Acute Respiratory Failure
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Acute respiratory failure
- Ventilation/perfusion mismatching
  - Most common cause of hypoxemia
  - Normal is 1:1 ratio or "1"
  - Ventilation > blood flow is a ratio >1
  - Ventilation < blood flow is a ratio <1

Acute respiratory failure
- Causes of decreased ventilation to an area of lung
  - Increased secretions obstructing the airway
  - Bronchospasm
  - Hypoxemia due to partial ventilation of alveoli responds to increased FiO2
  - Pulmonary embolus causes decreased perfusion with normal ventilation
Diffusion defects

Six barriers for diffusion of O₂ and CO₂:
1. Surfactant
2. Alveolar membrane
3. Interstitial fluid
4. Capillary membrane
5. Plasma
6. RBC membrane

Factors affecting diffusion capacity:
- Distance between the alveolar membrane and the capillary membrane in 1-2 cells thick
- Fluid in the interstitial space increases the distance between the membranes

Diffusing capacity:
- Diffusing capacity is decreased by fibrotic changes of the lung tissue
- As the diffusing capacity decreases, the pO₂ is first affected, leading to hypoxemia
- CO₂ is more readily diffusible than oxygen, therefore hypercapnia is a late sign of a diffusion defect

Diffusion defects:
- Decreased ventilation leads to increased pCO₂ (hypercapnia)
- Increased ventilation leads to decreased pCO₂ (hypocapnia)
Hypoventilation and V/Q mismatch result in hypercapnia.
Hypercapnia leads to increased cerebral blood flow causing headache, increased CSF pressure, and papilledema.
Symptoms include restlessness, anxiety, slurred speech, decreased level of consciousness.

Definition: the volume of air (inspired gas) that fills the upper and lower airways.
The airways do not play a part in gas exchange.
Space is normally 25-30% of the inspired volume.
Major mechanism to change in pCO₂ is the alteration in the volume of dead space (Vd) in relation to the entire.

Dead space increases when perfusion to a ventilated area is decreased.
The area is no longer participating in gas exchange.
Older patients have a lower ventilatory response to hypoxia and hypercapnia.
Possible decrease compensatory change in heart rate, increased stroke volume, and cardiac output seen in younger patient.
Therefore an increase in pCO₂ and decrease in pH are concerning in the older patient.
Nursing assessment

- Neuro:
  - Change in mental status due to hypoxemia and hypercapnia
  - Anxiety
  - Restlessness
  - Confusion
  - Lethargy
  - Can lead to severe somnolence and coma

- Respiratory
  - Rate
  - Depth of respiration
  - Pattern of respirations
  - Cough
  - Presence of sputum
    - Amount and character
  - Diaphoresis
  - Accessory muscle use
  - Retractions
    - Indicate respiratory muscle fatigue
  - Palpate for symmetrical chest expansion and tactile fremitus
  - Auscultation to assess adequacy of airflow and presence of adventitious breath sounds

- Cardiac
  - Observe for signs and symptoms of decreased perfusion
    - Hypotension
    - Dysrhythmias
    - Decreased peripheral pulses
    - Decreased skin temperature
    - Cyanosis
    - Pallor
  - Nutritional status
    - Important for maintaining respiratory muscle strength
Additional assessment: Serial chest X-rays and labs
- Electrolytes
- Determine adequate muscle function
- Hgb and Hct
- Oxygen carrying capacity
- WBC
- Possible infection or status of infection
- SpO2
- Assess adequacy of oxygenation and ventilation

Goal of treating the patient with acute respiratory failure
- Maintain patent airway
  - ET or trach
  - Precautions to prevent premature extubation
  - Secure placement
  - Reassuring/reorienting patient
  - Sedation
  - Restraints
  - Therapeutic paralysis
- Optimize oxygen delivery
  - Supplemental O2
    - Nasal prongs, mask, ventilator
  - Patient with unilateral lung disease position with better functioning lung down to perfuse side with better ventilation
  - Adequate Hgb level and cardiac output

Goal of treating the patient with acute respiratory failure
- Minimize oxygen demand
  - Rest
  - Agitation, restlessness, fever, sepsis, and patient-ventilator dyssynchrony all contribute to increased O2 demand and consumption
- Treat the cause of ARF
  - ARDS, COPD, asthma exacerbation, pneumonia, and PE
COPD

- Progressive and often irreversible airflow limitations associated with an inflammatory response to noxious particles or gases
- Fourth leading cause of death in U.S.A. after heart disease, cancer, and stroke and increasing
- Primary cause is tobacco smoke
- Second most common cause is uncontrolled asthma

COPD-continued

- Primary pathogenic mechanism is chronic inflammation
- Exposure to an irritant causes airway inflammation (injury to tissues)
- Tissue injury results in repair of the injury with remodeling, leading to scarring
- Scarring from remodeling results in narrowing and obstruction of the airways
  - Remodeling inhibits the airways ability to relax
COPD-continued
- Obstruction of the alveolar walls and connective tissue lead to permanent enlargement of air spaces and enlargement of mucus secreting glands and increased number of goblet cells, resulting in increased mucus production
- Cellia are destroyed and no longer able to clear thick tenacious mucus blocking the airway
- Pulmonary capillary walls thicken, inhibiting gas exchange

COPD-continued
- COPD has an on-going gradual decline in lung function
  - Little respiratory reserve
  - Any condition that increases the work of breathing worsens the V/Q mismatching

COPD-continued
Common causes of ARF in COPD
- Acute exacerbation
- Heart failure
- Dysrhythmia
- Pulmonary edema
- Pneumonia
- Dehydration
- Electrolyte imbalance
COPD-continued

Physical exam
• Barrel chest
• Accessory muscle use
• Pursed lip breathing
• Clubbing due to prolonged hypoxia
• Wheezing
• Decreased breath sounds
• Prolonged expiratory phase
• Crackles

ABG
• Mild hypoxia in early disease
• Worsening hypoxia and hypercapnia (also see in more advanced disease)
• Over time kidney increases HCO₃ production and retention in effort to keep blood pH normal

COPD-continued

• Change in character of sputum may indicate infection which can lead to ARF

COPD-continued

Symptoms
• Anxiety
• Chest pain or tightness
• Weakness
• Malaise
• Weight loss
• Fever
• Sleep problems
• Wheezing
• Retractions

• Tachycardia and hypotension may result from decreased cardiac output
COPD-continued
- Chronic changes may see pO2 and pCO2 be almost the same
- In failure pO2 drops further as pCO2 increases causing acidosis with hypoxia

COPD-intervention
- Support
- Treat the cause
- Correct hypoxemia with supplemental O2 to achieve pO2 >60 or SpO2 >90%
  - High flow O2 may blunt hypoxic drive, leading to CO2 retention
- Bronchodilators
  - SABA
- Steroids
  - IV Solu-Medrol or oral steroids for 10-14 days
  - Decreased length of hospital stay
  - Improved FEV1
  - Side effects include hyperglycemia, increased risk of infection, skeletal muscle myopathy

COPD-intervention
- Antibiotics indicated when patient presents with increased SOB with increased sputum production and purulence or change in color
- Common organisms include:
  - h. flu
  - Strep pneumoniae
  - m. catarrhalis
- Sputum culture guides choice of drug
  - Commonly use third generation cephalosporins (ceftriaxone or cefepime) or second generation fluoroquinolones (Avelox or Levaquin)
COPD-intervention

- Ventilatory assistance
  - NPPV-noninvasive positive pressure ventilation (CPAP)
  - Candidate is hemodynamically stable with patent airway, minimal secretions, no N/V or mental status changes
- Intubation/mechanical ventilation indicated when despite aggressive therapy the patient develops mental status changes, respiratory muscle fatigue, respiratory acidosis, or significant hypoxemia

Acute respiratory failure and asthma

Asthma

- Chronic inflammatory disorder of airways that are hyperresponsive
- Inhaled allergies, viruses, or other irritants cause
  - Bronchoconstriction
  - Airway edema
  - Mucous plugging
  - Airway remodeling
Asthma

- Airway obstruction leads to air trapping with prolonged exhalation
- V/Q mismatch with increased intrapulmonary shunt
- Unlike COPD airflow limitations, the obstruction is mostly reversible

Asthma

Early symptoms

- Wheezing
- Dyspnea
- Chest tightness
- Cough
- Initially hyperventilates, leading to respiratory alkalosis
- As airway narrows it becomes more difficult to exhale and lungs become hyperinflated and stiff causing increased work of breathing

Asthma

Physical exam

- Tachycardia
- Tachypnea
- Agitation
- Possible accessory muscle use
- Suprasternal retractions
- Peak flow <50% of patient’s normal
Asthma

**Asthma asthmaticus**
- Bronchoconstriction does not respond to bronchodilators and progresses to ARF

**Symptoms**
- Fatigue due to severe dyspnea
- Cough
- Increased work of breathing
- Hypercapnia
- Hypoxia
- Respiratory acidosis and cardiac output decreases due to decreased venous return related to increased intrathoracic pressure

**Asthma**

- Treatment
  - Supplemental O2
  - Systemic steroids
  - Bronchodilators
  - Some patient may need intubation/mechanical ventilation

- Recovery phase:
  - Begin teaching management techniques, i.e. asthma action plan

**Acute Respiratory Failure and Nosocomial Pneumonia**

**Ventilator Associated Pneumonia (VAP)**
Acute Respiratory Failure and VAP

- Occurs >48 hours after hospitalization
- Excluding any infection incubating at admission
- VAP is 83% of nosocomial pneumonias in the ICU
- Can extend the hospital stay as much as 13 days
- Pathogens are inherent to the patient’s endogenous flora or hospital environment
- Usually secondary to aspiration of colonized bacteria from the oropharynx or GI tract

Acute Respiratory Failure and VAP

**Stage 1**
- Early onset
  - <4 days from intubation
  - Commonly associated with community acquired organisms
  - S. pneumoniae, MSSA, h. flu

**Stage 2**
- Late onset
  - Usually enteric gram negative rods and antibiotic resistant nosocomial organisms
  - Pseudomonas a., enterobacter, acinetobacter species, MRSA

Acute Respiratory Failure and VAP

- Symptoms include:
  - Fever >38.5 or <36.5
  - WBC >11,000 or <5000
  - Purulent ET secretions with positive cultures
Acute Respiratory Failure and VAP

- Interventions aimed toward prevention and treatment
- Prevention most effective is hand hygiene

- Avoid reintubation
- Oral ETT instead of nasal
- ETT with continuous aspiration of subglottic secretions
- Suction secretions above cuff before deflating or repositioning tube
- Aspiration of oropharyngeal secretions is the primary route for mechanically ventilated patients to acquire VAP
- Elevate the HOB 30-45 degrees unless contraindicated
- Comprehensive oral hygiene program (chlorhexadine)

VAP is a potentially severe infection with increased risk of mortality without appropriate antibiotic therapy
### Acute Respiratory Failure and VAP

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<th>Early onset VAP</th>
<th>Late onset VAP</th>
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<td>- Monotherapy for patient with history of previous antibiotic therapy</td>
<td>- Combination therapy</td>
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<tr>
<td>- Combination therapy for patient with COPD, prolonged steroids, or malnutrition</td>
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