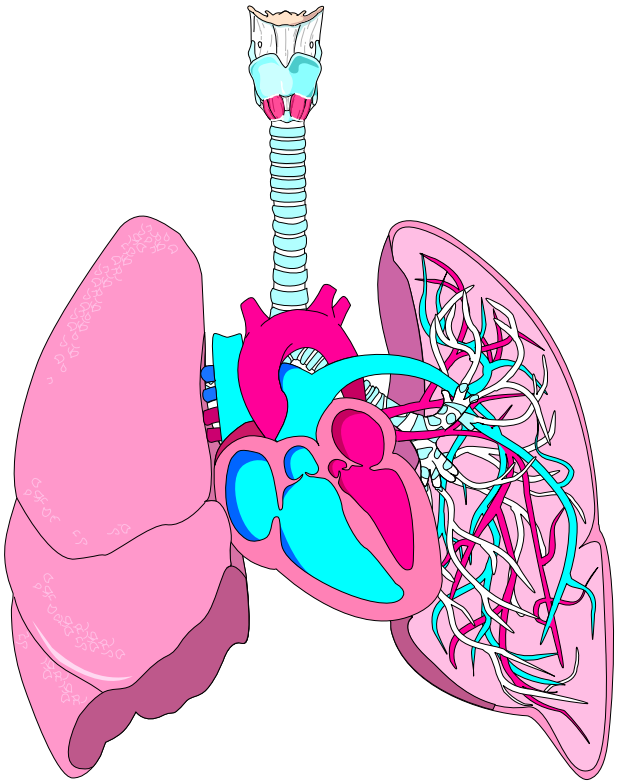
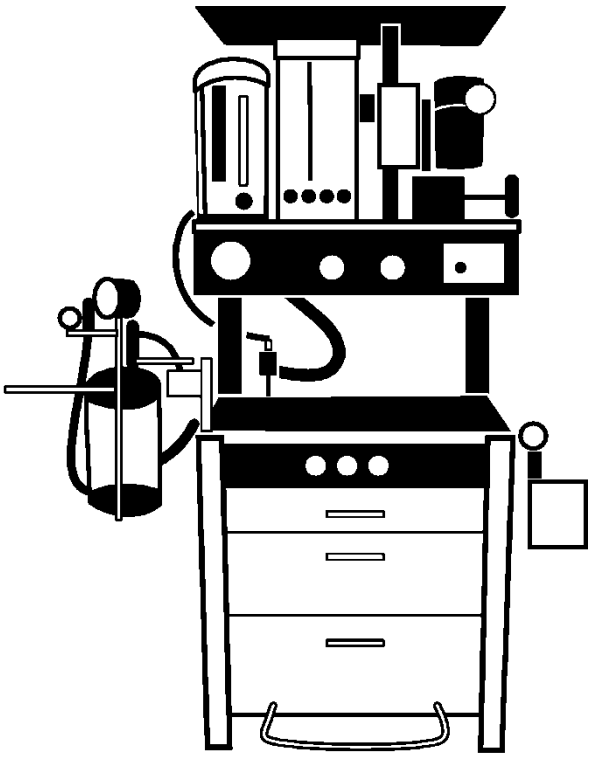
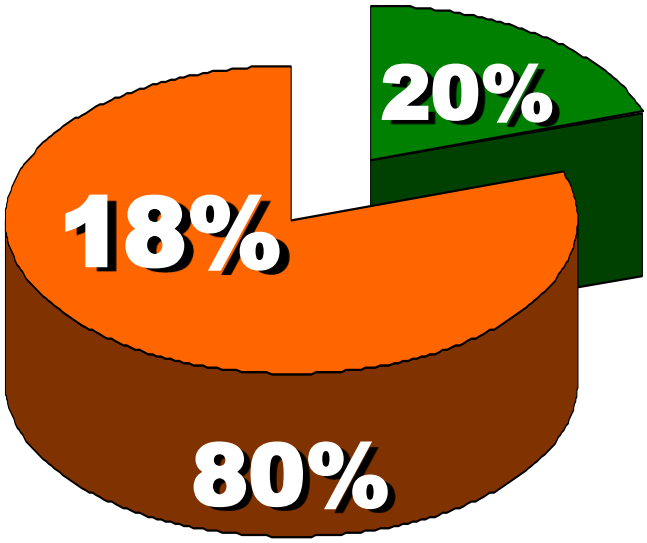
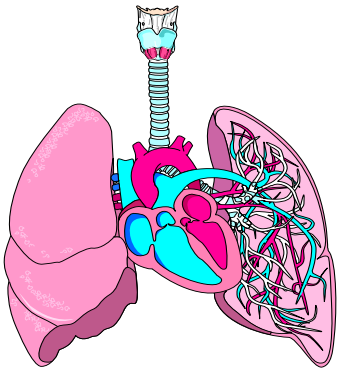
Pulmonary







PULMONARY

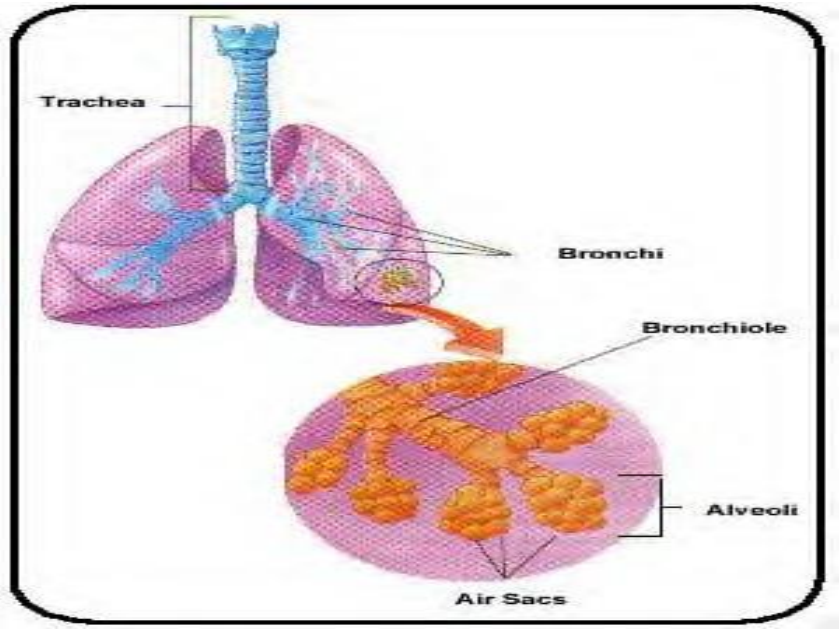


I. Introduction

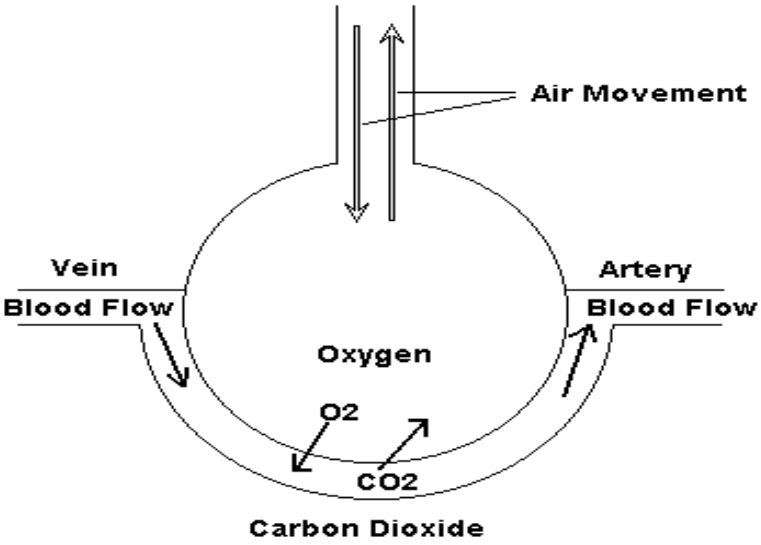
A. AACN-CCRN/CCRN-E Blueprint 18%

- Acute Lung Injury (e.g. ARDS, RDS)
- Acute Pulmonary Embolus
- Acute Respiratory Failure
- Acute Respiratory Infections (e.g. acute pneumonia, bronchiolitis)
- Air-Leak Syndromes (e.g. Pneumothorax, PIE, pneumopericardium)
- Aspiration (e.g. aspiration pneumonia, foreign-body)
- COPD, Asthma, Chronic Bronchitis, emphysema
- Pulmonary Hypertension
- Status Asthmaticus
- Thoracic Surgery
- Thoracic Trauma (e.g. fractured ribs, lung contusions, tracheal perforation)

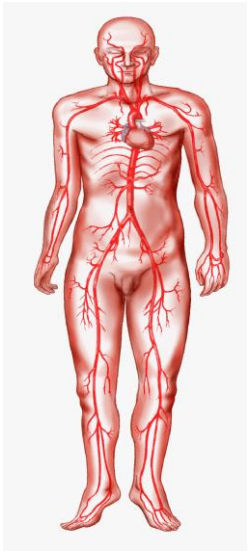
B. Review of Anatomy

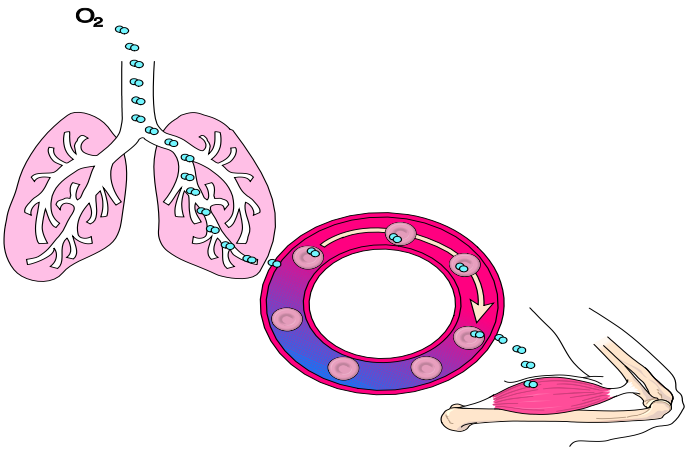


C. Pulmonary Physiology



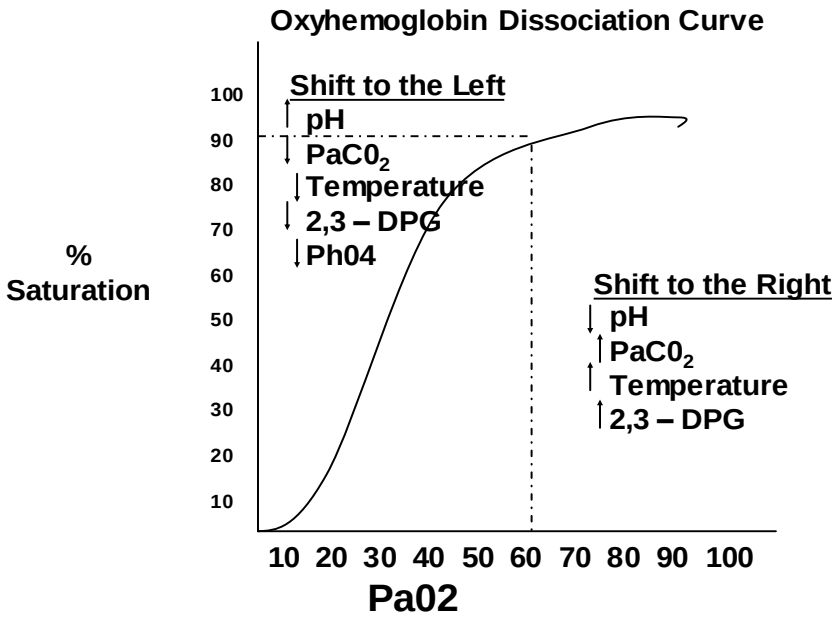
Gas Exchange at the Alveolar Level





1. Definitions
   - ❍ Ventilation: The process of moving air into and out of the lungs
   - ❍ Diffusion: The process of molecules of gas pass from an area of high concentration to one of lower concentration. Alveolar Diffusion and Capillary Diffusion
   - ❍ Perfusion: The process of transporting gases to the body (capillary) via the circulatory system.

Oxygen Hemoglobin Relationship



- Dead Space Ventilation: Alveolus is receiving ventilation but not perfusion (pulmonary emboli)

- Intrapulmonary Shunting: Alveolus is receiving perfusion but not ventilation (atelectasis, pneumonia)

II. Assessment of Gas Exchange

Variable	Normal Arterial Range
Pa0$_2$	80 - 100mmHg
pH	7-35 - 7.45
PaC0$_2$	35 - 45mmHg
HC0$_3$	22 - 26mEq/L
Sa0$_2$	> 95%
Base Excess	-2 - + 2

Indications of Oxygenation Status?

Indications of Ventilation Status?

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 $PaC0_2$ levels. Proper acid base balance can be maintained or disrupted by "blowing off" or "retaining" $C0_2$ by increasing or decreasing the respiratory rate and/or depth (minute ventilation Ve). Regulation or disruption in the balance can happen very quickly. Normal $PaC0_2$ 35 - 45mmHg

Metabolic Acid Base Regulation: The kidneys regulate the $HC0_3$ 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 $HC0_3$ 22 - 26 mEq/L

Options

Normal pH, Normal $PaC0_2$, Normal $HC0_3$	=	Normal
Acidosis with High $PaC0_2$, Normal $HC0_3$	=	Resp Acid
Acidosis with Low $HC0_3$, Normal $PaC0_2$	=	Met Acid
Alkalosis with Low $PaC0_2$, Normal $HC0_3$	=	Resp Alk
Alkalosis with High $HC0_3$, Normal $PaC0_2$	=	Met Alk

Determination of Compensation

Disorder	pH	Primary	Compensation
Resp Acidosis	⇓	⇑ $PaC0_2$	⇑ $HC0_3$
Resp Alkalosis	⇑	⇓ $PaC0_2$	⇓ $HC0_3$
Met Acidosis	⇓	⇓ $HC0_3$	⇓ $PaC0_2$
Met Alkalosis	⇑	⇑ $HC0_3$	⇑ $PaC0_2$

Full discussion of ABG and practice examples page 170

III. Ventilator Management (modes are classified by inspiratory trigger)

A. Volume Cycled Ventilator Modes: Volume is Set, Pressure is Variable Depending in the Compliance of the Lungs

1. Controlled Mandatory Ventilation (CMV)
   - ❍ Complete ventilator control
   - ❍ Inspiration is triggered by time
   - ❍ Volume is set and the set amt is delivered with each breath
   - ❍ The ventilator has no regard for what the patient's own respiratory effort
   - ❍ Used with general anesthesia
   - ❍ Not tolerated well with conscious patient who has any respiratory effort

2. Assist/Control Mode (AC)
   - ❍ Inspiration is triggered by time
   - ❍ A preset TV is delivered at the set rate
   - ❍ Should the patient initiate a breath (trigger the vent) the preset TV is delivered
   - ❍ Used post anesthesia
   - ❍ Hyperventilation can occur

3. Synchronized Intermittent Mandatory Ventilation (SIMV)
   - ❍ Inspiration is triggered by time
   - ❍ A preset TV is delivered at the set rate
   - ❍ Spontaneous breath initiated by the patient will not be augmented to the preset TV, unless the breath happens to occur at or near the same time as the "planned" breath
   - ❍ Used with weaning the patient from the ventilator

B. Pressure Cycled Ventilator Modes: Pressure is Set, Volume is Variable

1. Continuous Positive Airway Pressure (CPAP)
   - ❍ No preset VT or rate
   - ❍ It is a spontaneous breathing mode of ventilation
   - ❍ A continuous pressure is delivered by the machine
   - ❍ Used when the pt has an adequate respiratory drive and muscle strength but needs a little support. Also used as a weaning mode

2. Pressure Support (PS)
   - ❍ PS is used in combination with SIMV
   - ❍ On the spontaneous breaths a set amount of pressure is delivered to assist the patient reach a higher TV than they would on their own
   - ❍ No preset TV is delivered, on spontaneous breaths, the patient determines the TV but it is augmented by the pressure that is delivered at the beginning (only the beginning) of the breath
   - ❍ Used with weaning

3. Pressure Control (PC)
   - ❍ Ventilation is time triggered with a preset inspiratory pressure
   - ❍ The TV will vary depending on the compliance of the lungs
   - ❍ The pressure is delivered in a decelerating pattern and the patient has no control over the ventilatory pattern
   - ❍ The level at which to set the pressure is determined by the pressure needed to deliver the desirable TV
   - ❍ The mean pressure in the lungs goes up with PC but the peak inspiratory pressure (PIP) actually goes down
   - ❍ Used primarily in ALI and in conditions with poor lung compliance (stiff lungs)
   - ❍ This is not tolerated well by the conscious patients. Sedation must be used
   - ❍ High potential for barotrauma

4. Inspiratory/Expiratory Ratio
   - ❍ Normal respiratory effort 1:2

- ❍ In PC this can be changed 1:1, 2:1, 3:1, 4:1 to increase pressure (volume) and oxygenation delivery time
- ❍ Not tolerated well at all – pt should be given sedatives and paralytics

5. Bilevel Positive Airway Pressure (BiPAP)
   - ❍ Noninvasive (spontaneous breathing) mode via Face Mask
   - ❍ Preset Inspiration Pressure
   - ❍ Preset Expiration Pressure
   - ❍ Augments Alveolar Ventilation
   - ❍ Improves Oxygenation

C. Complications of Ventilator Therapy

- ❍ Technical Problems w Ventilator or Settings
- ❍ Airway Problems
- ❍ Ventilator Associated Pneumonia
- ❍ Physiological Changes with use of Positive Pressure Ventilation
  - Increases in Intrathoracic Pressure
  - Decreased Preload
  - Decreased Cardiac Output
  - Fluid Retention
- ❍ Oxygen Toxicity
- ❍ Barotrauma & Volutrauma

IV. Acute Respiratory Failure

A. Definition: Failure of the Pulmonary System to Provide Adequate Oxygenation or Ventilation. Sudden Drop in $Pa0_2$ or Elevation in $PaC0_2$

B. Pathophysiology

- ❍ Alveolar Hypoventilation
  - Respiratory Depression: Drugs, Head Injury, Muscle Weakness
- ❍ Ventilation-Perfusion Mismatching
  - Increased Dead Space
  - Intrapulmonary Shunting
- ❍ Diffusion Impairment
  - Hypoventilation
  - Low CO States
  - Low H/H

- Decreased 0_2 Consumption: Sepsis, Toxins

C. Treatment Options

- ❍ Ventilate
- ❍ Oxygenate
- ❍ Treat Underlying Cause
- ❍ Treat Acid Base Imbalance
- ❍ Supportive Therapy

V. Restrictive Lung Disorders: Pulmonary Disorders that restrict the lungs from expanding. Lung compliance and volumes are decreased**.** Examples include:

- ❍ Acute Respiratory Distress Syndrome (ARDS)
- ❍ Bacterial Infections
  - o Pneumonia
  - o TB
  - o Lung Abscess
  - o Emphysema
  - o Fungal Infections
- ❍ Occupational Lung Diseases
- ❍ Sarcoidosis
- ❍ Atelectasis

Common Signs and Symptoms:

- ❍ Refractory Hypoxemia
- ❍ Dyspnea
- ❍ Increased WOB, Shallow Breathing

A. Acute Respiratory 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 increase capillary permeability, pulmonary edema, and alveolar collapse. These manifestations can, and frequently do, cause lung damage, failure and subsequently death.

1. Etiology

- ❍ Sepsis (#1 cause)
- ❍ Shock (Hypoperfusion States)
- ❍ Systemic Inflammatory Response Syndrome (SIRS)
- ❍ Pulmonary Contusion
- ❍ Trauma
- ❍ Intravenous Fluid Overload
- ❍ Massive Blood Transfusions
- ❍ Pulmonary, Fat or Amniotic Embolism
- ❍ Smoke or Toxic Chemical Inhalation
- ❍ Pulmonary Aspiration
- ❍ Near Drowning
- ❍ Narcotic Overdose
- ❍ Pancreatitis
- ❍ Head Injury
- ❍ Severe Anemia
- ❍ Disseminated Intravascular Coagulation
- ❍ High-Altitude Sickness
- ❍ Eclampsia
- ❍ Cardiopulmonary Bypass
- ❍ Anesthesia

2. Pathophysiology

- ❍ Direct or Indirect Lung Injury
- ❍ Systemic Inflammatory Response Syndrome (SIRS)
- ❍ Increased Capillary Permeability
- ❍ Pulmonary Edema
- ❍ Decreased Surfactant and Alveolar Collapse
- ❍ Ventilation/Perfusion V/Q Mismatching
- ❍ Refractory Hypoxemia
- ❍ Shunting
- ❍ Further Inflammation from Hypoperfusion
- ❍ Development of Hyaline Membranes and Scarring
- ❍ Decreased Compliance
- ❍ Increased Airway Resistance
- ❍ Pulmonary Hypertension
- ❍ Gas Exchange is Virtually Impossible

3. **Diagnostic Criteria for ARDS**
   - ❍ 1994
   - ❍ Acute Onset
   - ❍ $Pa0_2/Fi0_2$ Ratio $\leq$ 200mmHg (ALI $\leq$ 300mgHg)
   - ❍ Bilateral Infiltrates
   - ❍ No Evidence of LV Failure (PAOP < 18mmHg)

The Berlin Definition of ARDS – JAMA vol 307, No 23 (June 2012)

Timing	Within **1 week** of known clinical insult or new or worsening resp symptoms
Chest Imaging	**Bilateral opacities** – not fully explained by effusion, lobar/lung collapse, or nodules
Origin of Edema	**Resp failure** not fully explained by cardiac failure or fluid overload.
Oxygenation:	
Mild	200 mmHg < $Pa0_2/Fi0_2 \leq$ 300mmHg w PEEP or CPAP $\geq$ 5cm H_20
Moderate	100 mmHg < $Pa0_2/Fi0_2 \leq$ 200mmHg w PEEP $\geq$ 5 cm H_20
Severe	$Pa0_2/Fi0_2 \leq$ 100mmHg w PEEP $\geq$ 5 cm H_20

4. Syndrome Progression

- ❍ Early Exudative Phase: Initiated within 24hr lasting up to 4 days
  - Increased Capillary Permeability
  - Tachypnea, Restlessness
  - Respiratory Alk
  - None to Mild Hypoxia

- ❍ Proliferative Phase: Pulmonary Edema days 3-10
  - Fine Crackles or Rales
  - Interstital/Alveiolar Infiltrates on Chest X-Ray
  - Hypoxia and Decreased Oxygen Saturation
  - Increased WOB
  - Agitation

- ❍ Fibrotic Phase: Lung Tissue Damage day 7-14
  - Worsening Hypercarbia and Hypoxemia
  - Lactic Acidosis/Anaerobic Metabolism
  - MODS

5. Treatment Options (supportive not curative)

- ❍ Treatment for Underlining Cause of Lung Injury
- ❍ Mechanical Ventilation
  - Positive pressure ventilation can reverse and prevent atelectasis
  - Reduce work of breathing
  - Improve gas exchange
  - Decrease respiratory muscle fatigue
  - Improving compliance
  - The challenge is to meet physiologic needs of the pt with minimal lung damage
  - Modes of Ventilation
    - PC
    - PC R I:E
    - High frequency jet ventilation
    - APRV aka BiLevel
    - Nitric Oxide
    - Permissive Hypercapnia
    - Independent Lung Ventilation
- ❍ Preventing Infection
- ❍ Pharmacologic Support
  - Pulmonary Dilators
  - Vasoconstrictors
  - Anti-Inflammatory Agents
  - Antioxidants
  - Steroids
  - Narcotics
  - Sedatives
  - Fluids
- ❍ Patient Positioning

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

1. Etiology

- ❍ **Origin**
  - Bacterial
  - Viral
  - Fungal
  - Aspiration

- **Site**
  - Bronchial
  - Alveolar
  - Lobar
- **Source**
  - Community Acquired Pneumonia (CAP)
  - Hospital Acquired Pneumonia (HAP)
  - Ventilator Acquired Pneumonia (VAP)

2. Pathophysiology

- Lower Respiratory Tract Invasion
- Inflammatory Reaction
- Increased Capillary Permeability
- Phagocytotic Cells Migrate to Site
- Alveoli Fill with Exudate
- Impair Gas Exchange From Shunting

3. Clinical Presentation

- Dyspnea & Tachypnea
- Productive Cough
- Pleuritic Chest Pain
- Fever, Chills, Rigors, Fatigue
- Anorexia
- Night Sweats
- Pleural Effusion
- Crackles, Rhonchi
- Tachycardia

4. **Diagnostic Measures**

- Chest X-Ray: Localized Infiltration
- Sputum Culture: Positive for Microbes
- CBC: Positive for Infection Elevated WBC Ct
- Bronchoscopy: Visualize Inflammation/Consolidation

5. **Treatment Options**

- Antibiotics
- Oxygen
- Mechanical Ventilation
- Positioning: Good Lung Down
- Fluids & Humidification

- ❍ Pulmonary Hygiene
- ❍ Manage Fever & Pain
- ❍ Prevention
  - Hand Washing
  - Sterile Suctioning
  - Mouth Care
  - HOB $\geq 45^0$
  - Pneumococcal Vaccine
  - Stress Ulcer Prophylaxis
  - Extubate ASAP

VI. 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:

- ❍ Chronic Obstructive Pulmonary Disease (COPD)
  - Emphysema
  - Bronchitis
- ❍ Asthma

A. Chronic Obstructive Pulmonary Disease

1. Etiology

- ❍ Bronchitis: 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.

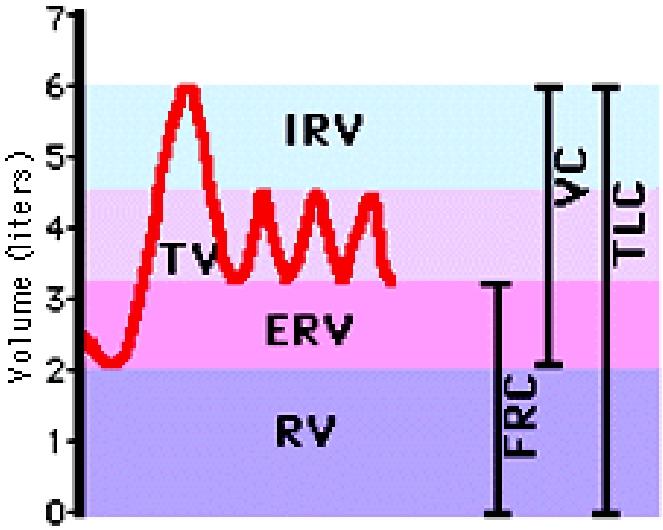
- ❍ Emphysema: Smoking is the #1 cause. Other causes include occupational exposure to certain particles (coal dust, asbestos, firefighters) & $Alph_1$-Antitryspsin Disease.

2. Pathophysiology (Emphysema)

- ❍ Irritation and Inflammation of Bronchioles → Mucus Production → Obstruction → Tissue Injury → Decrease Surfactant → Bronchiolar Collapse
- ❍ Obstruction → Air Trapping and Distention of Alveoli → Enlargement of Air Sacs and Loss of Elastic Recoil →

Multiple Alveoli Actually Fuse to One Large One → Decreasing Surface Area for Gas Exchange

- ❍ Increases in FRC – volume remaining in lungs after exhale
- ❍ Hypoxia
- ❍ V/Q Mismatch
- ❍ Pulmonary Hypertension
- ❍ Increased RV Afterload → Right Heart Failure (Cor Pulmonale) → R to L Septal Shift → Drop in LV Filling/CO



<u>Pulmonary Function Tests</u> (PFTs)

- ❍ Total Lung Capacity
- ❍ Vital Capacity
- ❍ Functional Residual Capacity
- ❍ Tidal Volume
- ❍ Inspiratory Residual Volume
- ❍ Expiratory Residual Volume
- ❍ Residual Volume

3. Clinical Presentation

- ❍ Dyspnea on Exertion → Dyspnea at Rest
- ❍ Productive Cough → Non Productive Cough
- ❍ Tachypnea with Small Tidal Volume
- ❍ Dropping FEV_1
- ❍ Malnutrition/Muscle Wasting (including diaphragm)
- ❍ Increase in AP Diameter
- ❍ Diminished Breath Sounds in Bases
- ❍ PFTs:
  - Increased: FRC, RV, TLC
  - Decreased: FEV_1, TV
- ❍ ABG: Hypoxia w Respiratory Acidosis Over Time will Develop a Degree of Metabolic Compensation
  Example: $Pa0_2$ 71, $PaC0_2$ 52, pH 7.29, $HC0_3$ 34, $Sa0_2$ 72
- ❍ Chest X-Ray: Flattened Diaphragm, Decreased Vascular Markings and Bullae
- ❍ Right Heart Failure

- ❍ Chronic Multi-System Dysfunction Related to Chronic Hypoxemia and Hypercapnia

4. **Critical Care Concerns:** COPD is a chronic medical condition. It is relevant to the CCRN exam because these patients are frequently admitted for problems that require critical 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 CC Admission

- ❍ Pneumonia
- ❍ Heart Failure
- ❍ Pulmonary Emboli
- ❍ Respiratory Failure
- ❍ Bronchospasm
- ❍ Spontaneous Pneumothorax
- ❍ None Compliance with Pulmonary Medical Therapies

5. **Treatment Options**

- ❍ Treat Primary Cause of Admission
- ❍ Oxygen Administration (with caution)
- ❍ Hydration & Humidification
- ❍ Removal of Secretions
- ❍ Pharmacology
    - Antibiotics
    - Steroids
    - Bronchodilators
    - Mucolytics
- ❍ Nutritional Support (high calorie, low carbohydrate)
- ❍ Lung Reduction Surgery

B. **Asthma:**

1. **Etiology**: A hyperactive airway to an intrinsic or extrinsic factor. Common causes include:

- ❍ Respiratory Infection
- ❍ Allergic Reaction to Inhaled Antigen
- ❍ Emotional Stress
- ❍ Exercise

- ❍ Idiosyncratic Reaction to NSAID or Beta Blocker
- ❍ Environment Toxin
- ❍ Mechanical Stimulation (coughing, laughing, cold air)
- ❍ Reflux Esophagitis

2. Pathophysiology

- ❍ A disease of inflammation that precipitates bronchospasm (0bstruction).
- ❍ Affects airways not alveoli and the bronchospasm is reversible
- ❍ Inflammation precipitates mucus production (more obstruction)
- ❍ Obstruction leads to air trapping and difficulty with expiration (harder, longer and less effective)
- ❍ Decreased Oxygenation and Carbon Dioxide Removal
- ❍ **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.
- ❍ Hyperinflation increases intrathoracic pressures which decreases venous return and increases RV afterload

3. Clinical Presentation

- ❍ Stimulation of Asthma Unrelieved by Typical Tx
- ❍ Increased Work of Breathing
- ❍ Rapid RR with Little Air Movement (Air Trapping)
- ❍ Long Expiratory Phase
- ❍ Expiratory Wheezes Initially → Minimal to No Air Movement on Inspiration or Expiration
- ❍ ABG: Hypoxia and Hypercapnia – Resp Acidosis
- ❍ PFT: Drop in FEV_1 & Peak Expiratory Flow
- ❍ Pulsus Paradoxus
- ❍ Restless and Anxious, Calming Down is a Bad Sign

4. Treatment Options

- ❍ Psychological Support
- ❍ Oxygen Therapy
- ❍ Maybe Mechanical Ventilation (big ETT), Low TVs
- ❍ Remove Irritate (if known)
- ❍ Hydration & Humidification

- ❍ Pharmacological
  - Bronchodilators
  - ABX
  - Corticosteroids
  - Inhaled Anticholinergic Agents
  - Sedatives & Muscle Relaxants
  - SubQ Epinephrine
- ❍ Monitor and Treat Pneumothorax
- ❍ Monitor and Treat Heart Failure

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

1. **Etiology**
   - ❍ Fat
   - ❍ Air
   - ❍ Amniotic Fluid
   - ❍ Thromboembolic (90% of all PEs) from DVT
     - Virchow's Triad
       - o Venous Stasis
         - ▪ Immobility
         - ▪ Dehydration
         - ▪ Paralysis
         - ▪ Obesity
       - o Hypercoagulability
         - ▪ A-Fib
         - ▪ Tumors/Cancer
         - ▪ Dehydration
         - ▪ Heart Failure (also immobility)
         - ▪ COPD Pt 2^0 A-Fib, Polycythemia
         - ▪ Previous PE
         - ▪ Pregnancy/Oral Contraception/Hormone Replacement Therapy
       - o Vascular Wall Damage
         - ▪ Trauma
         - ▪ Venous Catheters
         - ▪ Varicose Veins
         - ▪ Elevated LDL
         - ▪ Age

2. **Pathophysiology:** 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.
   - ❍ Pulmonary Artery Obstruction
   - ❍ V/Q Mismatching V > Q = Dead Space Initially
   - ❍ Non Perfused Alveoli will Collapse Secondary to Decreased Surfactant Production → Intrapulmonary Shunting
   - ❍ Circulation is Diverted to Open Vessels Engorging Them → Pulmonary Hypertension and Increased Hydrostatic Pressure → Capillary Leak → Intrapulmonary Shunting in well Perfused Areas
   - ❍ Pulmonary Infarction May Occur
   - ❍ Increased Pulmonary Vascular Resistance Increases Afterload on Right Ventricular → RV Failure and Potentially Infarction

3. Clinical Presentation
   - ❍ Sudden Onset Dyspnea & Pleuritic Chest Pain
   - ❍ Tachypnea
   - ❍ Refractory Hypoxemia
   - ❍ ABG: Hypoxemia with Respiratory Alkalosis
     Example: $Pa0_2$ 71, $PaC0_2$ 29, pH 7.59, $HC0_3$ 25, $Sa0_2$ 72
   - ❍ Fat Emboli: Petechiae on Thorax and Upper Extremities
   - ❍ Cardiac
     - Tachycardia
     - Cyanosis
     - Jugular Venous Distention
     - RV Failure
     - ECG: RV Hypertrophy, T wave Abnormalities
   - ❍ Diagnostic Tests
     - Chest X-Ray
     - V/Q Scan
     - Spiral CT
     - Pulmonary Angiogram
     - + D-Dimer
     - MRI
     - Lower Extremity Doppler Studies (not emergent)

4. **Treatment Options**
   - ❍ ABCs
   - ❍ Administer 100%
   - ❍ Intubated if Necessary
   - ❍ Consider Thrombolytics
   - ❍ Consider Embolectomy
   - ❍ IVC Filter Placement
   - ❍ Pain Management
   - ❍ Identify Causative Factor and Treat
   - ❍ Future Prevention

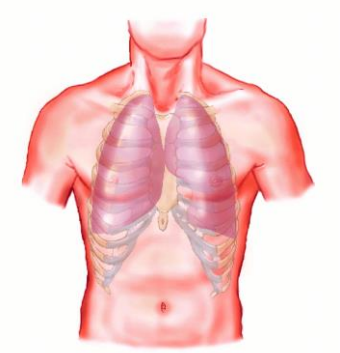
VIII. Chest Trauma

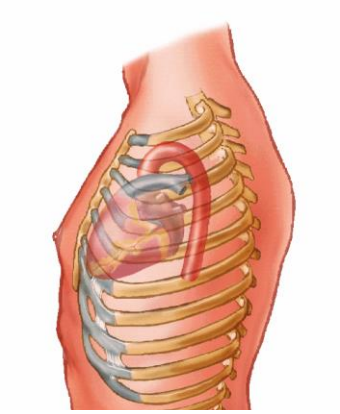
A. Mechanism of Injury

1. Blunt Trauma
   - ❍ MVC
   - ❍ Falls
   - ❍ Assaults
   - ❍ Pedestrians Struck
   - ❍ Recreational Sports
   - ❍ Explosives

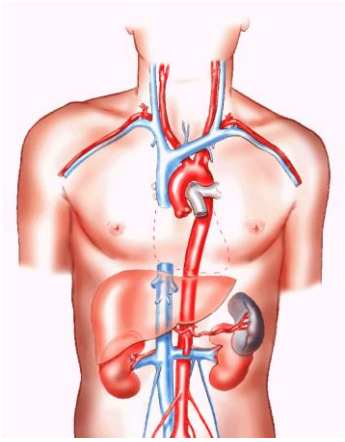
2. Penetrating Trauma: Pleural Cavity is Entered
   - ❍ Industrial Accidents: Construction & Farming
   - ❍ Projectiles
   - ❍ High-Energy: Ballistic Type/GSW
   - ❍ Low-Energy: Stabbings & Slashings

3. Index of Suspicion
   - ❍ Chest Wall
     - Rib Fractures
     - Soft Tissue
     - Pulmonary Contusions
     - Sternum Fracture
     - Flail Chest





- Pulmonary
  - Air Leak Syndromes
  - Rupture/Penetration to Trachea or Bronchus
  - Pulmonary Lacerations
- Cardiac
  - Cardiac Rupture
  - Cardiac Tamponade
  - Chamber Penetration
  - Cardiac Contusion
  - Pneumopericardium
- Vascular
  - Great Vessel Injury
  - Aortic Dissection
  - Chylothorax
- Other
  - Diaphragmatic Tear
  - Abdominal Injuries



B. Common Pulmonary Injuries

1. Pulmonary Contusion

- **Pathophysiology**: Bruising of the lung parenchyma. Fluid (blood & plasma) collects in the interstitial and alveolar spaces frequently decreasing compliance, causing shunting and hypoxemia. Contusion could lead to infection and ALI.

- **Clinical Presentation**
  - History of Blunt Chest Trauma
  - Pain with Inspiration
  - Hypoxemia (delayed)
  - Thick Bloody Sputum
  - Scattered Crackles & Maybe Wheezing
  - Chest X-ray: Contusion Present (delayed)
  - Chest CT: Parenchyma Hematoma/Contusion

- **Treatment Options**
  - Aggressive Pain Management
  - Oxygen Administration & Potentially Intubation
  - High Index of Suspicion of Other Injuries

- Aggressive Pulmonary Hygiene
  - Ambulation
  - Hydration & Humidification
  - Secretion Removal

4. Air Leak Syndromes

❍ **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
  - Tension Pneumothorax
  - Simple Pneumothorax
  - Hemothorax
  - Hemo/Pneumothorax
  - Pneumomediastium
  - 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
- Hypoxic on ABG

❍ **Treatment Options**

- Emergent Needle Decompression (tension)
- Chest Tube Placement (re-expand lungs & evacuate air/blood)
  - Insert High for Pneumo
  - Insert Low for Hemo
  - Suction Utilized
  - Potential for Air Leak

- Routine Milking or Stripping is NOT Recommended for Clot Prevention
- Oxygen Delivery & Potentially Intubation
- Sucking Chest Wound: Occlusive dressing on expiration. Monitor status carefully might need flutter valve
- Air Embolism: Trendelenberg position and Left Side to Trap Air in Heart (RV)
- Surgery May be Required

3. Rib Fractures/Flail Chest

❍ **Pathophysiology**

Free floating segment of rib cage from multiple rib fractures. Chest wall has paradoxical motion with inspiration and expiration causing decrease tidal volume (ventilation).

❍ **Clinical Presentation**

- History of Blunt Chest Trauma
- Paradoxical Chest Movements
- Pain
- Increased WOB
- Decreased TV
- Hypoxia
- Productive Cough

❍ **Treatment Options**

- Pain Relief
- Encourage Lung Expansion: IS, C&DB
- Humidification
- Possibly Intubate w Positive Pressure Ventilation
- Internal Fixation – very rare today
- High Index of Suspicion for Other Injuries

IX. Summary

Pulmonary ABG Appendix

The Physiology of Acid-Base Balance/Imbalance

Respiratory Acid Base Regulation: Ventilation (inspiration & expiration) and Diffusion (movement of gases) are responsible for $PaC0_2$ levels. Proper acid base balance can be maintained or disrupted by "blowing off" or "retaining" $C0_2$ by increasing or decreasing the respiratory rate and/or depth (minute ventilation Ve). Regulation or disruption in the balance can happen very quickly.

- Normal $PaC0_2$ 35 - 45mmHg
- Hypercapnia >45 mmHg Hypoventilation

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

- Hypocapnia < 35 mmHg Hyperventilation

  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 $HC0_3$ 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 $HC0_3$ 22 - 26 mEq/L
- Acidosis pH < 7.35, HCO_3 < 22 mEq/L & BE < -2

  Metabolic Acidosis Conditions That Increase $[H^+]$:
  DKA, renal failure, ingestion of acidic drugs, lactic acidosis

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

Metabolic Alkalosis Conditions That Increase Alkali: ingestion of antacids, admin of bicarbonate

Conditions that Decrease Acid: loss of gastric juices (vomiting, high NG output), diuretics

Evaluation Questions?

- What is $Pa0_2$?
- What is pH?
- What is $PaC0_2$?
- What is $HC0_3$?
- Options?

Options

Normal pH, Normal $PaC0_2$, Normal $HC0_3$ =

Acidosis with High $PaC0_2$, Normal $HC0_3$ =

Acidosis with Low $HC0_3$, Normal $PaC0_2$ =

Alkalosis with Low $PaC0_2$, Normal $HC0_3$ =

Alkalosis with High $HC0_3$, Normal $PaC0_2$ =

Determination of Compensation

Disorder	pH	Primary	Compensation
Resp Acidosis	⇓	⇑ $PaC0_2$	⇑ $HC0_3$
Resp Alkalosis	⇑	⇓ $PaC0_2$	⇓ $HC0_3$
Met Acidosis	⇓	⇓ $HC0_3$	⇓ $PaC0_2$
Met Alkalosis	⇑	⇑ $HC0_3$	⇑ $PaC0_2$

FIRST Evaluate Oxygenation

- Look at $Pa0_2$
- Is it Normal, High or Low?
- Low = Hypoxia - determine cause and possible treatment(s)

SECOND Evaluate ACID BASE Balance

- Look at pH
- Is it Normal, High or Low?
- < 7.35 = acidotic
- > 7.45 = alkalotic

THIRD Evaluate ACID BASE Balance

- Look at $PaC0_2$
- Is it Normal, High or Low?
- Look at $HC0_3$
- Is it Normal, High or Low

FOURTH Evaluate Findings

- Physiology/Biochemistry
- Compensation

FIFTH Treatment Options

Practice Examples

1. Mr. J is a 65yo male who is being admitted with a decreased LOC. His vital signs are: T – 39, HR 104, RR 10 and shallow and BP 96/60

ABG:

$Pa0_2$	95mmHg
$PaC0_2$	65mmHg
pH	7.25
$HC0_3$	25mEq/L

Step 1 Evaluate Oxygenation
Step 2 Evaluate pH
Step 3 Evaluate Acid Base: $C0_2$ and HCO_3
Step 4 Interpret? Compensation? Cause
Step 5 Treatment Options

2. Mr. J's wife is very upset. She is anxious and appears to be confused. The charge nurse takes her into another room and assesses her.
T – 37, HR 120, RR 32 and BP 156/84

ABG:

$Pa0_2$	100mmHg
$PaC0_2$	28mmHg
pH	7.60
$HC0_3$	23mEq/L

Step 1 Evaluate Oxygenation
Step 2 Evaluate pH
Step 3 Evaluate Acid Base: $C0_2$ and HCO_3
Step 4 Interpret? Compensation? Cause
Step 5 Treatment Options

3. Mrs. K is having a pre-op work up. Because she will be having general anesthesia an ABG is ordered. She is a ½ pack per day smoker

ABG:

$Pa0_2$	95mmHg
$PaC0_2$	45mmHg
pH	7.43
$HC0_3$	23mEq/L

Step 1 Evaluate Oxygenation
Step 2 Evaluate pH
Step 3 Evaluate Acid Base: $C0_2$ and HCO_3
Step 4 Interpret? Compensation? Cause
Step 5 Treatment Options

4. Suzie is a 15 yr old admitted for acute renal failure related to a severe kidney infection. T 37.9, HR 111, RR 29, BP 121/48

ABG:

$Pa0_2$	100mmHg
$PaC0_2$	31mmHg
pH	7.27
$HC0_3$	16mEq/L

Step 1 Evaluate Oxygenation
Step 2 Evaluate pH
Step 3 Evaluate Acid Base: $C0_2$ and HCO_3

Step 4 Interpret? Compensation? Cause
Step 5 Treatment Options

5. George is a 19 yr college student. He is admitted for gastroenteritis and dehydration from vomiting for 2 days.
T 38, HR 119, RR 20, BP 130/60

ABG:

$Pa0_2$	92mmHg
$PaC0_2$	37mmHg
pH	7.51
$HC0_3$	30mEq/L

Step 1 Evaluate Oxygenation
Step 2 Evaluate pH
Step 3 Evaluate Acid Base: $C0_2$ and HCO_3
Step 4 Interpret? Compensation? Cause
Step 5 Treatment Options

Answers:
1. Resp Acid 2. Resp Alk 3. Normal 4. Met Acid w Partial Resp Comp
5. Hypoxia w Met Alk