AACN PCCN Review

Pulmonary

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I. INTRODUCTION

PCCN Test Plan

Pulmonary: 14%

a. Acute Lung Injury (ALI)
b. Exacerbation of COPD
c. Obstructive Sleep Apnea
d. Pleural Space Abnormalities and Complications (e.g. pneumothorax, Hemothorax, pleural effusion, empyema)
e. Pulmonary Embolism
f. Pulmonary Hypertension
g. Respiratory Depression (e.g. Mediation-Induced, Decreased-LOC Induced)
h. Respiratory Failure
   • Acute
   • Chronic
i. Respiratory Infections (e.g. Pneumonia)
j. Severe Asthma
k. Thoracic Surgery
   • Lobectomy
   • Pneumonectomy

Pulmonary Testable Nursing Actions

a. Perform a comprehensive pulmonary assessment
b. Monitor normal and abnormal diagnostic test results
c. Interpret ABGs and report findings
d. Monitor patient for response to pulmonary medications
e. Manage patients requiring non-invasive O₂ or ventilation delivery systems
   • nasal cannula, face masks, venti-masks, non-rebreather mask, BiPAP, CPAP
f. Manage patients requiring mechanical ventilation – tracheostomy tube
g. Manage patients requiring respiratory monitoring devices
   • continuous SPO₂, intermittent SPO₂, end-tidal CO₂ (capnography)
h. Recognize signs and symptoms of respiratory complications, and seek assistance as needed
i. Maintain airway
j. Manage patients with chest tubes
k. Assist with procedures
   • thoracentesis, chest tube insertion
l. Administer Medications for procedural (conscious) sedation and monitor patient response


**Review of Anatomy**

a. Lungs  
b. Bronchi  
c. Bronchiole  
d. Alveoli  
e. Air Sacs

**Pulmonary Physiology**

![Diagram](image)

**Gas Exchange at the Alveolar Level**

**Definitions**

a. **Ventilation:** The process of moving air into and out of the lungs  
b. **Diffusion:** The process of molecules of gas pass from an area of high concentration to one of lower concentration. Alveolar Diffusion and Capillary Diffusion  
c. **Perfusion:** The process of transporting gases to the body (capillary) via the circulatory system.  
d. **Dead Space Ventilation:** Alveolus is receiving ventilation but not perfusion (pulmonary emboli)  
e. **Intrapulmonary Shunting:** Alveolus is receiving perfusion but not ventilation (atelectasis, pneumonia)
II. ASSESSMENT AND GAS EXCHANGE

Work of Breathing

a. Respiratory Rate at Rest?
b. Pulse Ox
c. Color? Central and Peripheral (Clubbing?)
d. Dyspnea?
e. Breathing Effort Visibly Labored?
f. Use of Accessory Muscles?
g. Breath Odor? (Sweet or Urine odor)
h. Pain Associated with Deep or Rapid Breathing?
i. Shallow or Uneven Expansion?
j. Cough? Productive? Description of Sputum?
k. Physical Position? Can They Breathe in a Supine Position? Orthopnea?
l. History of Pulmonary Disorder?
   • Smoking*
   • Bronchitis
   • Emphysema
   • Asthma
   • Pneumonia
   • TB
   • Environment Pulmonary Stressor

*Smoking cessation is always a teachable function of nursing

Auscultation

a. Bronchial (Tracheal)
b. Bronchovesicular
c. Vesicular
   • Absent
   • Diminished
   • Displaced
   • Pleural Friction rub
   • Fine Crackles (rales)
   • Course Crackles (rales)
   • Rhonchi
   • Wheeze
   • Stridor
Diagnostic Tests and Monitoring

a. Xrays
b. CT Scan
c. MRI
d. Ventilation/Perfusion Scan (V/Q)
e. Pulmonary Function Tests
f. Biopsy and Thoracentesis
g. Bronchoscopy
h. Polysomnography (sleep study for sleep apnea)
i. Labs: sputum, H&H, CBC
j. Monitoring
   • Continuous SP0₂
   • Intermittent SP0₂
   • End-Tidal CO₂ (capnography)

Healthy Pulmonary Living

a. Don’t Smoke
b. Good Hand Hygiene
c. Annual Flu Vaccine
d. Pneumonia Vaccine
e. Balanced Diet and Adequate Fluid Intake
f. Regular Exercise

Arterial Blood Gas Analysis

<table>
<thead>
<tr>
<th>Arterial Blood Gas Analysis</th>
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<tbody>
<tr>
<td>Variable</td>
</tr>
<tr>
<td>PaO₂</td>
</tr>
<tr>
<td>pH</td>
</tr>
<tr>
<td>PaCO₂</td>
</tr>
<tr>
<td>HC0₃</td>
</tr>
<tr>
<td>SaO₂</td>
</tr>
<tr>
<td>Base Excess</td>
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</tbody>
</table>

a. Indications of Oxygenation Status?
b. Indications of Ventilation Status?
c. Indications of Acid-Base State?
The Physiology of Acid-Base Balance/Imbalance

Respiratory Acid Base Regulation
Ventilation (inspiration & expiration) and Diffusion (movement of gases) are responsible for PaC0₂ levels. Proper acid base balance can be maintained or changed by “blowing off” or “retaining” C0₂ by increasing or decreasing the respiratory rate and/or depth (minute ventilation Ve). Regulation or disruption in the balance can happen very quickly.

Normal PaC0₂
35 - 45mmHg

Hypercapnia
a. >45 mmHg  Hypoventilation
b. Respiratory Acidosis pH < 7.35 & PaC0₂ > 45mmHg
   • Respiratory Depression: Over sedation, overdose, head injury
   • Decreased Ventilation: Neuromuscular disease, mechanical ventilation (hypoventilation)
   • Altered Diffusion: Pulmonary edema, severe atelectasis, pneumonia

Hypocapnia
a. < 35 mmHg  Hyperventilation
b. Respiratory Alkalosis pH > 7.45 & PaC0₂ < 35mmHg
   • Increased Ventilation: anxiety, fear, fever, hypoxia, pain, hypovolemia, head injury, mechanical ventilation

Metabolic Acid Base Regulation
The kidneys regulate the HCO₃ level in the blood by functioning as a buffer system for the acid base balance. The kidneys will retain or excrete bicarb or hydrogen ions and balance or disrupt the pH. This system of balance takes longer than the respiratory system.

Normal HCO₃
22 - 26 mEq/L

Acidosis pH < 7.35, HCO₃ < 22 mEq/L & BE < -2
a. Metabolic Acidosis Conditions That Increase [H⁺]:
   • DKA, renal failure, ingestion of acidic drugs, lactic acidosis
b. Conditions That Decrease Bicarbonate:
   • Diarrhea, GI losses, Body fluid losses, drugs causing a loss of alkali (laxatives)
Alkalosis pH > 7.45, HCO$_3$ > 26 & BE > +2
a. Metabolic Alkalosis Conditions That Increase Alkali:
   • Ingestion of antacids, admin of bicarbonate
b. Conditions that Decrease Acid:
   • Loss of gastric juices (vomiting, high NG output), diuretics

**Evaluation Questions?**

a. What is PaO$_2$?
b. What is pH?
c. What is PaCO$_2$?
d. What is HCO$_3$?
e. Options?

**Options**

a. Normal pH, Normal PaCO$_2$, Normal HCO$_3$ =
b. Acidosis with High PaCO$_2$, Normal HCO$_3$ =
c. Acidosis with Low HCO$_3$, Normal PaCO$_2$ =
d. Alkalosis with Low PaCO$_2$, Normal HCO$_3$ =
e. Alkalosis with High HCO$_3$, Normal PaCO$_2$ =

**Determination of Compensation**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>pH</th>
<th>Primary</th>
<th>Compensation</th>
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<tr>
<td>Resp Acidosis</td>
<td>↓</td>
<td>↑ PaCO$_2$</td>
<td>↑ HCO$_3$</td>
</tr>
<tr>
<td>Resp Alkalosis</td>
<td>↑</td>
<td>↓ PaCO$_2$</td>
<td>↓ HCO$_3$</td>
</tr>
<tr>
<td>Met Acidosis</td>
<td>↓</td>
<td>↓ HCO$_3$</td>
<td>↓ PaCO$_2$</td>
</tr>
<tr>
<td>Met Alkalosis</td>
<td>↑</td>
<td>↑ HCO$_3$</td>
<td>↑ PaCO$_2$</td>
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**III. ACUTE RESPIRATORY FAILURE**

**Definition**
Failure of the pulmonary system to provide adequate oxygenation or ventilation. Sudden drop in PaO$_2$ or elevation in PaCO$_2$

**Pathophysiology**

*Alveolar Hypoventilation*
Respiratory Depression
a. Drugs (includes conscious sedation),
b. Head Injury
c. Muscle Weakness
**Ventilation-Perfusion Mismatching**

a. Increased Dead Space
b. Intrapulmonary Shunting

d. Decreased $O_2$ Consumption: Sepsis, Toxins

**Diffusion Impairment**

a. Hypoventilation
b. Low CO States
c. Low H/H
d. Decreased $O_2$ Consumption: Sepsis, Toxins

**Treatment Options**

a. Ventilate
b. Oxygenate
c. Treat Underlying Cause
d. Treat Acid Base Imbalance
e. Supportive Therapy

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**Airway Management & Oxygen Therapies**

Many pulmonary disorders and acute respiratory distress/failure are treated with airway devices and oxygen therapies. Review devices: oral/nasal airways, nasal cannula, face masks, CPAP, BiPAP – the use, assessment, potential complications and nursing care concerns.

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**IV. RESTRICTIVE LUNG DISORDERS**

Pulmonary Disorders that restrict the lungs from expanding. Lung compliance and volumes are decreased.

**Examples include:**

a. Acute Lung Injury
b. Bacterial Infections
   - Pneumonia
   - TB
   - RSV
   - Lung Abscess
   - Emphysema
   - Fungal Infections
c. Occupational Lung Diseases
d. Sarcoidosis
e. Atelectasis
Common Signs and Symptoms

a. Refractory Hypoxemia
b. Dyspnea
c. Increased WOB, Shallow Breathing

Acute Resp Distress Syndrome (ARDS)

ARDS is a syndrome, not a disease; it is a group of physical manifestations that are primary pulmonary and result from direct or indirect lung injury followed by a significant inflammatory insult. The inflammation and resultant chemical mediator release cause increased capillary permeability, pulmonary edema, and alveolar collapse. These manifestations can, and frequently do, cause lung damage, failure and subsequently death. Treatment typically requires intubation and mechanical ventilation (in the ICU setting) and eliminating the causative factor.

Pneumonia

An Inflammatory Process of the Lung Parenchyma Caused by Infection that Leads to Alveolar Consolidation.

Etiology

Origin
a. Bacterial (75% CAP Streptococcus Pneumoniae)
b. Viral (RSV primarily in children, highly contagious by droplets, inflammation in small airways leads to obstruction)
c. Fungal
d. Aspiration

Site
a. Bronchial
b. Alveolar
c. Lobar

Source
a. Community Acquired Pneumonia (CAP)
b. Hospital Acquired Pneumonia (HAP)
c. Ventilator Acquired Pneumonia (VAP)

Pathophysiology
a. Lower Respiratory Tract Invasion
b. Inflammatory Reaction
c. Increased Capillary Permeability
d. Phagocytotic Cells Migrate to Site
e. Alveoli Fill with Exudate
Clinical Presentation
a. Dyspnea & Tachypnea
b. Productive Cough
c. Pleuritic Chest Pain
d. Fever, Chills, Rigors, Fatigue
e. Anorexia
f. Night Sweats
g. Pleural Effusion
h. Crackles, Rhonchi
i. Tachycardia

Diagnostic Measures
a. Chest X-Ray: Localized Infiltration
b. Sputum Culture: Positive for Microbes
c. CBC: Positive for Infection Elevated WBC Count
d. Bronchoscopy: Visualize Inflammation/Consolidation

Treatment Options
a. Antibiotics
b. Oxygen
c. Mechanical Ventilation
d. Positioning: Good Lung Down
e. Fluids & Humidification
f. Pulmonary Hygiene
g. Manage Fever & Pain
h. Prevention
   • Hand Washing
   • Sterile Suctioning
   • Mouth Care
   • HOB ≥ 45°
   • Pneumococcal Vaccine
   • Stress Ulcer Prophylaxis
   • Extubate ASAP

V. OBSTRUCTIVE LUNG DISORDERS
Pulmonary Disorders where airway obstruction and gas trapping are the primary problem. Expansion and compliance of the lung tissue is not the problem. Examples include:
a. Chronic Obstructive Pulmonary Disease (COPD)
   • Emphysema
   • Bronchitis
b. Asthma
Chronic Obstructive Pulmonary Disease

**Etiology**

**Bronchitis**
a. An inflammatory response to an irritant (infectious or noninfectious) that results in vasodilation, congestion, mucosal edema, and bronchospasm. Affects the small and large airways rather than the alveoli.
   - Chronic Bronchitis: Chronic cough w sputum production \( \geq \) 3 months per year for 2 successive years.

**Emphysema**
Smoking is the #1 cause. Other causes include occupational exposure to certain particles (coal dust, asbestos, firefighters) & Alph\( _1 \)-Antitrypsin Disease.

**Pathophysiology (Emphysema)**
a. Irritation and Inflammation of Bronchioles \( \rightarrow \) Mucus Production \( \rightarrow \) Obstruction \( \rightarrow \) Tissue Injury \( \rightarrow \) Decrease Surfactant \( \rightarrow \) Bronchiolar Collapse
b. Obstruction \( \rightarrow \) Air Trapping and Distention of Alveoli \( \rightarrow \) Enlargement of Air Sacs and Loss of Elastic Recoil \( \rightarrow \) Multiple Alveoli Actually Fuse to One Large One \( \rightarrow \) Decreasing Surface Area for Gas Exchange
c. Increases in FRC
d. Hypoxia
e. V/Q Mismatch
f. Pulmonary Hypertension
g. Increased RV Afterload \( \rightarrow \) Right Heart Failure (Cor Pulmonale) \( \rightarrow \) RV Hypertrophy \( \rightarrow \) Drop in LV Filling/CO

**Clinical Presentation**
a. Dyspnea on Exertion \( \rightarrow \) Dyspnea at Rest
b. Productive Cough \( \rightarrow \) Non Productive Cough
c. Tachypnea with Small Tidal Volume
d. Dropping FEV\( _1 \)
e. Malnutrition/Muscle Wasting (including diaphragm)
f. Increase in AP Diameter
g. Diminished Breath Sounds in Bases
h. PFTs:
   - Increased: FRC, RV, TLC
   - Decreased: FEV\( _1 \), TV
i. ABG: Hypoxia w Respiratory Acidosis. Over Time will Develop a Degree of Metabolic Compensation
j. Example: \( \text{PaO}_2 \ 71, \text{PaCO}_2 \ 52, \text{pH} \ 7.29, \text{HC0}_3 \ 34, \text{SaO}_2 \ 72 \)
k. Chest X-Ray: Flattened Diaphragm, Decreased Vascular Markings and Bullae
l. Right Heart Failure
m. Chronic Multi-System Dysfunction Related to Chronic Hypoxemia and Hypercapnia
Acute Care Concerns
COPD is a chronic medical condition. It is relevant to the PCCN exam because these patients are frequently admitted for problems that require acute care as a result of their chronic debilitated state. Assessment and treatment options MUST take into consideration their pulmonary function and dysfunction.

Common Reasons for Acute Care Admission
a. Pneumonia
b. Heart Failure
c. Pulmonary Emboli
d. Respiratory Failure
e. Bronchospasm
f. Spontaneous Pneumothorax
g. None Compliance with Pulmonary Medical Therapies

Treatment Options
a. Treat Primary Cause of Admission
b. Oxygen Administration (with caution)
c. Hydration & Humidification
d. Removal of Secretions
e. Pharmacology
   • Antibiotics
   • Steroids
   • Bronchodilators
   • Mucolytics
f. Nutritional Support (high calorie, low carbohydrate)

Severe Asthma

Etiology
A hyperactive airway due to an intrinsic or extrinsic factor. Common causes include:
a. Respiratory Infection
b. Allergic Reaction to Inhaled Antigen
c. Emotional Stress
d. Exercise
e. Idiosyncratic Reaction to NSAID or Beta Blocker
f. Environment Toxin
g. Mechanical Stimulation (coughing, laughing, cold air)
h. Reflux Esophagitis

Pathophysiology
a. A disease of inflammation that precipitates bronchospasm (obstruction).
b. Affects airways not alveoli and the bronchospasm is reversible
c. Inflammation precipitates mucus production (more obstruction)
d. Obstruction leads to air trapping and difficulty with expiration (harder, longer and less effective)
e. Decreased Oxygenation and Carbon Dioxide Removal
f. Acute Asthma or Status Asthmaticus: the individual’s “typical” asthma therapies don’t work, the bronchospasm, mucus production and air trapping continue potentially to the point where there is no air movement.
g. Hyperinflation increases intrathoracic pressures which decreases venous return and increases RV afterload

Clinical Presentation
a. Stimulation of Asthma Unrelieved by Typical Tx
b. Increased Work of Breathing
c. Rapid RR with Little Air Movement (Air Trapping)
d. Long Expiratory Phase
e. Expiratory Wheezes Initially \(\rightarrow\) Minimal to No Air Movement on Inspiration or Expiration
f. Pulsus Paradoxus
g. Restless and Anxious, Calming Down is a Bad Sign

Diagnostic tests
a. ABG: Hypoxia and Hypercapnia – Resp Acidosis
b. PFT: Drop in FEV\(_1\) & Peak Expiratory Flow

treatment Options
a. Psychological Support
b. Oxygen Therapy
c. Maybe Mechanical Ventilation (big ETT), Low TVs
d. Remove Irritate (if known)
e. Hydration & Humidification
f. Pharmacological
   - Bronchodilators
   - ABX
   - Corticosteroids
   - Inhaled Anticholinergic Agents
   - Heliox
   - Sedatives & Muscle Relaxants
   - SubQ Epinephrine
f. Monitor and Treat Pneumothorax
g. Monitor and Treat Heart Failure

VI. OBSTRUCTIVE SLEEP APNEA

(OSA) is the most common type. The pt experiences repeated episodes of apnea (stop breathing for > 15 seconds) during sleep secondary to upper airway obstruction. Central Sleep Apnea is not from upper airway obstruction but due to a dysfunction in the brain and sleep
center. These apnea episodes can last up to 20 seconds (see box for information specific to central sleep apnea).

**Etiology**

a. Obesity (> 50% of pts)
b. Neck Circumference > 17 inches
c. Nasal Obstruction
d. Men > Women
e. Postmenopausal Women
f. Age > 50 Years Old
g. Smoking
h. Hypertension

**Pathophysiology**

In OSA there is an occlusion of the upper airway (nasopharynx and oropharynx) leading to obstruction. This typically happens during REM sleep because the muscle structures become hypotonic. The obstruction typically leads to snoring. The snoring and lack of ventilation awakens the patient, they clear the obstruction and return to sleep. The repeated apnea episodes and subsequent sleep interruptions lead to acute and potential chronic health problems.

**Clinical Presentation**

a. Daytime Sleepiness/Fatigue
b. Morning Headaches
c. Cognitive, Personality, Behavioral Changes
d. Increased Potential for Trauma
e. Sexual Dysfunction
f. Night Sweats
g. Excessive Snoring at Night
h. Repeated Sudden Awakenings
i. Potential Long Term Complications
   - Hypertension (diurnal and pulmonary)
   - Cor Pulmonale
   - Stroke
   - MI

**Diagnostic Tests**

a. Sleep Study (Polysomnography)
b. Pulmonary Function Tests
c. ABG
d. Wt and Neck Measurements (not diagnostic)
Treatment Options

a. Wt Loss
b. Mechanical Tx – CPAP or BiPAP
c. Dental or Oral Appliances (airways)
d. Surgery
e. Maintain HOB > 30°
f. Avoid ETOH and Sedatives Before Sleep

Central Sleep Apnea
Apnea episodes during sleep caused from a problem in the brain not obstruction in upper airway. Common etiologies include:
a. Parkinson’s Disease
b. Alzheimer’s Disease
c. Damage to Brainstem: Encephalitis, Stroke TBI
d. Cervical Spine Damage: Injury, Radiation, Severe Arthritis, Degenerative Bone Disorders
Treatment centers on treating the primary cause of the apnea

VII. PULMONARY EMBOLI

Occlusion in the pulmonary arterial circulation, blocking flow to a region(s) of the lung and creating dead space ventilation.

Etiology

a. Fat
b. Air
c. Amniotic Fluid
d. Thromboembolic – 90% of all PEs from DVT

Virchow’s Triad
a. Venous Stasis
   • Immobility
   • Dehydration
   • Pregnancy/Oral Contraception/Hormone Replacement Therapy
   • Paralysis
   • Obesity
b. Hypercoagulability
   - A-Fib
   - Tumors/Cancer
   - Dehydration
   - Heart Failure (also immobility)
   - COPD Pt 2° A-Fib, Polycythemia
   - Previous PE

c. Vascular Wall Damage
   - Trauma
   - Venous Catheters
   - Varicose Veins
   - Elevated LDL
   - Age

Pathophysiology

a. The pathophysiology and presentation must be viewed on a continuum. It will depend on the size of the blockage and length of time it has been occurring.
b. Pulmonary Artery Obstruction
c. V/Q Mismatching V > Q = Dead Space Initially
d. Non Perfused Alveoli will Collapse Secondary to Decreased Surfactant Production
   → Intrapulmonary Shunting
e. Pulmonary Infarction May Occur
f. Increased Pulmonary Vascular Resistance Increases Afterload on Right Venticule
   → RV Failure and Potentially Infarction

Clinical Presentation

a. Sudden Onset Dyspnea & Pleuritic Chest Pain
b. Tachypnea
c. Refractory Hypoxemia
d. ABG: Hypoxemia with Respiratory Alkalosis
   - Example: PaO₂ 71, PaCO₂ 29, pH 7.59, HCO₃ 25, SaO₂ 72
e. Fat Emboli: Petechiae on Thorax and Upper Extremities
f. Cardiac
   - Tachycardia
   - Cyanosis
   - Jugular Venous Distention
   - RV Failure (Increased Resistance/Afterload & Loud S2)
   - ECG: RV Hypertrophy, T wave Abnormalities
Diagnostic Tests

a. Chest X-Ray – Not Diagnostic but Rules Out Other Causes for the Respiratory Distress
b. V/Q Scan
c. Spiral CT
d. + D-Dimer
e. Pulmonary Angiogram
f. MRI
g. Lower Extremity Doppler Studies (not emergent)

Treatment Options

a. ABCs
b. Oxygen administration
c. Intubated if Necessary
d. Consider Thrombolytics
e. Consider Embolectomy
f. IVC Filter Placement
g. Pain Management
h. Identify Causative Factor and Treat
i. Future Prevention

VIII. THORACIC SURGERY

Common Thoracic Surgeries

Most tissue related lung surgeries are for lung cancer. Segmental or wedge resections are also done for localized inflammatory processes, TB, or abscess. Lung reduction surgeries are primarily done for emphysema.

**Tracheal Surgery:** Placement of temporary to permanent trach for airway management  
**Pneumonectomy:** Removal of entire side of lung  
**Lobectomy:** Removal of one lobe  
**Segmental Resection:** Removal of one segment. Right side has 10 segments and the left has 8.  
**Wedge Resection:** Only a portion, typically a well circumscribed diseased portion  
**Decortication:** A fibrinous peel is removed from the visceral pleura, allowing for re-expansion of the lung. Typically done for empyema.
Nursing Care
a. Oxygen Therapy
b. Hemodynamic Monitoring: CVP
c. Positioning
d. Initiating Coughing and Deep Breathing
e. Promote Abdominal Breathing
f. Pain Management
g. Nutrition
h. Chest Tube Drainage System
   • Assisting with Chest Tube Insertion
   • Monitoring Patient with Chest Tube
i. Assess for Subcutaneous Emphysema
j. Assess for Thoracic Air Leaks
k. Special Tx for Pneumonectomy:
   • Cannot lie on operative side
   • Palpate trachea for midline position

Pleural Space Abnormalities

Pathophysiology
Air Enters the Pleural Space from a tear in the visceral or parietal pleura as the result of Blunt or Penetrating Chest Trauma. Iatrogenic causes include central line placement, invasive chest procedures (biopsy, thoracentesis) and mechanical ventilation (barotrauma). Occasionally spontaneous. Lung collapses because of the change in intrapleural pressure.

Clinical Presentation
Classifications
a. Tension Pneumothorax
b. Simple Pneumothorax
c. Hemothorax
d. Hemo/Pneumothorax
e. Pneumomediastium
f. Sucking Chest Wound
   • Respiratory Distress (degree depends on classification)
   • Tachycardia and Hypotension
   • Diminished Breath Sounds Over Affected Area
   • Tension: Tracheal Deviation, JVD
   • Visualized on Chest X-Ray
   • Hypoxia on ABG
Treatment Options

a. Emergent Needle Decompression (tension)
b. Chest Tube Placement (re-expand lungs & evacuate air/blood)
   - Insert High for Pneumo
   - Insert Low for Hemo
   - Suction Utilized
   - Potential for Air Leak
   - Milking (not stripping) Recommended for Clot Removal
c. Oxygen Delivery & Potentially Intubation
d. Sucking Chest Wound: Occlusive dressing on expiration. Monitor status carefully might need flutter valve
e. Air Embolism: Trendelenberg position and Left Side to Trap Air in Heart (RV)
f. Surgery May be Required