to monitor cellular oxygenation
  - Make sure you have enough hemoglobin molecules ("trucks") to get the oxygen to the cells. Transfuse early!

- Make sure that is enough fluid in the pipes (blood vessels) to supply adequate tissue perfusion
  - Monitor CVP to assess volume status.

- Use vasoactive medications to keep the "pipes" toned up and "pumps" squeezing the blood to the tissue.
ARDS - Paralysis / Position

- The ARDS patients requires aggressive sedation to decrease oxygen demands.

- Continuous Lateral Rotation Therapy
  - Nurse driven protocol to identify patients at high risk have shown decreased length of ventilator time and decreased incidence of ventilator acquired pneumonia, which is an ARDS trigger

- Prone positioning
  - Uses gravity to assure more uniform pleural pressures
  - Can open collapsed alveoli
Pneumonia

- An acute infection of the lung parenchyma, including alveolar spaces and interstitial tissue.
  - Community-/Health care associated-/Hospital acquired
  - Causative organisms are different.

- Causative agent is inhaled / enters pharynx
  - May be transmitted from one patient to the next
  - Subglottic secretions pool above ETT cuff
    - Within 24 hours, 95% of ETT were partially covered with bacteria
    - Nasal Nasogastric tubes lead to colonization of nasopharynx

- Factors that increase risk of colonization:
  - Decreased salivary flow rate
  - Poor oral hygiene
  - Systemic antibiotics
  - No oral fluid or food
Pneumonia (con’t)

- Causative agent moves into lungs from pharynx:
  - Alveoli become inflamed and edematous.
  - Alveoli spaces fill with exudate and consolidate.
  - Patient may complain of cold or flu-like symptoms
  - Alveoli spaces fill with exudate and consolidate.
  - Diffusion of oxygen is obstructed, causing hypoxemia
  - WBC will be elevated with increase of immature WBC’s, if bacterial.
Pneumonia - Treatment

- Prevent nosocomial pneumonia!!
  - Keep HOB elevated
  - Perform frequent oral care
  - Strict handwashing

- If suspected:
  - Obtain culture to identify causative organism
  - Start antibiotic promptly
  - Hydrate unless contraindicated
    - 2-3 liters / 24 hours
  - Initiate enteral feeding early to improve nutrition
Air Leak Syndromes
Air-Leak Syndromes - Types

- Air in the pleural space with complete or partial collapse of the lung. Several types:
  - Open pneumothorax
  - Closed pneumothorax
  - Iatrogenic pneumothorax
  - Spontaneous pneumothorax
  - Tension pneumothorax
Tension Pneumothorax

- Occurs when air flows freely into the pleural space during inspiration and becomes trapped

- Results in lung collapse and mediastinal shift to the opposite side

- Clinical findings:
  - Shortness of breath, progressing to extreme dyspnea
  - Unilateral absence of breath sounds
  - Asymmetry of chest movement
  - May see tracheal deviation and subcutaneous emphysema
  - May see distended neck veins and hypotension
  - MAY NOT BE ABLE TO WAIT FOR CHEST X-RAY TO CONFIRM

Needle Decompression
Chest Tube Principle: The Water Seal
Chest Drainage Systems

- Disposable chest drainage systems use the principle of the water seal to allow air / fluid to escape from the pleura.

- In addition, they have 2 other chambers:
  - Fluid collection.
  - Suction control.
Chest tube drains fluid from pleural space
Chest Trauma - Hemothorax

- Collection of blood in pleural space

**Source:**
- Left hemothorax
  - Rib fracture 36%
  - Pulmonary tissue 35%
  - Aorta 15%
- Right hemothorax
  - Rib fracture 51%
  - Pulmonary tissue 27%
  - Liver 10%

**Manifestations:**
- Dyspnea, tachypnea
- Cyanosis, hypoxemia
- Shock

**Treatment:**
- Chest drainage
- Volume replacement
- Thoracotomy
  - More than 1500 ml blood with initial chest tube insertion
- Bleeding more than 300 /hr for 3 hours
  - Hemodynamic instability
  - Tension hemothorax
Chest Tube Management

Air Leaks
- Identified by bubbling in the water seal chamber.
- An air leak is not uncommon immediately after tube placement.
- Indicates that the lung has not fully reexpanded or that there is a leak in the system.
- To prevent air leaks in the tubing or drainage system, ensure all connections are secure.
- All new leaks should be investigated.

Tidaling
- Pressure changes that occur in the pleural space with breathing can be viewed as fluctuations (tidaling) in the level of water within the tube.
- In normal spontaneous breathing, water levels will go up with inspiration (more negative) and return to baseline during exhalation.
Chest Tube Management

- Check collection chamber for:
  - Volume / rate of drainage
  - Appearance of drainage

- "Milk" clots out gently

- NO STRIPPING

- Keep collection chamber below chest level

- **Do not clamp the chest tube**
  - The only time a chest tube should be clamped is if the drainage unit is disrupted or is being changed.

- If the chest tube is accidentally dislodged:
  - Apply occlusive dressing to site
  - Monitor patient’s respiratory status, notify physician, and obtain chest x-ray.

- If the drainage system is damaged:
  - Immerse distal end of chest tube into a bottle of sterile water, notify physician, and attach new drainage unit per policy
Thoracic Surgery /Trauma
Pleural Effusion

- An abnormal accumulation of fluid in the pleural space.
  - Not a diagnosis in itself,
  - Usually due to increased permeability of the pleural membranes
  - Signs and symptoms are variable, and depend on the volume of fluid and how quickly it accumulated.

- Treatment
  - Thoracentesis, chest tube
  - Treat the cause!
Pulmonary Resection

- Type and location of surgery will dictate the type of surgical approach used.
  - Most common is postero-lateral thoracotomy
  - Care is taken to avoid drainage of blood or secretions into unaffected lung during surgery

- Hemorrhage is an early, life-threatening complication that can occur after lung resection.
  - Chest tube output more than 100 cc/hr, fresh blood, or sudden increase in drainage signals possible hemorrhage
After lobectomy, turn the patient onto the NON-OPERATIVE side.

- When the “good” lung is dependent, blood flow is greater to the area and V/Q matching is better.
- When the affected lung is dependent, this results in increased blood flow to an area with less ventilation.

After pneumonectomy, position the patient supine or on the OPERATIVE side.

- Promotes incision splinting and deep breathing
- Positioning on the unaffected side can result in drainage of secretions to the unaffected lung

Optimizing oxygenation and ventilation is critical!
Pulmonary Resection - Treatment

- Pain management is very important
  - May use intrathoracic infusion, PCA.

- Return to activity
  - ROM to shoulder on operative side can prevent frozen shoulder
  - Usually sit in chair on day of surgery with gradual increase in activity. May take 6 months to 1 year to return to pre-surgery level.

- Chest tube management
Chest Trauma

- Can be blunt or penetrating
- Level of injury corresponds with specific anatomical injuries

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<th>Level of Injury</th>
<th>Anatomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>C 4</td>
<td>Hyoid bone</td>
</tr>
<tr>
<td>C 6</td>
<td>Cricoid cartilage</td>
</tr>
<tr>
<td>T 2</td>
<td>Suprasternal notch</td>
</tr>
<tr>
<td>T 4</td>
<td>Aortic arch, trachea bifurcation</td>
</tr>
<tr>
<td>T 6</td>
<td>Pulmonary artery</td>
</tr>
<tr>
<td>T 8</td>
<td>Vena Cava foramen in diaphragm</td>
</tr>
<tr>
<td>T 10</td>
<td>Esophageal hiatus in diaphragm</td>
</tr>
<tr>
<td>T 12</td>
<td>Aortic hiatus in diaphragm</td>
</tr>
<tr>
<td>L 2</td>
<td>Right crus of diaphragm</td>
</tr>
<tr>
<td>L 4</td>
<td>Umbilicus</td>
</tr>
</tbody>
</table>
Chest Trauma: Pulmonary Contusion

- Bruising of pulmonary tissue, usually due to blunt trauma.
- Pathophysiology
  - Causes inflammation
  - Increased capillary permeability
  - Fluid leak cause pulmonary edema
  - WBC’s migrate to the area
  - Fluid, inflammatory debris, damaged cells from pus and disrupt the capillary / alveolar membrane
  - Alveoli collapse
  - Hypoxemia occurs

- Manifestations:
  - Bruising on chest wall
  - Tachypnea, dypsnea, bloody sputum
  - Increased airway pressure, decreased PaO2 / FiO2 ratios

- Treatment:
  - Assure airway
  - Mechanical ventilation with PEEP
  - Negative fluid balance to control pulmonary edema

- MAY LEAD TO ARDS!
Chest Trauma: Rib Fractures

- Simple fractures may result in decreased ventilation due to pain.

- 1\textsuperscript{st} rib fractures are associated with higher incidence of great vessel injury and cervical spine injury.

- Lower rib fractures are associated with abdominal injuries.

- Manifestations:
  - Pleuritic chest pain
  - Contusion
  - Decreased respiratory effort

- Treatment:
  - Splinting
  - Monitor for underlying tissue damage, development of pneumothorax or hemothorax
Multiple fractures may result in flail segments
- Result from 2 or more segments of fractured ribs
- Allows a free floating segment that moves paradoxically
- Lungs do not expand as usual, resulting in hypoxemia
- May damage underlying tissue

Manifestations:
- Pleuritic pain
- Dyspnea
- Crepitus
- Hypoxemia

Treatment
- Oxygen, ventilation
- Stabilize with tape (one side only, do not wrap chest)
- ORIF

Complications
- Pneumothorax
- ARDS
- Atelectasis
Chest Trauma: Hemothorax

- Collection of blood in pleural space
- Source:
  - Left hemothorax
    - Rib fracture 36%
    - Pulmonary tissue 35%
    - Aorta 15%
  - Right hemothorax
    - Rib fracture 51%
    - Pulmonary tissue 27%
    - Liver 10%
- Manifestations:
  - Dyspnea, tachypnea
  - Cyanosis, hypoxemia
  - Shock
- Treatment:
  - Chest drainage
  - Volume replacement
  - Thorocotomy
    - More than 1500 ml blood with initial chest tube insertion
  - Bleeding more than 300 /hr for 3 hours
    - Hemodynamic instability
    - Tension hemothorax
Airway Disorders
Chronic Obstructive Pulmonary Disease (COPD)

- Patients with COPD may have frequent exacerbations that can cause acute respiratory failure
  - Asthma
  - Emphysema
  - Chronic Bronchitis

- Most common precipitating events:
  - Airway infections
  - Right sided heart failure, due to high pulmonary pressures common in COPD
  - Non-compliance with COPD treatment
Chronic Obstructive Pulmonary Disease (COPD)

- More than 14 million Americans affected
  - Cigarette smoking (85-90%, per ALA, 2011)
  - Occupation – coal miners, firefighters
  - Alpha-1 anti-trypsin deficiency

- Results in:
  - **Emphysema** – chronic inflammation
    - Results in air trapping in the alveoli
  - **Chronic bronchitis** – mucus production
    - Results in chronic, productive cough for more than 3 months in 2 consecutive years.

- Symptoms:
  - Productive cough in AM
  - Resistance to airflow causes wheezing, dyspnea,
  - Incidence of pulmonary infections increases
COPD - Treatment

- Bronchodilation – Treats disease immediately
  - Beta 2 agonist
  - Anticholinergic
- Steroids – Reduces airway edema, but effect will not be seen until next day.
- Advair – anti-inflamm/bronchodilator
- Aminophylline: Smooth muscle relaxant
- Oxygen – Best to use controlled delivery device.
- Maintain airway patency – To mobilize thick, tenacious secretions, consider use of:
  - Humidification
  - Hydration
  - Suctioning, percussion, vibration, postural drainage
- Treat infections with appropriate antibiotics
  - Use antipyretics to decrease any fever and O2 consumption
Assisted Ventilation (BiPAP) in COPD

- Avoid mechanical ventilation as long as possible!
  
  Criteria for ventilation:
  - Respiratory muscle fatigue
  - Refractory hypoxemia
  - Respiratory acidosis (pH < 7.30)
  - Cardiovascular instability

- If pCO2 is elevated with normal pH, probably a chronic CO2 retainer
  
  Try Non-Invasive ventilation first!

- If pCO2 is elevated and pH is decreased will likely require mechanical ventilation

Remember:
For non-invasive ventilation to work, must be alert, cooperative and able to handle secretions
Status Asthmaticus

- A recurrent, reversible airway disease characterized by increase airway responsiveness to a variety of stimuli that produce airway narrowing.

- Triggers cause IgE release, which stimulates mast cells to release histamine, causing swelling and inflammation of the smooth muscles of the larger bronchi and mucous membrane swelling and excessive secretion of mucus.

- Airway narrowing is greatest during expiratons. Air is trapped in alveoli, which become hyperinflated.

- Excess mucus causes V/Q mismatch and shunt
  - Has circadian influence:
    - Worse around 3 am.
    - Best around 3 pm.
  - Warnings of impending severe attack:
    - Increased sleep disturbances and use of nocturnal bronchodilators
    - Morning chest stiffness or heaviness
    - Runny nose, sneezing, increase in cough
Asthma - Presentation

- Tachypnea, dyspnea, wheezing due to bronchoconstriction
  - May have increased sputum
  - Absence of rhonchi and wheezing indicates absence of airflow
    **Not a good sign!**

- Anxious, diaphoresis, use of accessory muscles, tachycardia

- Elevation of pCO2 is also a late sign. Usually pCO2 is decreased / normal.
Asthma - Treatment

- Bronchodilators
  - Beta adrenergic agonists – Alupent, Bronchosol
  - Anticholinergic agents – Atrovent

- Steroids to decrease mucosal swelling and histamine release

- IV magnesium
  - Acts as bronchodilator, decreases inflammation

- Antibiotics
  - Strong link between sinus infections and asthma exacerbations

- Hydration – More effective than expectorant
  - Mucolytics are contraindicated because they may cause increased bronchospasm.

- If ventilation is required, avoid high PIP and PEEP
  - Sedation with propofol may increase bronchodilation
Emphysema

- Damaged air sacs in a person's lungs, causing them to lose their elasticity.
- Permanent fissures in the tissues of a person's lungs.
- Limited air supply
Chronic Bronchitis

- Inflammation and swelling of the lining of the airways, leading to narrowing and obstruction of the airways.
- Production of mucous, which can cause further obstruction of the airways.
- Increases the likelihood of bacterial lung infections.
- **Daily Cough**
Pulmonary Hypertension
Pulmonary Hypertension
Pulmonary Hypertension

- A progressive, life threatening disorder of the pulmonary circulation characterized by high pulmonary artery pressures, leading to right ventricular failure.

- Primary pulmonary HTN
  - Associated with autoimmune diseases
  - Mostly effects women in childbearing years
  - Believed to be caused by endothelial dysfunction that leads to re-modeling of the pulmonary artery

- Secondary Pulmonary HTN
  - is due to chronic disorders such as pulmonary fibrosis / sarcoidosis, collagen vascular disease, liver disease, portal hypertension, diet supplements, sleep apnea, HIV

- Signs / symptoms
  - Dyspnea
  - Weakness / fatigue
  - Recurrent syncope
  - Signs of right heart failure
    - Tricuspid murmur
    - Jugular vein distension, pulsation
    - Increased pulmonary pressures
Anticoagulants – Prevent thrombus formation
Diuretics – To control edema
Oxygen / calcium channel blockers – Prevents further vasoconstriction
Pulmonary vasodilators – Some therapy cannot be interrupted or rebound pulmonary hypertension will be so severe that it is fatal!

- Prostacycline inhibitor therapy
  - Flolan (epoprostenol) – IV medication with immediate action and 3-5 minute half life. CANNOT Interrupt!
  - Remodulin (tresprostinil) – Similar to Flolan, longer half life
  - Ventavis (ilopost) – Intermittent inhalation agent

- Phosphodiesterase inhibitors
  - Sildenafil – oral agent

Definitive treatment:
Lung transplant
Mr. Smith, 57, is one-day post abdominal aortic aneurysm (AAA) repair. This morning he developed atrial fibrillation with subjective dyspnea. His HR = 121 but otherwise his vital signs are normal. What pulmonary complications is Mr. Smith suffering from?

a) Pneumonia  
b) ARDS  
c) Asthma  
d) Pulmonary Embolism
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d) **Pulmonary Embolism**
2. How does the D-dimer lab test help to diagnose pulmonary embolism (PE)?

a) A positive test indicates PE
b) A negative test rules out PE
c) A positive test rules out PE
d) A negative test indicates PE
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b) **A negative test rules out PE**

A negative test rules out PE

d) A negative test indicates PE
3. Nursing interventions that decrease the incidence of hospital-acquired pneumonia include:

a) Placing gastric tubes through the nose
b) Administering systemic antibiotics
c) Brushing the patient’s teeth with a toothbrush
d) Keeping the patient NPO
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Questions??

- You Guys ROCK !!!

- Review questions DAILY!

- Stay Confident!