Disorders of Ventilation

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Class 9 Objectives

- Upon completion of this lesson, the student will be able to
  - design nursing interventions for patients presenting with atelectasis, pneumonia, TB, and COPD,
  - recognize clinical manifestations of respiratory disease for different age groups,
  - state the signs and symptoms of respiratory failure and pulmonary embolism.
- Describe the pathophysiology of ARDS, signs/symptoms, and treatment.
- Compare and contrast the etiology, signs/symptoms, and treatment of the different diseases presented under the term of COPD with a classmate.

Respiratory Failure

- Dynamics: alveolar hypoventilation, V/Q disequilibrium, decreased FiO2 & inadequate exchange of gases between alveoli & blood
- Results in hypoxia, hypercapnia, and acidosis
- Work of breathing becomes difficult, exhaustion occurs, and there is no energy to breathe.
- Risk factors: pneumonia, Hantavirus, sepsis, atelectasis, bronchospasms, CHF
  - atelectasis: areas of the lung where alveoli are collapsed (alveoli are airless & no gas exchange)
Respiratory Failure
Failure of Oxygenation: Etiology

- "Hypoxemia" = reduced PaO2 concentrations
  - PaO2 < 80 mm Hg (Horne & Derrico, 1999)
  - Hypoventilation can reduce the PaO2 & PAO2
  - Factors leading to hypoventilation are drugs (opioids), neurological disorders (e.g. ICP) & COPD
- Ventilation/Perfusion Mismatching: V/Q ratio
  - Perfusion exceeds ventilation at the lung base
    - $4:5 = 0.8 = \text{normal V/Q}$
  - "In respiratory failure, the V/Q mismatching is the most common cause of hypoxemia" (Hartshorn, 1997, p. 325)

Respiratory Failure
Clinical S & S

- Severe dyspnea (difficulty breathing)
  - $> \text{RR & HR}$
  - use of accessory muscles of ventilation
- Cyanosis: indicative of unoxygenated hemoglobin
  - check nailbeds & mucous membranes
- Clubbing: check fingers and toes
- Multi-organ failure leading to death
- Tx: O2 & ventilation support

Pulmonary Embolism

[Image borrowed from http://www.benlovejoy.com/pulmonary_embolism_main.html]
Pulmonary Embolism

Image borrowed from the online NHS encyclopedia

Pulmonary Embolism: “PE”

A complication of a venous thromboembolism
- d/t immobility or leg injuries
- The most common preventable cause of hospital deaths
- 60-80% of fatal PE cases are not suspected

S & S
- Pleuritic chest pain (> with inspiration)
- Dyspnea: check ABGs
- tachycardia
- Dry cough & low grade fever
- Hemoptysis & syncope
- Abnormal ECG (up to 85% of PE patients)

Pulmonary Embolism: “PE”

- A common and challenging diagnosis
  - Diagnostic procedures:
    - Presenting symptoms assessed by medical team
    - Pulmonary angiography: ? Accuracy
    - V-P lung scan: not real definitive
    - CT angiography: accurate non-invasive tool
    - Can diagnose other intrathoracic disease as well
    - MRI, Echocardiography, CT angiography & venography
  - Treatment
    - Anticoagulation: heparin drip then coumadin
    - Streptokinase
    - Surgery
Chronic Obstructive Pulmonary Disease (COPD)

• Refers to 3 diseases:
  – Chronic bronchitis
  – Emphysema
  – Asthma

• Causes obstruction of airflow into the lungs

Chronic Bronchitis

Pathophysiology

• Bronchial inflammation with hypertrophy and hypersecretion of the bronchial mucus glands
• Pulmonary “fibrosis” (scarring) occurs due to inflammatory response, leading to “stenosis” of airway passages and airway obstruction
• Causes: inhalation of chemical or physical irritants
  – tobacco smoke, smog, occupational hazards
  – viral or bacterial infections

Chronic Bronchitis

• Clinical Sx:
  – productive cough is the earliest symptom
  – chronic productive cough for 3 months each year for 2 consecutive years (excluding other causes)
  – > Work of breathing (WOB) to overcome obstruction
• > PaCO2 with < PaO2
  – stimulus to breathe is low level of oxygen
  – breathlessness, rhonchi, cyanosis, increased susceptibility to infections
  – cor pulmonale: right-sided heart failure due to pulmonary hypertension; pulmonary edema occurs
  – “Blue Bloaters”: appearance of arterial blood is poor
Chronic Bronchitis

- Tx:
  - Cigarette smoking cessation programs
  - Prophylactic antibiotic treatment
  - Bronchodilators
  - Anti-inflammatory medications
  - Expectorants & hydration
  - O2 therapy
  - Vaccine against pneumococcal pneumonia

Emphysema

- A nonreversible obstructive disease characterized by the destruction of alveolar walls & connective tissue
- Terminal airways collapse during expiration & secretions are retained
  - < forced expiratory volume (FVE)
- Causes:
  - inhalation of physical or chemical irritants (almost always cigarette smoke)
  - genetic: very rare

Emphysema

- Clinical Sx:
  - tachypnea caused by hypoxia & hypercapnia
  - barrel chest configuration
  - non-productive cough
  - pursed-lip breathing
    - use of accessory muscles to aid in exhalation
  - respiratory acidosis
  - “Pink Puffers”
Emphysema

• Tx:
  – stop smoking and live in clean air
  – relaxation and energy conservation
  – breathing techniques to reduce air trapping
  – bronchodilators, antibiotics, hydration,
  – chest physiotherapy,
  – O2 therapy to assist with ADLs
• Prognosis:
  – poor for those who continue to smoke

Asthma

• Intermittent airway obstruction due to bronchospasm, bronchial edema, and > mucus secretions.
• Hyper-responsiveness of the airways after exposure to one or more irritating stimuli.
• Immunologic (Allergic, Extrinsic)
  – usually occurs in children
  – follows other allergic disorders (e.g., eczema)
  – IgE levels are elevated
• Nonimmunologic (Nonallergic, Intrinsic)
  – usually does not occur until adulthood
  – associated with recurrent upper RTI
  – IgE levels are not generally elevated

Asthma
Pathophysiology

Episode may be triggered by
  – physical exertion
  – change in temperature and humidity
  – emotional stress: PNS [] constricts bronchioles
  – Animal dander
  – Strong fumes
• Mast cells degranulate [] releasing histamine, SRSA, and ECF-A
Asthma

- Clinical S & S:
  - tachypnea (>RR)
  - wheezing
  - coughing at night
  - hyperventilate
  - < PaCO2
  - anxiety
  - dyspnea
- Tx: is based on staging
  - mild to severe
  - Prevent exposure to allergens
  - Avoid cigarette smoke
  - Inhalation of steroids
  - Oral use of steroids
  - Bronchodilators
  - Relaxation techniques

Pneumonia

- Inflammation of the respiratory unit tissue caused by a microorganism that is inhaled, circulated, or aspirated
- Common bacterial agents:
  - gram positive: strep pneumonia, mycoplamsa, staph aureas
  - gram negative: E. coli, proteus, P. aeruginosa
- Non-bacterial agents:
  - pneumocystis carinii, fungi, viruses, Legionella
- Clinical Sx: fever, chills, productive or dry cough, malaise, pleural pain, dyspnea, hemoptysis, leukocytosis
- Tx: rest, hydration, decongestants, cough suppressants, antibiotics for bacterial, Vitamin C

Tuberculosis

- A communicable infection of lung tissue
  - Mycobacterium tuberculosis
  - Lower respiratory tract infection
- Inhalation of droplets: colonizes respiratory bronchioles or alveoli
- Risk factors: living in close quarters, immigrants, HIV, malnourished, homeless in shelters
- Primary: overt disease occurring within 2 yrs. after infection
- Reactivation: disease that occurs later
**Tuberculosis**
- Patho: tubercles are formed in the lung
- Primary: first TB infection
  - About 5% or Americans infected with TB develop active clinical disease
  - A CMI reaction occurs and sensitized T cells develop
    - Positive skin test indicates a CMI reaction and previous exposure to the bacillus
  - Sputum culture will reveal the bacillus of an active tuberculosis
  - Chest x-ray demonstrates current or previous tubercle formation

**Tuberculosis**
- Clinical manifestations of active disease:
  - fevers (afternoon), malaise, night sweats, anorexia, productive purulent cough with chest pain
- Prevention: education & screening to < risk of infection and transmission
- Tx: of active disease
  - INH, Rifampin, and other non-resistant antibiotics

**Lung cancer**
- Defined as a malignant neoplasm arising in the epithelial lining of the respiratory tract or any lung tissue
- Four types:
  - squamous cell: 30% bronchogenic cancers
  - adenocarcinoma: 35-40% of all bronchogenic cancers
    - arises from the glands of the lungs
  - small cell (" oat cell"): 25% of all lung cancers
  - large-cell undifferentiated: 10-15% of all lung cancers
    - rapid metastasis, comfort measures
- Local tumor growth is invasive & erodes blood vessels and adjacent structures
Lung cancer
The most common fatal malignancy in the US for both males and females (2000)
156,900 people will die d/t this disease (2000)
many are minorities: black men have > incidence
• Primary risk factor is tobacco
  – Women have a higher risk of lung cancer from smoking than men. Why?
  – Air pollution, asbestos, chemicals, and dusts
• Clinical Sx: persistent cough, hemoptysis, recurring lower respiratory tract infection, respiratory failure

Pulmonary edema
• Third spacing in the interstitial spaces around the alveoli
• Conditions associated with pulmonary edema
  – Cardiogenic
    • MI, shock related to cardiac failure, hypertension
  – Noncardiogenic
    • Septic shock, aspiration pneumonia, fat emboli, burns
• Gas exchange is compromised
  – Hypoxemia occurs when alveolar-capillary membrane is impaired
• Clinical Sx: > WOB, dyspnea, pink frothy sputum, chest x-ray shows “whiteout”

ARDS
• A widespread breakdown of the alveolar and/or capillary membranes
• Patho: injury to lung results in hypoperfusion which damages the alveolar epithelium
  – > mediators of inflammatory response causes injury
  – > Permeability of alveolar capillary membrane
  – > edema leads to a < production of surfactant
  – A major cause of severe respiratory failure in clients with previously healthy lungs
• Risk factors:
  – Elderly and severe infections: > mortality rate
  – Occurs after major pulmonary, cardiovascular, or systemic insult
ARDS

- Clinical Sx:
  - Rapid, shallow breathing
  - Respiratory alkalosis
  - Dyspnea
  - Refractory hypoxemia
  - Diffuse alveolar infiltrates

- Tx: prevention of trauma to body
  - diuretics, digoxin, anti-inflammatory medications, O2 & ventilator therapy

Developmental differences

- CHILDREN: < surfactant until 28 weeks gestation
- lung tissue develops until ~ 8 years old
- airways are small
- muscles underdeveloped
- > exposure
- < immune system
- nasal breathers until 2 months

- ELDERLY: < muscle mass makes > WOB
- < immunity
- > risk for pneumonia
- > malnourishment
- pathology may place patient at risk for aspiration

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