CCRN Review: The Pulmonary System

The pulmonary system exists for the purpose of gas exchange

Pulmonary: 18%

- Acute Lung Injury (ARDS)
- Acute Pulmonary Embolus
- Acute Respiratory Failure
- Acute Respiratory Infections
- Air-leak Syndromes (Pneumo)
- COPD, Status Asthmaticus, Chronic Bronchitis, Emphysema
- Pulmonary HTN
- Thoracic Surgery and Trauma

CNS Control of Respirations

- Peripheral chemoreceptors located in carotid body and aortic body
- Sensitive to changes in the PO2, with hypoxemia stimulating chemoreceptor discharge
- Minor role in sensing PCO2

Gas Exchange Process: 4 Steps

- Step 1 – Ventilation
- Step 2 – Diffusion
- Step 3 – Perfusion
- Step 4 – Diffusion

CNS Control of Respirations

- Respiratory generator
  - Located in the medulla
    • Medulla responds to changes in CO2 and pH
    • Respond, not directly to PCO2, but the pH of the ECF surrounding chemoreceptor
  - Input from other CNS regions
    • Pons
      • Normal, coordinated breathing
    • Cerebral Cortex
      • Exerts a conscious or voluntary control over ventilation

Step 1 – Ventilation

- Moving air between atmosphere & alveoli
- Measures
  - Minute ventilation
    • RR x TV
  - Indirect pCO2
Step 2 – Diffusion

- Gases are moved across the alveolar-capillary membrane to the pulmonary bed
- Occurs down a concentration gradient – no active metabolic work takes place
- Measures
  - ABGs
  - Pulse oximetry
  - A-a gradient – indicates if gas transfer is normal
  - P/F Ratio -

CO2 transport

- Carried in the blood in 3 forms
  - Physically dissolved (PaCO2)
  - Chemically combined with Hgb (carbaminohemoglobin)
  - As bicarbonate through a conversion reaction

Step 3 – Transport of Gases

- Transport of gases in the circulation with 97% bound to hemoglobin & 3% dissolved in plasma

- Measures
  - Oxyhemoglobin dissociation curve

Step 4 – Diffusion

- Process by which gases are moved between systemic capillary bed and body tissues
  - Hypoxemia – Low O2 in the blood stream
  - Hypoxia – Low O2 in tissue

Normal Lung Function

- Requires dry, patent alveoli, closely situated to appropriately perfused capillaries

- The normal pulmonary capillary endothelium is selectively permeable
  - serum protein remains intravascular
  - fluid crosses the membranes under the control of hydrostatic & osmotic forces
**Balance of Hydrostatic & Osmotic Forces**

- Allows small quantities of fluid into the interstitium
- Mechanisms to prevent alveolar edema
  - Retained intravascular protein
  - Interstitial lymphatics
  - Return large quantities of fluid to the circulation
  - Tight junctions between alveolar epithelial cells
  - Prevent leakage into the air spaces

**Oxyhemoglobin Dissociation Curve: Shifts**

- Shift to the Right
  - More O₂ is unloaded for a given PO₂
  - Increases O₂ delivery to the tissue
  - Caused by
    - Acidosis
    - PCO₂ increase
    - Increase in body temperature
- Shift to the Left
  - O₂ is not dissociated from Hgb until tissue and capillary O₂ very low
  - Decreases O₂ delivery to the tissue
  - Caused by
    - Alkalosis
    - PCO₂ decrease
    - Carbon monoxide poisoning

**Other General Principles**

- Transport of oxygen to body tissues mostly influenced by CO₂, Hgb concentration and O₂/Hgb binding and releasing factors.

**Answer**

- pH 7.18
- pCO₂ 80
- pO₂ 35
- HCO₃ 29

- A shift to the right!
- More O₂ is unloaded for a given PO₂
  - Increases O₂ delivery to the tissue
Questions thus far??????

Acute Respiratory Failure
• Impairment of oxygenation and / or ventilation
  – Pao2 < 55 mmHg or Sao2 < 88%
  – Paco2 > 50-55 mmHg with accompanying acidemia, or pH < 7.30

Diagnostic Studies
• ABG
  – Decreased PaO2 and / or
  – Hypercapnia
• Radiologic
  – Findings depend on primary disease

Management
• Noninvasive VS invasive ventilation
• Positioning
  – HOB elevated 30°
  • Maximize ventilation
  • Prevent aspiration
• Skin care
• Pain management / sedation
• Nutrition
• ATBs as appropriate

Signs & Symptoms
• Dyspnea
• Neuro:
  – Hypoxemia
  • Anxiety, irritability, restlessness, confusion
  – Hypercarbia
  • H/A, lethargy, confused, obtunded, coma
• Pulmonary
  – Flared nostrils, increased respiratory rate, use of accessory muscles, dyspnea, SOB
• Cardiovascular
  – Tachycardia, bounding pulses, dysrhythmias

Indications for Mechanical Ventilation
• Pneumonia
• COPD
• ARDS
• Pulmonary edema
• Lung trauma
• Asthma
• Near Drowning
• Multiple sclerosis
• Muscular dystrophy
• Myasthenia gravis
• Spinal cord injury
• General anesthesia
• Overdose
• Obesity
Goals of Mechanical Ventilation

- Provide adequate ventilation
  - direct measure is minute ventilation (6-8 liters per minute)
  - indirect measure is CO2 of arterial blood (35-45)
- Provide adequate oxygenation
  - measured by pO2 of arterial blood gas
  - if correlation has been established, can be inferred by peripheral oxygen saturation

Complications from O2 Therapy

- Oxygen toxicity
  - An oxygen concentration in excess of 50% for > 24 hours increases the potential for development of oxygen toxicity & lung damage
  - Can impair Alveolar type II cells
    - These cells produce surfactant!

Provide Adequate Ventilation

- Hypoventilation
  - High PaCO2
  - Respiratory acidosis
  - Inadequate minute ventilation
- How do we correct???
  - Assure adequate minute ventilation
  - TV x RR = minute ventilation
- Hyperventilation
  - Low PaCO2
  - Respiratory alkalosis
  - Excessive minute ventilation
- How do we correct???
  - Assure appropriate minute ventilation
  - TV x RR = Minute Ventilation

Provide Adequate Oxygenation

- Maximal alveolar ventilation increase oxygen exchange
- Deliver high level of oxygen
- Add PEEP to better inflate alveoli thus improving oxygen exchange

Other Considerations

- Nitrogen
  - Most plentiful gas (normally)
  - Promotes alveolar expansion
  - When completely displaced by 100% oxygen
    - Can result in atelectasis

PEEP

- Improves oxygenation by maintaining alveolar airflow during expiration
  - Airways have a tendency to collapse during expiration as a result of increasing pressure outside the airway
- Optimal levels are achieved by the lowest level of PEEP needed to raise the PaO2 without resulting in cardiovascular compromise (5-15 common)
ARDS

• A syndrome of acute respiratory failure characterized by non-cardiac pulmonary edema and manifested by refractory hypoxemia caused by intrapulmonary shunt

• Nearly always occurs suddenly

• Overall mortality of ARDS ranges from 25% to 58%

PaO2/FiO2 Ratio

• A ratio of the partial pressure of arterial oxygen to the fraction of inspired oxygen.

  • Normal is 300-500 mmHg

  • FiO2 is expressed as a decimal.

ARDS Definition

• A syndrome of acute respiratory failure characterized by non-cardiac pulmonary edema and manifested by refractory hypoxemia caused by intrapulmonary shunt

• ARDS refers to the severe end of the spectrum of ‘Acute Lung Injury’ (ALI).

Examples

• WNL - a patient has PaO2 of 90 (normal 80-100) on room air (21%)
  
  \[ \frac{90}{0.21} = 428 \text{ mmHg} \]

• ALI - a patient has a PaO2 of 90 on FiO2 of 40%
  
  \[ \frac{90}{0.40} = 225 \text{ mmHg} \]

What is Acute Lung Injury?

• Defined as syndrome of acute and persistent lung inflammation with increased vascular permeability.

• Characterized by 3 clinical features
  
  – Bilateral radiographic infiltrates
  
  – PaO2/FiO2 ratio between 201-300 mmHg.
  
  – No clinical evidence of an elevated left atrial pressure. If measured, PAOP in \( \leq 18 \) mmHg.

ARDS = ALI + Worse Hypoxia

• ARDS
  
  – Hypoxia is WORSE
  
  – PaO2/FiO2 \( \leq 200 \) mmHg

• Distinction between ALI and ARDS is somewhat arbitrary, since the degree of gas exchange disturbance does not correlate reliably with the extent of the underlying pathology.
Example #1 – a patient has a \( \text{PaO}_2 \) of 60 on \( \text{FiO}_2 \) of 80%
- \( \text{P/F ratio} = \frac{60}{0.80} = 75 \text{ mmHg} \)

Example #2 – a patient has a \( \text{PaO}_2 \) of 50 on \( \text{FiO}_2 \) of 100%
- \( \text{P/F ratio} = \frac{50}{1.0} = 50 \text{ mmHg} \)

Epidemiology
- Within ICUs, approximately 10-15% of admitted patients meet criteria for ARDS
- Up to 20% of mechanically ventilated patients meet criteria of ARDS!

Causes
- More than 60 causes of ARDS have been identified
- Additional causes continue to emerge as adverse pulmonary reactions to new therapies are discovered.

Common Causes
- Sepsis – most common cause
- Aspiration
- Infectious pneumonia
- Severe trauma
- Surface burns
- Multiple blood transfusions (>15)
- Leukoagglutinin reactions
- Pancreatitis
- Drug overdose
- Near drowning
- Smoke inhalation
- Cardiopulmonary bypass
- Pulmonary contusion
- Multiple fractures
- Following upper airway obstruction
- Drug reaction
- Venous air embolism
- Neurogenic pulmonary edema
- Acute eosinophilic pneumonia
- Bronchiolitis obliterans
- Organizing pneumonia (BOOP)
- Miliary TB
ARDS: Pathophysiology

• Acute phase - damage to integrity of the blood-gas barrier
  – Damage to type 1 alveolar epithelial cells
  – Increased endothelial permeability
  – Interstitial edema is found
  – Leakage of protein-containing fluid into the alveoli
  – Impaired production and fx of surfactant

ARDS: Pathophysiology

• Resultant physiologic abnormalities
  – Shunting of blood through atelectatic or fluid-filled lung units
  – Increased physiologic dead space
    • Frequently exceeding 60% of each breath
    • Compliance is reduced
    • Increased resistance to blood flow

Results In:

• Impaired Gas Exchange
• Decrease lung compliance
• Pulmonary hypertension
Decreased Lung Compliance

- Hallmark of ARDS
- Low compliance due to the stiffness of poorly or non-aerated lung.

Signs & Symptoms

- Severe dyspnea
- Increased work of breathing
- Refractory hypoxemia
- Diminished LOC if hypoxemia is severe
- Radiographic diffuse bilateral infiltrates
  - interstitial and alveolar
  - without cardiomegaly
- PAOP – normal or low

Any questions????

Treatment

- The mainstay of therapy for ARDS is:
  - Management of the underlying disorder causing it.
    - ID treatable sources
  - Main treatment is supportive.

Pulmonary Hypertension

- Occurs in about 25% of pts with ARDS subjected to mechanical ventilation.
- Contributing factors
  - hypoxic vasoconstriction
  - vascular compression (by + pressure vent)

Pharmacological Treatment

- Antibiotics
- Steroids (stress dose, watch glucose for osmotic changes)
- Diuretics
- Avoid excessive fluid administration
**Mechanical Ventilation**

- Tidal Volume
  - Initial 6 ml/kg
  - Permissive hypercapnea
- FiO₂ 100% (maintain SaO₂, 92-94%)
- PEEP
  - 5-10 cm H₂O is effective in reducing intrapulmonary shunting and improving oxygenation
  - Frequently see PEEP > 12

**Etiology**

- Organisms
  - *Streptococcus pneumoniae*
    - Most common cause
  - *Mycoplasma pneumoniae*
  - *Haemophilus influenzae*
- Viruses
  - Relatively uncommon, accounting for 25-50% of nonbacterial pneumonias
  - Influenza A – most common

**Mechanical Ventilation**

- Sedation
  - May require Neuromuscular Blockade
- May use Pressure Controlled Ventilation
  - Sets pressure limits, allowing TV to fluctuate and prevents alveolar over distension

**Signs & Symptoms**

- Symptoms
  - Dyspnea
  - Chest pain
  - Wheezing cough
  - Fever, chills, rigor, weight loss
  - Night sweats
  - Fatigue
  - Weakness
  - Decreased energy
  - Anorexia

- Signs
  - Consolidation
  - Pleural effusion
  - Airway involvement (wheezing)
  - Crackles, rhonchi
  - Tachypnea
  - Tachycardia
  - Fever
  - Purulent sputum
  - Dry cough
  - H/A, fatigue, sore throat
  - N/V, diarrhea

**Pneumonia**

- Inflammation of lung parenchyma often characterized by consolidation
- Exudate, inflammatory cells, fibrosis
- Usually caused by infectious agents or microbes,
  - Can be caused by aspiration of gastric contents

**Diagnosis**

- Sputum gram stain
  - Determine organisms and coverage
- Sputum cultures / sensitivities
- CBC with Diff
- Blood cultures
- Chest x-ray
  - Generally shows localized infiltrates
- Bronch / invasive diagnostics
Treatment

- Antibiotics
- Fluids
- Oxygen
- Mechanical Ventilation

Status Asthmaticus

- Characteristics
  - Unrelenting acute asthma
  - Broncho-constriction despite treatment
  - Predominantly high deadspace region
  - Increased work of breathing
    - Bronchospasm, inflammation, mucous production / plugs
  - Increased minute ventilation

Asthma

- Chronic disease
- Characterized by airway hyper-reactivity
- Produces airway narrowing of a reversible nature

Pathophysiology

- Airway narrowing from
  - Bronchial smooth muscle spasm
  - Inflammation of bronchial walls, leading to increased permeability and thickening
  - Mucous plugging from increased production and decreased clearance of secretions

Pathophysiology

- Increased responsiveness to stimuli
- Widespread narrowing of airway
- Cellular infiltration and mucosal edema
- Airway hyperreactivity
  - Smooth muscle contraction
  - Excessive mucus production
  - Diminished secretion clearance
- V/Q abnormalities
- Increased work of breathing & airway resistance
- Hyperinflation of the lung, increased residual volume
- Host defect of altered immunologic state

Precipitating Events

- Infection, sinusitis
- Smoking
- Recent exposure (pollens, dust mites, animals, beta blockers)
- Emotional factors
- Gastroesophageal reflux
- Exercise
**Impending Status Asthmaticus**

- Recurrent episodes over a short period (2-7 days)
- Change in pattern of symptoms
  - Wheezing more severe or frequent
- Worsening dyspnea
  - Exercise limitation, at rest
- Cough with tenacious sputum
- Irritability

**Treatment**

- Bronchodilators
- Corticosteroids
- Oxygen
- Hydration
- Antibiotics if infection suspected
- Mechanical ventilation
  - Low tidal volumes
  - May need sedatives

**Acute Pulmonary Embolus**

- Extremely dyspneic
- Inspiratory and expiratory wheezing usually audible
- Prolonged expiratory phase
  - Pt tries to exhale trapped air through narrow airways
- Tachypnea
- Tachycardia
- Use of accessory muscles
- Flaring nares, pallor, cyanosis, increased work of breathing, and fatigue

- Disappearance of wheezing may be ominous sign
  - Airways completely obstructed

**Other predisposing factors**

- Age > 40
- Immobility
- Previous DVT
- Anesthesia / surgery
- Pregnancy / post-partum
- Trauma
Hemodynamic Consequences

- Obstruction stimulates neurohumoral stimuli
  - Increases PA pressures & PVR
  - Results in increased RV work
- Pulmonary HTN (mean PAP > 20 mmHg)
- RV will fail if mean PA pressure > 40 mmHg

Disruption in blood flow

- Alveoli become nonfunctioning units
  - Don’t participate in gas exchange
  - Increases deadspace
- To maintain gas exchange
  - Ventilation is shifted to the noninvolved areas of the lung
  - Results in constriction of distal airways
  - Leads to alveolar collapse and atelectasis

Signs & Symptoms

- Depends on severity
  - Sudden onset of chest pain
  - Cough
  - Hemoptysis
- Massive PE
  - >50% vascular occlusion
    - Mental clouding
    - Anxiety
    - Feeling of impending doom
    - Apprehension
- Dyspnea, tachypnea, increased work of breathing, tachycardia, reduced blood pressure, restlessness, syncope, asymmetric chest expansion

Goals of Care

- Restore pulmonary artery blood flow
- Maintain / Restore hemodynamic stability
- Relieve chest pain

PE Diagnostics

- ABG
  - May indicate respiratory alkalosis
- CXR
  - Nonspecific, frequently normal
- V/Q scan – not definitive, but suggestive
- Search for DVT – anticoagulation
- Pulmonary angiogram – most definitive test
- CT-angio may also be done
- ECG – usually normal, except in massive PE (new RBBB)

Pneumothorax

- Characteristics
  - Air in the pleural space
  - Leading causes
    - + pressure ventilation
    - Diagnostic procedure
  - Tension pneumo present when intrapleural pressure exceeds atmospheric pressure throughout expiration
  - Severity depends on size, underlying lung dz, whether a tension pneumo is present
Pneumothorax

- Symptoms
  - Dyspnea
  - Chest pain
    * Usually pleuritic
    * Typically acute onset

- Signs
  - Tachypnea
  - Tachycardia
  - Hypotension
  - Decreased respiratory excursion
  - Widened intercostal spaces
  - Absent or reduced breath sounds
  - Hyperresonant to percussion
  - Tracheal shift to contralateral side
  - Hypoaoxia +/- hypercapnia on ABG

Chest Trauma

- Blunt injuries
- Etiology & Risk factors
  - Auto accidents
  - Falls
  - Assaults
  - Explosions

- Penetrating injuries
- Etiology & Risk factors
  - All those causing blunt injuries &
  - Bullets
  - Knives
  - Shell fragments
  - Free-flying objects
  - Industrial accidents

Therapy

- Re-expansion of the collapsed lung
  - Chest tube insertion
- Adequate oxygenation
- Maintain cardiac output
- Reduce / control pain
- Observation

Signs & Symptoms

- Varies with specific injury
  - Tachypnea, dyspnea, pain, respiratory distress may occur with any injury

Chest Trauma

- Pathophysiology depends on type and extent of injury

- Trauma to the chest or lungs may interfere with any of the components involved with inspiration, gas exchange and expiration

Blunt injuries

- Visceral injuries without chest wall damage
  - Pneumothorax, hemothorax
  - Lung contusion
  - Diaphragmatic injury
  - Myocardial contusion, aortic rupture
  - Rupture of the trachea or bronchus
Blunt injuries

- Soft tissue injuries
  - Possibly a sign of severe underlying damage
    - Cutaneous abrasion
    - Ecchymosis
    - Laceration of superficial layers
    - Burns
    - Hematoma

- Others
  - Fracture of the sternum
    - Occurs either as a result of direct impact or as the indirect result of overflexion of the trunk
  - Rib fractures
    - As a result of overflexion or straightening.
    - Can be unifocal or multifocal
    - Multiple fractures result in flail chest
      - Often complicated by injuries to soft tissues and pleura
  - S/SX
    - Pain accentuated by chest wall movement, deep inspiration or touch
    - Flair chest – dyspnea and localized pain

Penetrating injuries

- Open sucking chest wounds
  - If opening is < diameter of trachea, minimal symptoms
  - If opening is > more air enters the pleural space, collapses the lung, impairs ventilation and gas exchange, results in dyspnea

Blunt injuries

- Others
  - Fracture of the sternum
    - Occurs either as a result of direct impact or as the indirect result of overflexion of the trunk
  - Rib fractures
    - As a result of overflexion or straightening.
    - Can be unifocal or multifocal
    - Multiple fractures result in flail chest
      - Often complicated by injuries to soft tissues and pleura
  - S/SX
    - Pain accentuated by chest wall movement, deep inspiration or touch
    - Flair chest – dyspnea and localized pain

Penetrating injuries

- Hemothorax, hemopneumothorax
- Combined thoraco-abdominal injuries
  - Bowel sounds may be heard in chest
  - Trachea / large airway damage
    - Sub-Q emphysema
  - Wounds of heart / great vessels
    - Dyspnea and backache, intense pain in chest or back unaffected by respiration

Penetrating injuries

- Pleural cavity & chest wall entered
- Damage to deeper structures – more serious
- Extent of injury and organs injured predicted by course of wound and nature of penetrating instrument
- High velocity projectiles do more damage

Diagnostic Studies

- CXR
- MRI or CT if stable enough
- Aortography – confirms dx of rupture of aorta or other great vessels
- Bronchoscopy – dx rupture of trachea or bronchus
- ECG
Goals of Care

- Patent airway
- ABG levels and pulmonary parameters restored and maintained
- Chest wall integrity and stability restored
- Establish integrity of pleural space
- Minimize chest pain and dyspnea

- Questions.........................