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Medical Diagnosis/Disease: COPD

NCLEX IV (8): Physiological Integrity/Physiological Adaptation

NCLEX IV (7): Reduction of Risk

Anatomy and Physiology
Normal Structures

Primary function:

- Gas exchange
- Maintain acid base balance (pH) through regulation of CO₂.
- Supports vocalization, smell, and defense against pathogens.

Major divisions:

- Upper respiratory tract- nose, nasal cavity, sinuses, pharynx; filters, warms, and humidifies incoming air; provides resonance for speech.
- Lower respiratory tract- larynx, trachea, bronchi, bronchioles, lungs, alveoli; conducts air to and from gas exchange surfaces; performs gas exchange at alveolar level.

Anatomy overview:

- Nose/nasal cavity- lined with cilia and mucus to trap particles; warms and moistens air.
- Sinuses- air filled cavities; lighten skull and provide resonance.
- Pharynx- passageway for air and food; divided into naso-, oro-, and laryngo.
- Epiglottis- flaps that closes during swallowing to prevent aspiration.
- Larynx (voice box)- contains vocal cords and glottis; provides open airway and voice production; connects pharynx to trachea.
- Trachea (windpipe)- extends from larynx to main bronchi. Reinforced by C-shaped cartilage rings to prevent collapse; lined with ciliated mucosa from mucociliary clearance.
- Bronchial tree- right and left main bronchi->secondary (lobar)->tertiary (segmental) bronchi->bronchioles->terminal bronchioles->alveolar ducts->alveoli; airway diameter decreases, smooth muscle increases as branches narrow.
- Alveoli- tiny air sacs where gas exchange occurs; surrounded by pulmonary capillaries; type I cells: thin for diffusion; type II cells: secrete surfactant to reduce tension and prevent alveolar collapse; macrophages remove debris and pathogens.
- Lungs- right lung: 3 lobes (superior, middle, inferior); left lung: 2 lobes (superior, inferior); cardiac notch for heart; covered by pleura: visceral- lines lungs, parietal- lines thoracic cavity, pleural fluid- lubricates and reduces friction.

Physiology

- Ventilation: inspiration and expiration (passive at rest and active during exercise or distress).
- Gas exchange: occurs across respiratory membranes (alveolar epithelium + capillary endothelium + basement membrane); O₂ diffuses from alveoli->blood; CO₂ diffuses from blood->alveoli.

Pathophysiology of Disease

COPD is a progressive, irreversible lung disease characterized by chronic airflow limitation due to inflammatory damage in the airways, alveoli, and pulmonary vasculature. Encompasses chronic bronchitis and emphysema, often coexisting.

Pathophysiological process (step by step)

- Chronic inflammatory response- exposure to irritants -> inflammation of bronchi, bronchioles, and alveoli; inflammatory cells (neutrophils, macrophages, lymphocytes) release cytokines and proteases; results in airway wall thickening, fibrosis, and mucus gland hypertrophy.
- Airflow limitation- mucus hypersecretion (chronic bronchitis)-> narrowed airways and airflow obstruction; loss of elastic recoil and alveolar wall destruction (emphysema)-> air trapping and hyperinflation; cilia destruction-> impaired clearance of secretions.
- Gas exchange abnormalities- damaged alveoli reduce surface area for O₂-CO₂ exchange; leads to hypoxemia (low O₂) and hypercapnia (elevated CO₂); V/Q (ventilation perfusion) mismatch causes further oxygenation problems.
- Air trapping and hyperinflation- expiration becomes difficult -> air trapped alveoli; **over time, lungs remain partially inflated-> barrel chest, flattened diaphragm, increases work of breathing.**
- Pulmonary hypertension- chronic hypoxia-> pulmonary vasoconstriction-> **high pulmonary artery pressure; right ventricle hypertrophies to pump against resistance-> right sided heart failure (cor pulmonale).**
- Systemic effects- chronic hypoxia-> increased erythropoietin->polycythemia; increased blood viscosity -> decreased perfusion and oxygenation; weight loss and muscle wasting due to increased energy expenditure for breathing.

Anticipated Diagnostics

Labs

ABG- indicates gas exchange efficiency and severity of respiratory failure

SpO₂- monitor for hypoxemia; titrate O₂ cautiously to avoid CO₂ retention.

CBC- increased viscosity can impair perfusion; monitor for clot risk. Serum Alpha-1 Antitrypsin Level- confirms hereditary risk for early-onset emphysema.

Electrolytes chem 7- monitor during acute exacerbations. May show hypochloremia from chronic CO₂ retention or hypokalemia from diuretic use.

Sputum culture and sensitivity- identifies bacteria in infectious exacerbations. BNP (B-type natriuretic peptide)- helps differentiate COPD exacerbation vs. HF when dyspnea is present.

Additional Diagnostics

CXR- confirms hyperinflation and rules out pneumonia or HF. Pulmonary function tests (PFT)- confirms airflow limitations.

IS- used to diagnose and monitor disease progression. CT of chest- provides detailed visualization of lung parenchyma. ECG/EKG- evaluates cardiac involvement secondary to pulmonary hypertension. Echo- confirms cor pulmonale/right sided HF. Exercise tolerance test- determines oxygen therapy needs.

Contributing Risk Factors

Smoking- #1 cause; triggers inflammation, ciliary damage, and mucus hypersecretion. Environmental, exposures Genetic- alpha-1 antitrypsin deficiency (inhibits enzyme deficiency causing alveolar destruction). Chronic infections- recurrent respiratory infections accelerate lung damage. Aging- natural decline in lung elasticity and ciliary clearance.

Signs and Symptoms

Early manifestations:

- Cough- earliest symptom; chronic, intermittent, often productive; result of mucus hypersecretion and airway irritation.
- Sputum production- thick, white or yellow, copious in chronic bronchitis; indicated inflammation and impaired clearance.
- Dyspnea on exertion- initially during activity, progresses to at rest.
- Fatigue- increased work of breathing, hypoxemia.

Advanced symptoms:

- Dyspnea at rest
- Use of accessory muscles (neck, shoulders).
- Pursed lip breathing
- Prolonged expiratory phase.
- Wheezing or crackles
- Diminished breath sounds indicating air trapping (barrel chest)
- Clubbing of nails
- JVD
- Hepatomegaly- sign of cor pulmonale right sided HF secondary to pulmonary hypertension.
- Headache, confusion, drowsiness, irritability.
- Unintentional weight loss, muscle wasting, poor appetite.

Possible Therapeutic Procedures

Non-surgical

O2 therapy

Nebulizer/inhalation therapy
Bronchodilator therapy
Inhaled corticosteroids
Mucolytics- thins secretions
Chest physiotherapy/postural drainage- mobilize secretions by gravity. Position pt with up for drainage. Perform before meals to prevent nausea.
Positive expiratory pressure therapy (PEP)- keeps airways open during exhalation; often used at home with flutter valve (IS) or acapella device.
BiPAP/CPAP
Pulmonary rehab program- increase exercise tolerance and quality of life. Includes energy conservation and nutrition teaching.

Vaccinations- flu and pneumonia

Nutrition therapy

Breathing exercises
Smoking cessation programs

Surgical

Bullectomy- removes large, nonfunctional bullae (air filled spaces) that compress healthy lung tissue.

Lung volume reduction surgery (LVRS)- removes diseased, hyperinflated portions of the lung to allow remaining tissue to function better.

Lung transplant- replaces diseased lungs with donor lung.

Endobronchial valve therapy- one way valves placed in diseased lung segments to reduce hyperinflation.

Prevention of Complications

(What are some potential complications associated with this disease process)

NI to prevent complication:

Airway clearance:

- encourage hydration, teach effective coughing, use chest physiotherapy, postural drainage, and PEP devices.

Infection prevention

- annual flu and pneumonia vaccines; **hand hygiene**; promptly report sputum color change or fever

oxygen safety

- **use prescribed flow**; avoid open flames or smoking near O2; **humidify oxygen to prevent mucosal drying**.

Activity and energy conservation

- teach pursed lip breathing and diaphragmatic breathing. Plan rest periods between activities. **Encourage pulmonary rehab**.

Monitor for Cor Pulmonale

- monitor for edema, JVD, weight gain, fatigue.

Avoid respiratory depressants

- use sedatives or opioids cautiously.

Medication adherence

- reinforce importance of consistent inhaler use; educate on inhaler technique and oral care after steroids.

Complications:

Acute exacerbation of COPD
Respiratory failure- confusion, somnolence, cyanosis.

Pneumonia

Cor pulmonale right sided HF
pneumothorax/Bullae rupture- lung collapse

Polycythemia- chronic hypoxemia, risk of clots
Osteoporosis- long term corticosteroid use and immobility.

Depression and anxiety

Parenteral Therapies

Anticipated Medication Management

Short acting bronchodilators-
albuterol rescue inhaler

Long acting bronchodilators- long
term therapy not for acute attacks.

Anticholinergics-

Inhaled corticosteroids

Combination inhalers

Systemic corticosteroids- prednisone
(short course for exacerbations)

Mucolytics

Antibiotics

Oxygen therapy

Vaccines

Non-Pharmacologic Care Measures

Positive expiratory pressure therapy
(PEP)- keeps airways open during
exhalation; often used at home with
flutter valve (IS) or acapella device.

BiPAP/CPAP

Pulmonary rehab program- increase
exercise tolerance and quality of life.

Includes energy conservation and
nutrition teaching.

Breathing exercises

Smoking cessation programs

Care Needs

What stressors might a patient with this diagnosis be experiencing?

Physical- dyspnea, fatigue, activity
intolerance, sleep disturbance, fear
of suffocation.

Emotional/psychological- anxiety,
depression, frustration from loss of
independence, fear of disease
progression.

Social- isolation due to limited
mobility of oxygen use in public

Financial- cost of meds, O2,
equipment, hospital stays, pcp.

Role/lifestyle- loss of work ability,
dependence on caregiver, altered
self image.

Client/Family Education

List 3 potential teaching topics/areas

- medication and device use

- breathing and energy conservation- pursed lip and
diaphragmatic breathing, spacing activities, positioning,
when to call pcp.

- resources like case management, pulmonary PT/RT,
therapy, caregiver support groups.

NCLEX I (1): Safe and Effective Care Environment

Multidisciplinary Team Involvement

(Which other disciplines do you expect to share in the care of this patient)

PCP

Pulmonologist

RT

Case management

PT/OT

Pharmacist

Mental health

Home health nurse

nutritionist