Content Description

This session discusses the pathophysiology of respiratory failure, its presentation and treatment. It will discuss mechanical ventilation for the patient with acute respiratory failure. It also discusses Acute Respiratory Distress Syndrome (ARDS) and Acute Lung Injury (ALI), including pathophysiology, nursing care, and collaborative management. Finally, it will present pulmonary hypertension and cor pulmonale, thoracic surgery, pulmonary trauma and sleep disordered breathing. 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 the pathophysiologic process and treatment of the patient with respiratory failure requiring mechanical ventilation

2. Describe the causes, presentation, diagnosis and collaborative management of Adult Respiratory Distress Syndrome

3. Discuss the pathophysiology, presentation, diagnosis, and collaborative treatment of pulmonary hypertension and cor pulmonale, thoracic surgery, pulmonary trauma and sleep disordered breathing

REFERENCES

NOTE: Please refer to outline for references pertaining to this session
Certification Review: Pulmonary Part 2

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>
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<td>□ Acute respiratory failure/Chronic ventilatory failure</td>
<td>□ Thoracic surgery</td>
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<td>Acute pulmonary embolus</td>
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<td>Acute respiratory infections</td>
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<td>Pneumothorax</td>
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<td><strong>CCRN and PCCN</strong></td>
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<td>Exacerbation of COPD</td>
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<td>Severe asthma/Status asthmaticus</td>
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<td>Aspiration</td>
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Note for PCCN candidates: This presentation includes discussions of mechanical ventilation and references to pulmonary artery catheter measurements, and vasoactive and neuromuscular blockade medications. These topics will not be tested in the PCCN exam.

I. Acute respiratory failure
   A. Definition
      Inability of the lungs to maintain adequate gas exchange
   B. Types of respiratory failure
      Hypoxemic (Type I) respiratory failure
         PaO2 < 50 mmHg; PaCO2 normal or low
         Due to pneumonia, pulmonary edema, atelectasis, ARDS, chest injury, PE
      Hypercapnic (Type II) respiratory failure
         PaCO2 > 50 mmHg; pH < 7.30; PaO2 may be normal or low
         Due to chronic bronchitis, emphysema, opioid OD, spinal cord injury
   C. Etiologies
      1. Impaired ventilation – disruption of muscles of respiration or neurologic control
         a. SCI above C4
         b. Neuromuscular diseases
         c. CSN depression - drug OD, increased ICP
         d. Respiratory muscle fatigue
      2. Impaired gas exchange – damage to alveolar-capillary membrane
         a. Pulmonary edema
         b. ARDS
         c. Aspiration pneumonia
      3. Airway obstruction – blockage of airway lumen, excessive secretions, bronchoconstriction
         a. Foreign body aspiration
b. Asthma  
c. Bronchitis  
d. Pneumonia  

4. Ventilation-perfusion abnormalities – disruption of alveolar ventilation or capillary perfusion  
   a. Pulmonary embolism  
   b. Emphysema  

D. Clinical manifestations  

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<thead>
<tr>
<th>Respiratory distress</th>
<th>Hypoxemia</th>
<th>Hypercapnia</th>
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<tr>
<td>Nasal flaring</td>
<td>Restlessness ⇒ confusion ⇒ lethargy ⇒ coma</td>
<td>Headache</td>
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<tr>
<td>Pursed lip breathing</td>
<td>Tachycardia ⇒ dysrhythmias</td>
<td>Irritability</td>
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<td>Three-point positioning</td>
<td>Tachypnea</td>
<td>Confusion</td>
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<td>Only one or two words spoken between breaths</td>
<td>Dyspnea</td>
<td>Inability to concentrate ⇒ somnolence ⇒ coma</td>
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<td>Cough</td>
<td>Use of accessory muscles</td>
<td>Bradypnea</td>
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<tr>
<td>Use of accessory muscles</td>
<td>Mild hypertension (early) ⇒ hypotension (late)</td>
<td>Tachycardia ⇒ dysrhythmias</td>
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<tr>
<td>Sternal retractions</td>
<td>Possible cyanosis</td>
<td>Hypotension</td>
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<td>Intercostal retractions</td>
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<td>Facial redness</td>
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E. Management  

Treat the cause  
Maintain adequate gas exchange  
\[ \text{PaO}_2 > 60 \text{ mmHg; SaO}_2 > 90 \text{ mmHg; hgb} > 12 \text{ g/dl, hct} > 36\%; \]  
\[ \text{CO} 4.0 - 8.0 \text{ L/min, CI} 2.5 - 4 \text{ L/min} \]  
Oxygen therapy - may use bi-pap mask  
Preset positive pressure is delivered during inspiration and expiration; used in an effort to avoid mechanical ventilation  

HOB 30° - 45°  
Turn, cough, deep breathe; chest PT; expectoration of sputum, suctioning  
Assessment of VS, breath sounds Q1 - 4 hours  
Teach patient purse-lipped, diaphragmatic breathing  
Encourage fluids; administer IV fluids as needed  
Monitor ABGs  
Pharmacology (See back page for detailed pharmacologic information):  
   β-adrenergic bronchodilators: Proventil, Alupent  
   Expectorants: guaifenesin  
   Antibiotics  
   Antipyretics  
   Inotropes: dobutamine, amrinone  
   Antidysrhythmics
Monitor for complications: dysrhythmia, pneumonia, pulmonary edema, PE, barotrauma, oxygen toxicity, acid-base imbalance, GI ulceration, DIC, sepsis, shock
May require mechanical ventilation with or without PEEP

II. Mechanical ventilation
A. Description: used to support alveolar ventilation, reduce work of breathing, or aid in supporting oxygenation
B. Indications
   PaO₂ < 60 mmHg when Fio₂ is above 0.6 L; PaCO₂ < 30 or > 50 mmHg;
   RR > 30 - 35 BPM or < 10 BPM, Ph < 7.30 with rising PaCO₂
C. Modes and uses of mechanical ventilation
   Negative pressure: how we normally breathe; ex. Iron lung or cuirass. Not used in acute failure
   Positive pressure: force air into lungs. Reverse of normal breathing.

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<th>Assist control (A/C)</th>
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<tr>
<td><strong>Pressure</strong></td>
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<td>![Pressure graph]</td>
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Tidal volume and rate guaranteed. Preset machine breaths and spontaneous patient breathes all receive set tidal volume.
Uses: initial settings, resting settings during weaning

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<th>Synchronized intermittent mandatory ventilation (SIMV)</th>
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<tr>
<td><strong>Pressure</strong></td>
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<td>![Pressure graph]</td>
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Mandatory preset tidal volume and rate. Patient spontaneous breaths receive tidal volume that patient is able to draw in.
Uses: surgical patients briefly requiring ventilator support, weaning
Continuous Positive Airway Pressure (CPAP)

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<th>Pressure</th>
<th>Volume</th>
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<td><img src="#" alt="Pressure Chart" /></td>
<td><img src="#" alt="Volume Chart" /></td>
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Preset pressure above atmospheric at end-expiration during spontaneous breathing. All breaths spontaneous, supported.

Uses: weaning trials with or without pressure support ventilation

Pressure support ventilation (PSV)

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<th>Volume</th>
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Preset positive pressure delivered when spontaneous breath is initiated. Ends when inspiration is concluded.

Uses: weaning with SIMV or CPAP

D. Positive End-Expiratory Pressure (PEEP)

1. Maintenance of pressure above atmospheric at end-expiration during ventilator-initiated or assisted breathing
2. Maintains alveolar air flow during expiration
   - Physiologic PEEP = 3 - 5 cm
   - Therapeutic PEEP = 5 - 20 cm
3. Goal is to maintain PEEP at the level that provides SaO₂ > 90% without compromising CO
4. Actions of PEEP
   - Increased FRC and time for gas exchange
   - Improves PaO₂ without increasing FiO₂
     - Lungs can tolerate FiO₂ up to 50% indefinitely; > 50% can cause alveolar collapse and Type II pneumocyte injury
   - Decreases surface tension
   - Prevents alveolar collapse at end-expiration
   - Reduces shunt, improves V/Q matching
   - Decreases work of breathing
5. Adverse effects of PEEP
   - Hemodynamic: decreases venous return (\(\bar{V}CVP\)); increases right ventricular afterload (\(\bar{V}PVR\)); decreases CO
Barotrauma: the higher the PEEP, the greater the risk of increased ICP.

E. Mechanical ventilator parameters
   Tidal volume 8-10 ml/kg
   Rate usually 8 - 16 BPM
   FiO₂ initially 100% for the first 20 minutes; then adjust to keep PaO₂ at least 60 mmHg
   May need to add PEEP to achieve optimal PaO₂.
   Humidification 100%; temperature 32° - 37°C
   I:E ratio 1:1.5 or 1:2

F. Alarms
   High peak inspiratory pressure – difficulty or inability to deliver tidal volume
   Due to increased airway resistance
   Secretions, bronchospasm, tubing kink, coughing, biting ER tube, water in tubing
   Due to decreased compliance
   Pneumothorax, pulmonary edema, atelectasis, pneumonia, ARDS
   Low exhaled volume - low resistance to delivery of tidal volume
   Due to disconnection from ventilator, cuff leak, circuitry leak
   High respiratory rate – usually due to patient just intubated, oxygenation not yet under control, or patient not tolerating weaning.
   Apnea - patient fatigued, over sedated, decreased LOC

G. Complications
   Decreased CO₂, barotrauma, atelectasis, hyper/hypocapnia, oxygen toxicity, aspiration, infection, anxiety, inability to wean, patient-ventilator asynchrony

H. Nursing care
   1. Monitor oxygenation – ABGs, pulse oximetry, end-tidal CO₂
   2. Secure ETT or tracheostomy
      Move ETT from side to side every 24 hr.
      Suction above cuff prior to move
   3. Suction as needed
      Do not lavage with saline
   4. Pulmonary assessment.
   5. Cardiac assessment
   6. Ensure bag-valve-mask and suctioning equipment is available in room
   7. Support patient and family
   8. Facilitate communication with sign board, pad and pencil
   9. Mouth care every 2-4 hours
   10. Head of bed at 30 degrees
   11. Assess patients’ readiness to wean

I. AACN Practice Alert: Ventilator-associated pneumonia (VAP)
   1. Colonization of oropharynx is a critical factor in the development of nosocomial pneumonia.
   2. Growth of potentially pathogenic bacteria in dental plaque proves an opportunity for infection
   3. Show to be responsible for the development of VAP
5. Brush teeth, gums and tongue at least twice a day with soft toothbrush
6. Provide oral moisturizing to oral mucosa and lips every 2-4 hours
7. Use oral chlorhexidine gluconate 0.12% rinse twice a day during perioperative period for cardiac surgery patients
8. Document frequency of oral care

J. Weaning
1. Description
   Gradual withdrawal of ventilatory support for patients who have been mechanically ventilated for more than 24 hours
2. Indications of readiness to wean
   Improvement in disease process
   Adequate strength, nutrition, LOC, afebrile
   Hemodynamically stable, hgb acceptable
   Does not require > 5 cm PEEP or Fio₂ > 50%
   PaO₂ at least 60 mmHg
   PaCO₂ < 45 mmHg or at patients baseline
   RR < 25 BPM
   Psychologically prepared
3. Methods
   T-piece
   ETT attached to tubing by T-junction, and connected to oxygen source.
   Increased work of breathing
   If tolerate for 30 minutes, consider extubation
   SIMV with PSV
   Decrease mandatory breaths, then decrease PSV to 8 cm H₂O
   CPAP with or without PSV
   If tolerate CPAP alone for 30 minutes, consider extubation
   CPAP with PSV: Gradually decrease PSV to 8 cm H₂O
   Evidence-based practice: weaning protocols must be used. Don’t have to know various methods, only that regulatory agencies require a protocol for weaning.
4. Management
   Semi- or high-fowler’s
   Avoid sedation
   Reduce carbohydrate ingestion
   Assess for signs and symptoms of fatigue:
   ↓LOC
   SBP↑ or ↓20 mmHg; HR ↑ 30 bpm or above; RR > 30 or < 10
   PVCs, runs of VT
   Use of accessory muscles
   SaO₂ < 90%; PaCO₂ ↑ 5 - 8 mmHg; pH < 7.30
   Patient complaint of dyspnea, fatigue, pain
III. **Adult Respiratory Distress Syndrome (ARDS)/Acute Lung Injury (ALI)**

A. **Description**
   A fulminant form of respiratory failure, characterized by acute lung inflammation and diffuse alveolocapillary injury

B. **Causes** - May either be as a result of direct injury to alveolocapillary membrane or indirect damage from chemical mediators in response to systemic disorders

<table>
<thead>
<tr>
<th>Direct Pulmonary Injury</th>
<th>Indirect Pulmonary Injury</th>
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<tbody>
<tr>
<td>Aspiration</td>
<td>Sepsis</td>
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<tr>
<td>Inhalation injury</td>
<td>Pancreatitis</td>
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<tr>
<td>Pneumonia</td>
<td>Multiple trauma</td>
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<tr>
<td>Embolism</td>
<td>Shock</td>
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ALI can also be caused by Transfusion-Related Acute Lung Injury (TRALI)
   Mediator activation, endothelial damage and capillary leak as a result of blood transfusion
   Clinical findings like that of ALI and ARDS, but with correlation to components in blood products

C. **Role of mediator systems in the development of ARDS**
   Neutrophils, macrophages, complement, platelet activation factor, humoral vasoactive substance and other mediators are activated, causing:
   - Initiation of inflammatory response
   - Edema from injury to lung tissue
   - Increased permeability
   - Pulmonary hemorrhage
   - Injury to endothelium
   - Pulmonary hypertension
   - Emboli
   - Increased airway resistance

D. **Pathophysiologic consequences of ARDS/ALI**
   - Interstitial pulmonary edema
   - Shunting leading to ventilation-perfusion mismatches and hypoventilation
   - Increased alveolar deadspace
   - Decreased lung compliance, increased work of breathing and airway resistance.
   - Refractory hypoxemia

E. **Clinical manifestations**
   - Rapid deterioration of pulmonary function is a hallmark of ARDS
   - Respiratory rate greater than 30 BPM, intercostal and suprasternal retractions
   - Crackles and/or decreased breath sounds
   - Crackles, rhonchi on auscultation; dullness on percussion

F. **Diagnostic findings**
   - CXR: Patchy infiltrates ⇒ diffuse infiltrates ⇒ consolidation (“white-out”)
   - PFTs: $V_t$, $VC$, FRC, compliance all decreased, shunting, increased A-a gradient,
   - $P/F$ ratio: ALI ≤300 mmHg; ARDS ≤200 mmHg
   - Arterial Blood Gas results
   - Early stages ARDS/ALI
pH > 7.45 (respiratory alkalosis)
PaCO₂ < 35 mmHg (hypocapnia)
PaO₂ < 70 mmHg (refractory hypoxia)

Later stages ARDS
pH < 7.35
PaCO₂ > 45 mmHg
HCO₃ < 22
Mixed respiratory and metabolic acidosis.

Hemodynamic parameters: PAOP < 18 mmHg, ↑ PAD, ↑ PVR; CO, CI normal until later stages

G. Collaborative management of ARDS/ALI
Goal of treatment is adequate oxygenation and tissue perfusion, and systems support while lungs heal

Oxygen therapy
CPAP/BiPAP mask may be sufficient for ALI
ARD will require mechanical ventilation
Low tidal volume therapy
Ardsnet study
Assist control mode
Lower tidal volumes than traditionally given – 4-8 ml/kg
Lower plateau pressures - <30 cm H₂O
RR 6-35 bpm, keeping pH 7.30-7.45
FiO₂ and PEEP adjusted to keep PaO₂ at 55-80 and/or SpO₂ at 88-95%
Set in prescribed combinations according to ARDSnet protocol.

Decrease metabolic oxygen needs to decrease oxygen demand
Decrease anxiety and pain with sedation and narcotic analgesics
Decrease muscle movement with sedation and paralytics
Decrease fever with antipyretics and cooling blankets

Ensure adequate oxygen transport and delivery
Monitor H & H
Maintain PaCO₂ at normal levels
Monitor ABGs
Monitor PO₂

Fluid therapy
Maintain balance between fluid overload and fluid depletion
Need hemodynamic monitoring with PA catheter
Keep PAOP low
Administer isotonic solution as needed
Administer diuretics when indicated

Nutritional support
Enteral feedings preferred; may need TPN
Require protein, caution with carbohydrates

Pharmacology
Sodium bicarbonate for mild to moderate acidosis
Positive inotropes to increase cardiac output
Vasopressors for hypotension
Neuromuscular blockading agents to decrease metabolic oxygen demands
Anxiolytics to decrease anxiety, as adjunct to NMB
Antibiotics for sepsis or nosocomial pneumonia

Prone positioning
Used to improve gas exchange
  - Alveoli flooded with fluid and debris, impeding gas exchange
  - Promotes increased blood flow to different area of lung
  - Part of lung that is down receives best blood flow
  - Takes hours for fluid and debris to settle
  - Result is recruited alveoli

Most effective when used in early stages of ARDS
Multiple staff, pronating device, or specialty bed may be used

**Certification Questions**

1. Which of the following sets of arterial blood gas results indicates acute respiratory failure that requires immediate endotracheal intubation?
   - A. pH 7.30, PaO₂ 69 mm Hg, PaCO₂ 48 mm Hg
   - B. pH 7.50, PaO₂ 64 mm Hg, PaCO₂ 52 mm Hg
   - C. pH 7.20, PaO₂ 60 mm Hg, PaCO₂ 53 mm Hg
   - D. pH 7.50, PaO₂ 65 mm Hg, PaCO₂ 20 mm Hg

2. A patient remains intubated and ventilated in the ICU following a prolonged course of pneumonia. He has been on pressure support ventilation (PSV) and continuous positive airway pressure (CPAP) at 5 H₂O. His spontaneous breathing trial of FiO₂ at 50% lasted for 30 minute and produced the following ABG results: pH 7.28, PaCO₂ 67 mmHg, PaO₂ 70 mmHg. What are the most appropriate ventilator settings to promote weaning in this patient following this breathing trial?
   - A. Keep the FiO₂ at 50% and titrate the PS to maintain a respiratory rate less than 30/min
   - B. Return the patient to his previous ventilator settings with FiO₂ 40%
   - C. Place the patient on a T-piece at FiO₂ 60% and prepare for extubation
   - D. Increase the patient’s CPAP to 7 H₂O and recheck the PaCO₂ to ensure correction of acidosis.

3. A patient in the ICU with ARDS is intubated and mechanically ventilated on the following settings: tidal volume 600 ml, rate 12/min, FiO₂ 70%, PEEP 15 cm H₂O. The peak inspiratory pressure is 50 cm H₂O, and the patient’s respiratory rate is 12/min. Arterial blood gas values include pH 7.25, PaO₂ 60 mmHg, PaCO₂ 48 mmHg. Which of the following findings would best indicate clinical improvement in this patient?
   - A. Peak inspiratory pressure 43 cm H₂O
   - B. PaCO₂ 45 mmHg
   - C. PaO₂ 62 mmHg
   - D. Respiratory rate 14/min
4. The arterial hypoxemia associated with ARDS is attributed to:
   A. A fluid shift from the interstitial areas into the pulmonary vasculature
   B. Shunting resulting from the collapse of alveoli
   C. The formation of hyaline membranes over the alveoli
   D. Increased lung compliance

IV. **Thoracic Surgery**
   A. Types and indications
      1. Pneumonectomy
         a. Removal of entire lung
         b. Indications
            Malignancy
            Tuberculosis
            Bronchiectasis
            Multiple abscesses
      2. Lobectomy
         a. Resection of one or more lobes
            Malignancy
            Tuberculosis
            Bronchiectasis
            Abscesses
            Trauma
      3. Wedge resection
         a. Removal of wedge-shaped section of lung tissue
            Small lesions
            Pulmonary blebs
      4. Lung volume reduction
         a. Resection of damaged lung tissue to achieve more normal chest wall
            Severe emphysema
      5. Bullectomy
         a. Resection of large bullae
            Severe emphysema
   B. Collaborative postoperative management
      1. Ventilatory support
         a. Oxygenation
         b. Acid-base
         c. Work of breathing
      2. Chest tube management
      3. Pain control
         a. May have an epidural catheter, PCA pump
         b. Use of guided imagery and relaxation therapy has been shown to be effective
      4. Positioning
         a. Lobectomy “good lung” down
            Improves blood flow and V/Q matching
         b. Pneumonectomy operative side down
Assists with incisional splinting  
c. Early ambulation and sitting at bedside  
   Improves diaphragmatic excursion, ventilation and inflation  
d. Deep breath/IS  
   Improve lung reexpansion  
   Prevent atelectasis  
5. Monitor, prevent and manage complications  
   a. Acute respiratory failure  
   b. Hemorrhage  
   c. Dysrhythmias and pulmonary edema

V. Pulmonary hypertension  
A. Description  
   1. Primary or idiopathic  
      a. Cause unknown or genetic  
   2. Respiratory hypoxemic disorders  
      a. COPD, sleep-disordered breathing  
   3. Thromboembolic disorders  
      a. Large PE or multiple micorembolic thrombi  
   4. Pulmonary venous hypertension  
      a. CAD, acute MI, valvular disease, HF, dilated cardiomyopathy  
         Result of elevated pressures in left heart that increase pulmonary venous pressure  
B. Diagnosis  
   1. Clinical symptoms nonspecific in early stages  
   2. Echocardiogram  
      a. Right atrial and ventricular enlargement  
      b. Decreased right heart function (cor pulmonale)  
      c. Displaced intraventricular septum  
      d. Tricuspid regurgitation  
   3. Pulmonary function tests  
      a. Not diagnostic of PAH; rules out any other pulmonary diseases  
   4. Sleep study  
      a. Screen for sleep apnea, a cause of pulmonary venous hypertension  
   5. Right-sided cardiac catheterization  
      a. Invasive, considered gold standard, due to ability to measure pressures  
      b. Pulmonary artery mean pressure elevated  
      c. Elevated right atrial pressure is poor long-term prognostic sign  
      d. Evaluate ability of pulmonary vessels to dilate in response to inhaled nitrous oxide, a pulmonary arterial vasodilator  
C. Collaborative management  
   1. Medication therapy  
      a. Prostacyclin  
         Epoprostenol (Flolan) IV  
         Very short half-life, cannot be interrupted
Most effective in advanced acute stage
Treprostinil (Remodulin)
  Administer with SC infusion catheter and pump
  Half life of 3 hours
b. Bosentan (Tracleer)
  Only oral medication
  Blocks vasoconstrictor, endothelin
c. Anticoagulation
2. Administer and assess effectiveness of medications
3. Patient and family education
   Written medication list
   Symptoms to report to HCP
   Prevention of bleeding D/T anticoagulation
     Soft toothbrush, electric razor, no contact sports
   Monitor for excess bleeding
     Skin, gums, urine, stool

VI. Pulmonary Trauma
A. Mechanism of injury
   Blunt injury
     MVC, falls
     Acceleration/deceleration injuries
     Crush injuries
   Penetrating injury
     Guns, knives
     Injury depends on pathway
B. Flail chest
   Description
     Two or more ribs fractured in two or more places
     No longer attached to thoracic cage
   Assessment and diagnostic findings
     Paradoxical chest wall movement indication
     Wall moves in opposite direction as rest of chest during respirations
     Crepitus over area of fractures
     Fractures on CXR
     Hypoxia on ABG
   Collaborative interventions
     Adequate oxygenation
     May require intubation and mechanical ventilation
     Pain management
     May require fluids
C. Pulmonary contusion
   Hemorrhage followed by alveolar and interstitial edema
   May be localized to area of contusion or spread throughout lungs
   May lead to ALI and ARDS
May not be apparent for 48 hours

Diagnosis
  CXR: pulmonary infiltrate over area of contusion
  ABG: hypoxemia
  Worsen over 24 hours, then resolve or progress to ARDS

Interventions
  Pain management including intercostals nerve block or thoracic epidural analgesia
  Aggressive respiratory care
    Ambulation, deep-breathing exercises, turning, IS
    Aggressive removal of airway secretions
    May not tolerate chest PT if fractured ribs present

Complications
  ARDS, pneumonia, lung abscess, PE
  D. Pneumothorax
    Tension pneumothorax, open pneumothorax, hemothorax
  As discussed

Certification Questions
  1. A 55-year-old patient is postoperatively admitted to the PCU following right lower lobectomy for adenocarcinoma. He is alert and oriented with stable vital signs, respiratory rate 25/min, and requires 4 L of oxygen via nasal cannula to maintain an O2 saturation of 95%. During the night his SaO2 falls to 90%, his respiratory rate increases to 40, and he complains of increased pain in the right chest. His breath sounds are diminished in his bases bilaterally, but otherwise are clear. What are the most appropriate nursing actions?
     A. Place patient on a face mask at FiO2 .40 and draw an ABG
     B. Administer pain medication and encourage the patient to cough and use the incentive spirometer
     C. Obtain an order for chest x-ray to evaluate for hemothorax and increase oxygen to 6L/min
     D. Administer a sedative and encourage use of deep breathing and relaxation techniques

  2. Treatment for pulmonary artery hypertension with an elevated mean pulmonary artery pressure and signs of right heart failure including tricuspid regurgitation murmur consists of administration of oxygen and:
     A. Phlebotomy to maintain hematocrit at 48%
     B. Fluid bolus to increase right ventricular output
     C. Epoprostenol (Flolan) to dilate pulmonary arteries
     D. Inotropic agents to increase right ventricular contractility

  3. After a motor vehicle collision, a teenager is admitted with chest pain and an ineffective cough with hemoptysis. The admission chest X-ray reveals consolidation and pulmonary infiltration, likely evidence of a sever pulmonary contusion. The patient’s respiratory rate is 23/min, and his arterial blood gases on room air are pH 7.42, PaCO2 30 mmHg, PaO2 54 mmHg, HCO3⁻ 24 mEq/L. Which of the following pairs of interventions is most appropriate for this patient?
     A. Administer an analgesic, apply 100% O2 by face mask
Appendix

Respiratory medications

β-adrenergic agonist inhaler (albuterol [Proventil, Alupent])
- Bronchodilator - Relaxes bronchial smooth muscle
- Adverse effects: tachycardia, dysrhythmia, hypertension

Anticholinergic (Ipratropium [Atrovent]) inhaler
- Bronchodilator - Relaxes bronchial smooth muscle
- Used in combination with beta2-agonists (Combivent)
- Side effects as with beta2-agonist
- Not for use in acute exacerbations

Corticosteroids (Prednisone, methylprednisolone [Solu-medrol])
- Antiinflammatory
- Side effects: hyperglycemia, immunosuppression

Inhaled combination corticosteroids and long-acting β-adrenergic agonist (fluticasone/salmeterol [Advair], budesonide/formoterol [Symbicort])
- Fewer steroid-related complications at all but highest doses

Methylxanthines (Theophylline)
- Bronchodilator
- Side effects: Tachycardia, dysrhythmia, restlessness, headache
- Current guidelines do not recommend

Bibliography


