Session Number 305

CERTIFICATION REVIEW: Pulmonary Part 1

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Content Description

This session discusses the physiology of the pulmonary system, including oxygen delivery and consumption, the oxyhemoglobin dissociation curve, and SvO₂ monitoring and interpretation. It will also discuss acid-base imbalances and interpretation of arterial blood gases. Application of these concepts will be applied to patients with COPD, asthma, pneumonia, pulmonary embolus and pneumothorax. Causes, presentation, diagnosis, and collaborative management of these disease processes will be reviewed. Emphasis will be on possible questions that may be asked on these subjects in the CCRN, PCCN and CMC examinations. There will be time allotted for sample questions.

Learning Objectives
At the end of this session, the participant will be able to:

1. Describe mechanisms involved in pulmonary ventilation and perfusion.
2. Analyze a sample arterial blood gas for oxygenation, ventilation and acid-base balance.
3. Describe the cause, presentation, diagnosis and collaborative management of COPD, asthma, pneumonia, pulmonary embolus and pneumothorax

REFERENCES

NOTE: Please refer to outline for references pertaining to this session
Certification Review: Pulmonary Part 1

Approximately 18% of the CCRN exam, 14% of the PCCN exam, and 7% of the CMC exam will focus on the pulmonary system.

<table>
<thead>
<tr>
<th>CCRN, PCCN, and CMC</th>
<th>CCRN and PCCN</th>
<th>CCRN only</th>
<th>PCCN only</th>
<th>CMC only</th>
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<tbody>
<tr>
<td>Acute lung injury (ARDS, ALI, RDS)</td>
<td>Pulmonary hypertension</td>
<td>Thoracic surgery</td>
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<td>Acute respiratory failure/Chronic ventilatory failure</td>
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<td>Thoracic trauma</td>
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<td>☒ Acute respiratory infections</td>
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Note for PCCN candidates: This presentation includes discussions of SvO₂ interpretation and references to pulmonary artery catheter measurements. These topics will not be tested in the PCCN exam.

I. Review of Pulmonary Anatomy and Physiology

A. Gas exchange
   1. Alveolar-capillary membrane
   2. Diffusion pathway
   3. Type I pneumocytes
   4. Type II pneumocytes

B. Oxygen
   1. 97% of oxygen combined with hemoglobin
      More affinity of hemoglobin for oxygen at lung level
      Less affinity of hemoglobin for oxygen at tissue level
   2. Oxyhemoglobin dissociation curve
      Shows relationship between PaO₂ and hemoglobin saturation
      PaO₂ of 60 mmHg critical point
      Minimal increases in O₂ saturation above 60 mmHg
      Dramatic decreases in O₂ saturation below 60 mmHg
      Shifts in curve
      Shift to left-alkalemia, hypothermia, hypocapnia
      Hemoglobin more saturated, but less is unloaded
      Shift to right- acidemia, hyperthermia, hypercapnia
      Hemoglobin is less saturated, but more is unloaded
   3. Oxygen delivery to tissues - Do₂
Volume of oxygen delivered to the tissues by the left ventricle each minute
Dependent on:
Cardiac Output - Stroke volume and heart rate
Arterial oxygen content (CaO₂) - Hgb and oxygen saturation (SaO₂)

4. Oxygen consumption - VO₂
   Volume of oxygen consumed by the tissues each minute
   Measured by comparison of CaO₂ and CVO₂ (venous oxygen content)

5. Oxygen reserve in venous blood
   Determined by mixed venous oxygen saturation (SvO₂) - 75%

6. Critical oxygen delivery point
   Oxygen delivery and consumption are independent; when exceeded, consumption is dependent on O₂ delivery
   SvO₂ improves with increased Do₂ = independent
   SvO₂ does not improve with increased Do₂ = dependent

7. SvO₂ monitoring and interpretation
   Obtained with continuous monitoring by specialized PA catheter
   Measures balance between arterial oxygen supply and oxygen demand at tissue level
   Factors contributing to SvO₂: CO, Hbg, SaO₂, VO₂
   Normal 60%-80%
   Determining cause of decreased SvO₂
   CO: Changes in preload, afterload, heart rate, contractility will affect CO and subsequently, SvO₂
   Hypovolemia, heart failure, cardiogenic shock, hypertension, VT
   Hgb: Decreased Hgb from bleeding, anemia, or destruction of RBCs
   SaO₂: Anything that reduces oxygen supply
   ARDS, restrictive lung disease, PE, suctioning, position change, hypoxia
   VO₂: Increased VO₂ will decrease SvO₂ in critically ill patient
   Increased metabolic needs, routine nursing care, sepsis, MODS, burns, head injury, shivering, fever, seizure, pain, increased WOB
   Determining cause of increased SvO₂
   SaO₂: FiO₂ greater than tissues require
   VO₂: Low oxygen demand from anesthesia, neuromuscular blockade, hypothermia
   VO₂: Sepsis due to tissues unable to extract oxygen
   Other: Wedged catheter

II. Acid-Base Balance and Arterial Blood Gas Interpretation
   A. Regulated by several buffering systems; most effective is bicarbonate buffer system
   Carbon dioxide and water combine to form carbonic acid, a weak acid, which can combine to form bicarbonate, a strong base
\[ \text{CO}_2 + \text{H}_2\text{O} \rightleftharpoons \text{H}_2\text{CO}_3^- \rightleftharpoons \text{H}^+ + \text{HCO}_3^- \]

Lungs \hspace{2cm} Kidneys

Respiratory system regulates carbonic acid: carbon dioxide and water
Kidneys regulate bicarbonate: excrete or retain depending on pH

B. Analysis of Arterial Blood Gas
- Evaluates ventilation - \text{PCO}_2
- Evaluates acid-base - \text{pH}, \text{PCO}_2, \text{HCO}_3^-
- Evaluates oxygenation - \text{PaO}_2, \text{SaO}_2

C. Acid-Base Interpretation: The Tic-Tac-Toe method

<table>
<thead>
<tr>
<th>Acid</th>
<th>Normal</th>
<th>Base</th>
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<tr>
<td>\text{pH}</td>
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<tr>
<td>\text{PCO}_2 \text{ (mmHg)}</td>
<td>35-45</td>
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</tr>
<tr>
<td>\text{HCO}_3^- \text{ (mEq/L)}</td>
<td>22-27</td>
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Is the other value normal or opposite \text{pH}? If normal, there is no compensation; if on opposite side of \text{pH}, there is compensation. If all values are on the same side, it is a mixed disorder, and the body can't compensate.

RESPIRATORY ALKALOSIS

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<th>Normal</th>
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<td>&gt;7.45</td>
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<tr>
<td>\text{PCO}_2 \text{ (mmHg)}</td>
<td>&lt;35</td>
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</tr>
<tr>
<td>\text{HCO}_3^- \text{ (mEq/L)}</td>
<td>22-27</td>
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Causes: anxiety, CNS injury or infection; pulmonary embolus (early), pneumonia (early), fever, hyperthyroidism
S/S: tachycardia, anxiety, paresthesias, muscle irritability
Compensation: \text{HCO}_3^- < 22

RESPIRATORY ACIDOSIS

<table>
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<tr>
<td>\text{HCO}_3^- \text{ (mEq/L)}</td>
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Causes: COPD, pneumonia, pulmonary embolus (late), pneumonia (late), airway obstruction, CNS depression, neuromuscular disorders
S/S: tachycardia, dysrhythmia, tachypnea, diaphoresis; later, confusion, somnolence, coma
Compensation: \text{HCO}_3^- > 27
Causes: vomiting, NG suction, diuretics  
S/S: N/V/D, paresthesias, confusion, dizziness  
Compensation: PCO₂ > 45

Causes: Acid gain: DKA, renal failure, shock.  
Base lose: pancreatic fistula, diarrhea.  
Use anion gap to determine if acidosis is due to metabolic acid gain or base loss.  
Formula: (Na⁺ + K⁺) - (CO₂). Normal: 5 - 15. If normal, metabolic acidosis is caused by base loss.  
If >15, it is caused by metabolic acid gain.

S/S: N/V, abdominal discomfort, headache, tremor, hypotension.  
Compensation: PCO₂ < 35

Step 5  
Evaluate oxygenation  

<table>
<thead>
<tr>
<th>PaO₂</th>
<th>SaO₂</th>
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<tr>
<td>≥ 80 mmHg = normal</td>
<td>≥ 94% = normal</td>
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<tr>
<td>&lt; 80 mmHg = mild hypoxemia</td>
<td>&lt; 94% = mild desaturation</td>
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<tr>
<td>&lt; 60 mmHg = moderate hypoxemia</td>
<td>&lt; 90% = moderate desaturation</td>
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<tr>
<td>&lt; 40 mmHg = severe hypoxemia</td>
<td>&lt; 75% = severe desaturation</td>
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III. Chronic Obstructive Pulmonary Disease (COPD)

A. Progressive and often irreversible airflow limitations, associated with an abnormal inflammatory response to noxious particles of gases
   - Chronic inflammation
   - Airway remodeling
   - Destruction of alveolar wall and connective tissue
   - Enlarged submucosal glands and goblet cells
   - Destruction of cilia

B. Causes
   - Smoking
   - Air pollution
   - Occupational exposure to dust or chemicals
   - Genetic
     - $\alpha$-antitrypsin deficiency

C. Treatment of Stable COPD
   - Avoid exposure to irritants
   - Stop smoking
   - Avoid outdoors when pollutants are high
   - Vaccination
   - Bronchodilator and corticosteroid inhalers
   - Oxygen if end stage

D. Acute Exacerbation of COPD
   - Triggers
     - Infection
     - Cardiac disease
   - Presentation
     - Worsening dyspnea

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<thead>
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<td>pCO₂ 82</td>
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<td>HCO₃⁻ 33</td>
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<td>pO₂ 98</td>
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Changes in amount and character of sputum
Weakness, chest tightness, fever, anxiety
Wheezing
Retractions
Pursed lip breathing and upright position
Tachycardia and hypotension

Diagnosis
Presentation and history
Chest X-Ray
ABG: Respiratory acidosis and hypoxemia

Collaborative Management
Adequate oxygenation
Goal is PaO2 above 60 mmHg or SaO2 above 90%
Higher amounts do not significantly improve
Oxyhemoglobin dissociation curve

Bronchodilators
Corticosteroids
Antibiotics when indicated

IV. Asthma
A. Description
Chronic inflammatory disorder of airways
Airways hyperresponsive when allergens, viruses other irritants are inhaled
Obstruction due to bronchoconstriction, airway edema, mucus plugging and
airway remodeling
Causes air trapping, prolonged exhalation, V/Q mismatching
Differs from COPD due to asthma usually reversible

B. Triggers
Inhalant allergens
Animals, dust mites, cockroaches, indoor fungi, outdoor allergens
Occupational exposure
Irritants
Tobacco smoke, indoor and outdoor pollution
Fumes from perfume, cleaning agents, sprays

C. Factors influencing asthma severity
Viral respiratory infections
Rhinitis and sinusitis
Gastroesophageal reflux
Aspirin and NSAID sensitivity

D. Presentation
Wheezing, dyspnea, chest tightness, cough
Hyperventilation
ABG: respiratory alkalosis with hypoxemia
Tachypnea, tachycardia
Anxiety

E. Asthma exacerbation
Mild
Managed at home with short-acting beta2 agonists

Moderate to severe
Oxygen
Steroids
Inhaled bronchodilator therapy

F. Status asthmaticus
Presentation
Breathlessness at rest and need to sit upright
Speaking in single word; unable to peak in sentences or phrases
Lethargy or confusion
Paradoxical chest wall movement
Absence of wheezing (silent chest)
Respiratory acidosis with hypoxemia
pulsus paradoxus > 25 mm Hg
Peak flow < 50% of baseline

At risk for death from asthma
Previous severe exacerbation
Two or more hospitalizations for asthma in the past year
Three or more ED visits for asthma in past year
Hospitalization or ED visit for asthma in the past month
Cardiovascular disease
Chronic lung disease

Treatment
Oxygen
Nebulizer
Beta-agonist every 20-30 minutes times 3
May need to administer continuously
Add anticholinergic if no improvement
Corticosteroids
PO as effective as IV
May be too dyspneic to take orally
Continuous pulse oximetry
ABG if need to assess CO2
Normal or elevated indicates imminent respiratory failure
If no improvement after one hour:
IV magnesium sulfate
Heliox
Lighter than air
Delivers oxygen and nebulized β-agonist deeper into lungs
May require intubation and mechanical ventilation

V. Pneumonia and aspiration
A. Types of pneumonia
Community acquired (CAP)
At risk: alcoholism, impaired swallowing, altered MS, COPD:
comorbidities: diabetes, malignancy, CAD
Hospital acquired or nosocomial (HAP)  
Pneumonia occurring more than 48 hours after hospital admissions  
Aspiration of bacteria colonizing the oropharynx or GI tract  
At risk: Elderly, altered LOC, COPD, shock, mechanical ventilation, nasogastric tube and enteral feedings, supine position, poor hand hygiene

B. Assessment findings  
Dyspnea  
Fever  
Harsh crackles one area of lung  
ABGs:  
Early: respiratory alkalosis with hypoxemia  
Late: respiratory acidosis  
CXR: Consolidation one area of lung

C. Interventions for CAP and HAP  
Prevention is key  
Consideration of risks  
Universal precautions  
Education  
Adequate oxygenation  
Fluids unless contraindicated  
Antibiotic therapy after cultures obtained

D. Aspiration pneumonia (pneumonitis)  
Description  
Presence of abnormal substance in the airways  
May be chemical, mechanical, and/or bacterial

Risk factors  
Altered LOC  
Depressed gag, cough, or swallowing reflexes  
Feeding tube  
Artificial airway  
Ileus  
Gastric distention  
GI disorders  
Dysphagia  
Gastroesophageal  
Reflux disease

Interventions  
Suction upper airway to remove gastric contents  
Bronchoscopy to remove large particles or confirm  
Maintain adequate oxygenation  
Antibiotic therapy only if symptomatic after 48 hours

VI. Acute pulmonary embolism  
A. Description
Acute pulmonary embolisms caused by the movement of a clot from its site of origin through the right side of the heart, where it lodges in a branch of the pulmonary circulation.

Nearly 95% of all pulmonary emboli arise from thrombi in the deep veins of the legs (DVT).

Swelling, pain, + Homan’s sign, warmth are indications of DVT.

Patients at risk: Virchow’s Triad: stasis, hypercoagulability, vessel wall damage.

B. Clinical manifestations of PE

Sudden dyspnea, tachypnea, tachycardia and restlessness in patient with diagnosed DVT or known risk factors.

Pleuritic pain, pleural friction rub, hemothysis.

S₃, crackles.

ABG: initially, respiratory alkalosis; increased A-a gradient.

Hemodynamics: PAOP normal; PAD and CVP elevated.

Profound tachycardia, hypotension, cyanosis, stupor and syncope indications of massive PE.

C. Diagnostics

Pulmonary angiography definitive test.

V/Q scan indicative of perfusion defect may suggest PE; less invasive.

Ventilation and perfusion:

Ventilation - movement of air between atmosphere and alveoli and distribution of air within the lungs to maintain appropriate concentrations of oxygen and carbon dioxide in the alveoli.

Perfusion - movement of blood through the pulmonary capillaries.

Capillary pressure higher in bases than apices due to gravity.

Ventilation/perfusion ratio (V/Q):

Normal ratio 0.8.

Dead space - ventilation greater than perfusion (V>Q); V/Q > 0.8.

Shunt - Perfusion greater than ventilation (Q>V); V/Q < 0.8.

D. Management

Oxygen therapy, possibly mechanical ventilation, to keep PaO₂ > 60 mmHg.

Anticoagulation therapy.

Thrombolytic therapy.

Bedrest.

Monitor for complications: pulmonary infarction, pneumonia, ARDS, MI, dysrhythmia, shock.

Recurrent PE: placement of filter in inferior vena cava.

VII. Pneumothorax

A. Description

Defect in visceral pleura that allows air (or fluid) to enter pleural space and lung to collapse.

Simple - defect does not enlarge.

Tension - air enters on inspiration; cannot exit on expiration, defect enlarges.

Hemothorax - blood trapped in pleural space.
B. Clinical manifestations
   Acute onset: dyspnea and/or chest pain
   Respiratory distress
   Decreased breath sounds on affected side
   Crepitus
   Hypotension

C. Management
   Emergency needle thoracentesis
   Placement of chest tube
      Types
         Pleural - to remove free air - placed at 2nd ICS at MCL
         - to remove fluids (blood, pus) - placed at 5th or 6th ICS at midaxillary line
         Mediastinal - placed after cardiac surgery to drain air and blood

   Drainage collection system
      Usually three bottle system
      Drainage collection bottle allows blood, fluid, air to drain from pleural space
      Water seal bottle allows air to escape; prevents atmospheric air to go into pleural space
      Suction control bottle: Water to prescribed level; may be connected to wall suction. Actual amount of suction depends on amount of water in bottle. If connected to wall suction, should only mildly bubble.

Collaborative Management
   Maintain system
      Monitor chest tube drainage, document on I&O; tape all connections; prevent kinks and dependent loops in tubing; maintain suction at prescribed level; milking and stripping controversial
      Clamping -only done under specific conditions, then only briefly: changing collection bottle, locating air leak, need to elevate higher than chest. Also done in preparation for removal.

   Adequate oxygenation: respiratory assessment; position with unaffected lung down; deep breathing; incentive spirometry; daily CXR; oxygen therapy. Encourage cough and deep breathing to facilitate lung expansion
   Pain control

Certification Questions

1. A patient is admitted with a COPD exacerbation and worsening dyspnea. His admission vital signs are 38.1°C, HR 120 bpm, BP 180/80 mmHg, SaO2 90% on 2L/NC, RR 35 and slightly labored. Initial ABGs are:
   pH 7.33, PaCO2 57 mmHg, PaO2 61 mmHg, SaO2 87%, HCO3  35 mEq/L.
   Which intervention should the nurse anticipate based on the assessment data?
A. Intubation related to the hypercarbia
B. Aggressive diuresis
C. Placing the patient on room air
D. Increasing the O2 to 4L/NC

2. All of the following signs and symptoms were identified in a newly admitted patient with a history of severe asthma. Of these clinical findings, which poses the most significant concern to the nurse who suspects this patient may develop acute respiratory failure?
   A. Inability to readily speak a three-word sentence
   B. Wheezing audible without a stethoscope
   C. Respiratory rate of 38/min
   D. Inaudible breath sounds by auscultation

3. A 60-year-old patient is transferred to the PCU with a new onset of fever, leukocytosis, and a cough productive of large amounts of rust-colored sputum. His vital signs are as follows: temperature 39.5°C, heart rate 120/min, respiratory rate 35/min, SaO2 90% on room air, BP 90/40 mm Hg. The progressive care nurse knows that the highest priority interventions for this patient are to:
   A. Obtain sputum and blood cultures and start broad-spectrum antibiotics
   B. Facilitate obtaining both a chest x-ray and a sputum culture
   C. Administer acetaminophen and obtain a full set of cultures
   D. Initiate administration of oxygen and IV fluids

4. A left-sided tension pneumothorax is best described as an accumulation of air in the:
   A. Left pleural space, resulting in collapse of the left lung
   B. Right pleural space, resulting in collapse of the left lung
   C. Left pleural space, resulting in collapse of the left lung and compression of the right thoracic structure
   D. Left pleural space, resulting in collapse of the right lung and compression of the right thoracic structure

5. Dyspnea with a normal PAOP, an increase in PAD and pulmonary vascular resistance, and an increase in RAP would most likely indicate:
   A. Cardiac tamponade
   B. Left ventricular failure
   C. Myocardial infarction
   D. Pulmonary embolism
References


