



inflates in the correct amount of exercise O₂ to balance out V / CO₂

Anticipated Diagnostics
 Labs
 → ABG → Identify severity of exacerbation
 → chest x-ray
 → Spirometry

Additional Diagnostics
 HF assessment
 ECG can show R HF
 6 min walk
 clinical COPD questionnaire

Prevention of Complications
 (What are some potential complications associated with this disease process?)
 → Pulmonary hypertension
 → acute exacerbation
 → Acute respiratory failure

Psychosocial/Holistic Care Needs

Factors that might affect a patient with COPD diagnosis be experiencing?
 from smoking or intolerance being able to do by previously could

Environment Involvement
 (What are some environmental factors that are in the care of this patient?)

Propose is to capture foreign in the airway
 COPD usually → bacterial

Mucus → cause ↓ passage way surface to & from the lungs
 Sputum → Goblet cells produce → over production & hypersecretion causes it in COPD pt

Pulmonary Hypertension → constriction of pulmonary vessels from alveolar hypoxia
 → cause → dyspnea on exertion (chest pain), fatigue, dizziness
 → ↑ the workload of the R ventricle & causes right, SOB
 ventricular hypertrophy, eventually HF

→ Pulmonary function test → measure lung volume & air flow
 ↳ Pt. Blows into a spirometry → Deep breath, exhale as fast & hard as possible

Cor Pulmonale
 → Enlargement of the R ventricle → once developed, prognosis worsens
 → Most common symptom Dyspnea → lung sounds normal, or crackles in bases
 → Results from pulmonary hypertension → heart sound S₂ & S₄, & systolic murmur

Acute Exacerbation
 ↳ signaled by sudden change in pt. usual dyspnea, cough, or sputum
 ↳ ↑ in frequency as disease progresses

Acute Respiratory Failure → Oxygenation, ventilation are inadequate
 ↳ Pt w/ severe COPD w/ severe exacerbation
 → NOT a disease, a symptom that reflects insufficient lung function
 → Not enough O₂ transferred to blood, or inadequate CO₂ removal

Exacerbation → caused by a ~~viral~~ bacterial infection in lungs or airway

Chest x-ray → show flattening of diaphragm
 ↳ ABG → Identify the severity of exacerbation by assessing abnormal

Oxygenation
 ↳ ECG → can show signs of R HF
 ↳ Serum α₁-antitrypsin levels = Blood test, measures a protein that protects the lungs from damage

Student Name: Destiny Klinzer
 Medical Diagnosis/Disease: COPD Exacerbation (Bronchitis)

Alveoli over inflated & not taking in the correct amount of O₂ to balance out V / CO₂
 Pt. become w/ exercise ↓ resp. capacity

Inability to exhale air main characteristic of COPD

NCLEX IV (8): Physiological Integrity/Physiological Adaptation

Anatomy and Physiology
Normal Structures
 - Respiratory tract consist of Mouth/nose (Pull air into body), Pharynx (delivers air to trachea), Trachea (connects throat to lungs), Bronchial tubes (bottom of windpipe connect each lung), Bronchioles (small branch bronchial tube lead to alveoli), Alveoli (where gas exchange happens), Capillaries (blood vessels)
 - Diaphragm contract → inhale, relax → exhale

Pathophysiology of Disease
 - The loss of elastic recoil & airflow obstruction, from mucus hypersecretion, mucosal edema, & bronchospasms
 - Inflammation caused by the oxidants made by cigarette smoke & other inhaled particles
 - Oxidants affect the lungs as they inactivate anti-proteases (prevent natural destruction of lungs)
 - The main site of airflow limitation is in sm. airways
 - As peripheral airway becomes obstructed air is progressively trapped during expiration
 - As air is trapped, chest hyper-expands & becomes barrel shaped b/c resp. muscles can't effectively function

NCLEX IV (7): Reduction of Risk

Anticipated Diagnostics
Labs → Identify severity of exacerbation
 → ABG
 → chest X-RAY
 → Spirometry
Additional Diagnostics
 H&P assessment
 ECG scan show R HF
 6 min-walk
 clinical COPD questionnaire

- Pleura - separate lungs from chest wall (neg. pressure)

- Functional residual capacity is less residual air & ↓ elastic recoil, makes passive expiration of air difficult
 Pt inhale when in over-inflated

NCLEX II (3): Health Promotion and Maintenance

Contributing Risk Factors
 → cigarette smoking
 → Infection
 → Asthma
 → Air Pollution
 → Occupational chemicals
 → Dusts
 → Alpha
 → Genetics
 → Alpha-1 Antitrypsin Deficiency = Autosomal recessive disorder

Signs and Symptoms
 - Chronic intermittent cough
 - Dyspnea progressive (exercise)
 - Report chest heaviness
 - Is not able to take deep breath
 - Chest tightness
 - Fatigue, weight loss, anorexia
 - ↓ breath sounds, wheeze in all lung fields
 - Sputum color & amount not normal

Possible Therapeutic Procedures
Non-surgical O₂ therapy
 Nutrition therapy → extra protein & calories
Surgical lung volume reduction → Reduce lung size, remove diseased lung tissue.
 Bronchoscopic lung volume reduction
 Allow air to leave lung

NCLEX IV (7): Reduction of Risk

Prevention of Complications
 (What are some potential complications associated with this disease process)
 → Pulmonary hypertension
 → Poor Pulmonal
 → Acute exacerbation
 → Acute respiratory failure

NCLEX IV (6): Pharmacological and Parenteral Therapies

Anticipated Medication Management
 → Bronchodilators
 → Diuretics
 → Antibiotics
 → Oral systemic corticosteroids

NCLEX IV (5): Basic Care and Comfort

Non-Pharmacologic Care Measures
 → Place pt in high Fowler's for optimal resp. function
 → Promote adequate rest qom
 → Adequate nutrition + small frequent meals
 → Administer O₂ therapy

NCLEX III (4): Psychosocial/Holistic Care Needs

What stressors might a patient with this diagnosis be experiencing?
 - Guilt from smoking
 - Loss of independence
 - Not being able to do what they previously could

Client/Family Education

List 3 potential teaching topics/areas
 • Deep breathing & airway clearance exercise
 • Energy conservation techniques
 • Teach about meds & the set schedule the pt. will most likely follow

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)
 → RT
 → Dietitian
 → Physical therapy
 → Social Worker