

Northwest Community EMS System
Paramedic Education Program
CHRONIC RESPIRATORY DISORDERS
Connie J. Mattera, M.S., R.N., EMT-P

Reading assignment:

Aehlert Vol. 1 pp, 423-427

SOP: COPD/Asthma

Drugs to know: albuterol; epinephrine 1:1000; ipratropium, magnesium

Procedure manual:

MDI, Nebulizer therapy: mouth piece/ mask/in-line w/ BVM & C-PAP

OBJECTIVES:

Upon reading the textbook and completion of the class, homework questions, and labs, each participant will independently do the following with at least an 80% degree of accuracy and without critical error:

1. Discuss the epidemiology of pulmonary diseases and conditions.
2. Compare and contrast intrinsic vs. extrinsic risk factors for pulmonary diseases.
3. Cite examples of respiratory problems stemming from ventilation, perfusion, or diffusion dysfunction.
4. Generalize the characteristics of a chronic respiratory disease.
4. Explain the characteristics of conditions classified as chronic obstructive pulmonary disease (COPD).
5. Compare and contrast the pathophysiology of emphysema and chronic bronchitis.
6. Discuss abnormal assessment findings associated with chronic pulmonary diseases and conditions.
7. Sequence the pathogenesis of Cor Pulmonale.
8. Sequence the prehospital interventions for COPD with respiratory/ventilatory distress providing rationales for each intervention.
9. Explain the pharmacological preparations that paramedics use for the management of chronic respiratory diseases including albuterol, ipratropium epinephrine, and magnesium.
10. Discuss the pharmacological preparations used in managing patients with chronic respiratory diseases that may be prescribed by physicians.

Affective:

11. Value the need to conduct a complete assessment to detect chronic respiratory conditions and treat patients according to SOP.
12. Defend each patient's right of autonomy to consent or withhold consent for EMS care.

Psychomotor:

13. Obtain a history and perform a complete patient assessment for patients with chronic pulmonary diseases and conditions.
24. demonstrate the correct use of a hand held nebulizer, in-line nebulizer, and CPAP mask.

NWC EMSS Paramedic Education Program
CHRONIC OBSTRUCTIVE PULMONARY DISEASES (COPD)

Connie J. Mattera, M.S., R.N., EMT-P

I. **Introduction**

A. **Epidemiology**

1. **Incidence:** Respiratory complaints constitute a major number of EMS calls. It is estimated that up to 20% of the adults in the US have COPD or asthma.
2. **Morbidity/mortality:** Over 200,000 persons die from respiratory emergencies each year.
3. **Risk factors** that increase the development of pulmonary disease
 - a. **Intrinsic factors**
 - (1) **Genetic predisposition**
 - (a) Asthma: allergies
 - (b) COPD (congenital enzyme deficiency of alpha₁-antitrypsin)
 - (c) Carcinomas
 - (2) Associated cardiac or circulatory pathologies
 - (a) Pulmonary edema
 - (b) Pulmonary emboli
 - (3) **Stress**
 - (a) Increases the severity of respiratory complaints
 - (b) May be associated with the frequency of exacerbations of asthma and COPD
 - b. **Extrinsic factors**
 - (1) **Smoking**
 - (a) Most common cause: Increases the prevalence of COPD and carcinomas
 - (b) Increases the severity of all respiratory diseases
 - (2) **Environmental pollutants**
 - (a) May increase the prevalence of COPD
 - (b) Increases the severity of all obstructive disorders

B. **Rational approach to pulmonary pathophysiology**

1. There are many, many pulmonary diseases. Many diseases act in a variety of different ways on a number of body systems.
2. Learning the pathophysiology of every respiratory disease is impossible at the paramedic level and is not a useful exercise because of the dynamic nature of newly developing or identified pulmonary pathologies.
3. However, all respiratory problems, old or new, can be categorized as impacting **ventilation, diffusion, or perfusion** and usually present with **dyspnea** as one of the chief complaints.
4. Supportive treatment should be initiated rapidly and effectively once the problem has been identified as stemming from ventilation, diffusion, perfusion or a combination of factors.

C. **Ventilatory dysfunction** can originate in any of the components of the system.

1. **CNS :** Trauma, stroke or other medical neurological condition, CNS depressant drugs
2. **Spinal cord:** High C-spine injury

Chronic Respiratory Disorders

- 3. Nerves: Polio, Guillain-Barré syndrome, multiple sclerosis
- 4. Muscles: Myasthenia Gravis, muscular dystrophy
- 5. Airways:
 - Nasopharynx: congestion, trauma, inflammation of adenoids
 - Oropharynx: tongue, inflammation of the tonsils
 - Larynx: laryngospasm, epiglottitis, croup
 - Trachea: edema, F/B
 - Bronchioles: Smooth muscle spasm, inflammation or edema (asthma, acute bronchitis, chronic bronchitis, bronchiolitis), mucous accumulation (cystic fibrosis, asthma)
 - Chest wall impairment: Trauma
 - Hemothorax, pneumothorax
 - Empyema
 - Pleural inflammation

D. Diffusion

- 1. Atmospheric deficiency
- 2. Alveolar pathology
 - a. Asbestosis, other environmental lung diseases
 - b. Blebs/bullae associated with COPD
 - c. Inhalation injuries
- 3. Interstitial space pathology
 - a. High pressure (also known as cardiogenic)
 - (1) Left heart failure
 - (2) Idiopathic pulmonary hypertension
 - b. High permeability
 - (1) ARDS
 - (2) Asbestosis, environmental lung disease
 - (3) Near-drowning
 - (4) Post-hypoxia
 - (5) Inhalation injuries
- 4. Capillary bed pathology: Severe atherosclerosis

E. Perfusion

- 1. Inadequate blood volume/hemoglobin levels
 - a. Hypovolemia
 - b. Anemia
- 2. Impaired circulatory blood flow: pulmonary embolus
- 3. Capillary wall pathology: trauma; pulmonary contusion

F. This presentation will focus on dysfunction in the airways, with ventilation, diffusion, and/or perfusion.

II. Acute vs. chronic obstructive airway diseases

- A. Respiratory diseases can be classified according to the nature of their onset as either acute or chronic. Acute refers to those that have a rapid onset and short duration. Chronic generally refers to those that have a slow onset and persist over time.
- B. The acute and chronic obstructive diseases differ in that the lung tissues do not return to normal between exacerbations in chronic lung disease. Instead, the pulmonary damage is a slowly progressive process.
- C. **Definitions and general characteristics of COPD**
 - 1. Chronic Obstructive Pulmonary Disease (COPD) or Chronic Obstructive Lung Disease (COLD) is a collection of diseases that include emphysema, chronic bronchitis, asthma, bronchiectasis and cystic fibrosis.

Chronic Respiratory Disorders

2. Although it is common to call all patients with COPD "chronic lungers", for physiologic, pathologic, and clinical reasons it is still preferable to divide them into those with emphysema and those with chronic bronchitis. Most patients usually have a combination of both diseases.
 3. Obstructive airway diseases are those in which there is a diffuse obstruction to airflow within the lungs primarily in the bronchioles. Obstruction may be reversible or irreversible.
 4. Since the power to inspire or inhale gas is provided by the muscles of ventilation (diaphragm and intercostals) and the bronchioles naturally dilate on inspiration, it is much easier to overcome airway resistance during inhalation than during passive exhalation. The bronchioles naturally constrict on expiration.
 5. Therefore, these diseases **share the common characteristic of increased airway resistance on exhalation resulting in obstruction to expiratory airflow** with significant air trapping (CO_2) distal to the obstruction.
 6. Exacerbations (worsening of the condition) and remissions commonly occur.
- D. **Incidence:** very high in the U.S. affecting 10%-25% of the adult population.
- E. **Morbidity and mortality:** 50% mortality within 10 years of the diagnosis
- F. **Causes:** COPD is rarely seen in non-smokers. When combined with environmental exposures, serious lung disease can occur at early ages.

Other factors have been shown to precipitate symptoms in those who already have COPD

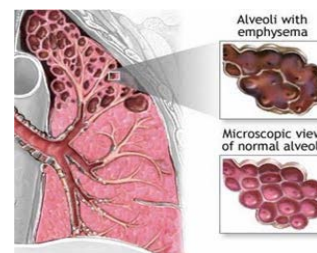
1. **Intrinsic factors**
 - a. Stress
 - b. Upper respiratory infections
 - c. Exercise
2. **Extrinsic factors**
 - a. Tobacco smoke
 - b. Drugs
 - c. Occupational hazards (chemical fumes, dust, etc.)
 - d. Allergens such as foods, animal dander, dust and molds.

III. Pulmonary emphysema

- A. **General facts:** Comes from Greek root word "*emphysan*" meaning to inflate. Most common form of COPD with over 3 million persons affected.

B. **Pathophysiology**

1. Emphysema results from a process of gradual and inconsistent weakening and **destruction of the walls of the terminal bronchioles and alveoli**. It has a major effect on lung compliance (ease of distensibility) and elasticity. When the alveoli and small bronchioles are destroyed, the elasticity and fiber network (connective tissue structure) of the alveoli is broken down. As fibrous and muscle tissues are lost, they become **more compliant and distensible**. Alveolar walls enlarge and merge together into large **blebs** or **bullae**. These greatly reduce the alveolar membrane surface area for diffusion causing an **altered ventilation/perfusion ratio**.
2. The blebs collapse more easily than normal lung tissue (especially on expiration). **CO_2 is trapped** in passages distal to the obstruction (**hypercapnia**) causing chronic **lung hyperinflation**. Residual volume increases while vital capacity remains relatively normal. Even in severe disease, inspiratory airway resistance tends to be normal.



3. Additional physiological causes of obstruction include inflammation, infection, secretions, muscular constriction or combinations. While there may be airway inflammation and some increased mucus production, many patients with emphysema produce little or no sputum.
4. The number of pulmonary capillaries is decreased, increasing resistance to pulmonary blood flow. This ultimately causes **pulmonary hypertension**, which may lead to primary right heart failure, **cor pulmonale** (rare, unless associated w/ chronic bronchitis), and death. *Cor Pulmonale heralds a poor prognosis.*
5. As the disease progresses, oxygen levels decrease which stimulates excess RBC production (**polycythemia**) and an abnormally high hematocrit. Patients maintain better tissue oxygenation than those with chronic bronchitis due to the polycythemia and don't appear chronically cyanotic ("**Pink puffer**").
6. **Two major types**
 - a. **Vesicular emphysema** involves the portion of the lung distal to the terminal bronchiole (acinus) where gas exchange takes place. This involves permanent, abnormal enlargement of the acinus with associated destructive changes.
 - b. **Interlobular or interstitial emphysema**: Affects tissues between alveoli.

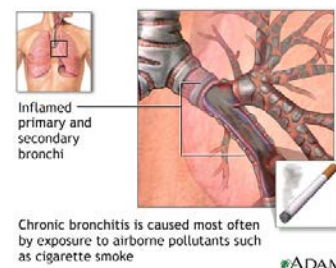


IV. **Chronic bronchitis**

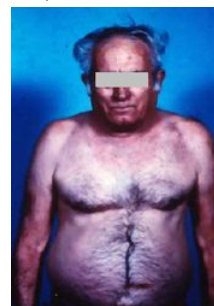
- A. **Etiology:** Typically self-induced by cigarette smoking and/or exposure to other environmental pollutants which may lead to major changes in the respiratory epithelium, bronchial mucosa and goblet cells. Patients start developing respiratory problems, i.e., numerous respiratory tract infections in their 40s.

B. **Pathophysiology**

1. Chronic irritation and inflammation of the entire tracheobronchial tree cause an **increase in the number and size of mucus-secreting cells and an excessive production of mucus** for most days of the month for at least 3 or more consecutive months for 2 or more consecutive years.
2. Patients produce at least 10 mL green or yellow sputum daily. Productive cough and dyspnea increase with disease severity. However, the **cilia are damaged causing decreased removal of mucus**. These secretions cause **airway obstruction from mucus plugging** and provide an excellent growth medium for microorganisms.
3. Thickening and rigidity of the bronchial mucosa results from vasodilation, congestion and edema. This increase in resistance can be with or without emphysemic changes. Unlike emphysema, the alveolar walls are not severely affected in chronic bronchitis and diffusion across the respiratory membrane remains normal.
4. It is the alveolar hypoventilation that adversely affects respiratory gas exchange leading to arterial hypoxia and CO₂ retention (hypercarbia) sometimes giving the patient a bluish complexion. The increased PaCO₂ may lead to irritability, somnolence, decreased intellectual abilities, headaches, and personality changes.
5. **Pathogenesis of Cor Pulmonale (primary right heart failure)**
 - a. Bronchial obstruction leads to ↓ alveolar oxygen and hypoxia. Hypoxia, acidosis and hypercarbia cause vasoconstriction of small, mid-sized arteries linking the right heart and lungs (hypoxic vasoconstriction) resulting in **pulmonary hypertension**.



- b. This generates an \uparrow cardiac workload for the right ventricle (\uparrow RV afterload) resulting in RV hypertrophy and failure. If the RV does not send blood forward to the lungs, blood cannot flow into it from the peripheral veins resulting in systemic venous congestion (JVD), hypertension, and peripheral edema ("Blue Bloater").
6. Over time, arterial hypoxia causes \uparrow RBC production (polycythemia) with \uparrow O₂ carrying capacity.
7. Patients more likely than those with emphysema to adjust to chronically elevated CO₂ levels by switching to an oxygen-dependent ventilatory drive.



C. Assessment

Emphysema	Chronic bronchitis
Chief complaint: Dyspnea on exertion (early stages); at rest (late). Severe exercise intolerance due to dyspnea. The patient's subjective feeling of distress may be the most accurate indication of their illness severity as the other S&S may be always present. Weight loss and chronic fatigue from increased energy demands for respiration.	Copious production of sputum; usually most plentiful during the morning, but may continue all day. Early disease: Dyspnea on exertion (DOE) due to airflow obstruction and cough with frequent infections. Advanced disease: Dyspnea at rest and continuous cough; shortness of breath (SOB). Severe exercise intolerance.
PMH: Ask about cigarette and tobacco use. Reported in pack/years. Ask the number of packs smoked per day and the number of years they have smoked. Multiply the two numbers. Medical problems related to smoking such as emphysema, chronic bronchitis and lung cancer usually begin after patient surpasses a 20 pack/year history, although that can vary significantly.	
Vital signs <ul style="list-style-type: none"> BP: Usually WNL; may progress to pulsus paradoxus P: Tachycardia; ECG: dysrhythmias; tall peaked P waves: "P Pulmonale" pattern RR: Tachypnea 1-2 word dyspnea is normal; respiratory effort is markedly increased using accessory muscles. Prolonged expiratory cycle with pursed lip breathing. This creates positive pressure similar to PEEP which prevents collapse of the lower airways by elevating intra-airway pressures allowing more gas exchange to occur and facilitates exhalation of CO₂. SpO₂: Depends on state of disease; often decreased. Chronic hypoxemia is often severe in chronic bronchitis. pCO₂: Hypercapnia is common to moderate degree. Hypercapnia results from hypoventilation with ventilation/perfusion mismatch. Increased carbon dioxide levels lead to \uparrow ventilation. Ventilatory drives: When hypercapnia becomes chronic, the central medullary chemoreceptors are depressed and the peripheral chemoreceptors on the aortic arch and carotid bodies that are sensitive to \downarrow O₂ levels may take over the ventilatory drive function. Only a small percentage switches to an oxygen dependent drive. Late in disease: Mental status changes: restlessness, impaired judgment, confusion, stupor, coma 	
HEENT: Usually normal; JVD if Cor Pulmonale Pulmonary/CV <ul style="list-style-type: none"> Barrel chest due to well developed accessory muscles and chronic hyperinflation of the lungs. Orthopnea: Progression of dyspnea. Cannot lie flat, must sit up to breathe. Charted as two pillow or three pillow orthopnea. Prolonged expiratory phase Suprasternal retractions 	

Emphysema	Chronic bronchitis
Decreased or diminished breath sounds in all lung fields unless infection present; then may or may not hear wheezes or crackles depending on the degree of air flow obstruction.	Coarse crackles due to occlusion of the larger airways with mucus plugs; wheezing.
Occasional cough with small amounts of whitish-gray mucus in the morning.	Changes in sputum color suggest purulence or respiratory infection.
GI/GU: Hepatic congestion if Cor Pulmonale. Assess for hepato-jugular reflux. Clubbing of the nail beds: The fingertip will enlarge and will lose the normal angle at the nail bed. Normal nail exits bed at a 160° angle. Clubbing causes a 180° + angle. Seen with polycythemia and chronic hypoxemia. Etiology unclear. Thought that capillaries dilate in an effort to bring oxygen to tissues. If Cor pulmonale present: peripheral edema.	
Skin: <i>Pink puffer</i>: Polycythemia causes ↑ blood viscosity = ↓ O ₂ to tissues. Cyanosis is a late and unreliable sign. Need > 5 Gm desaturated Hb or pO ₂ < 40 mmHg.	Skin: <i>Blue Bloater</i>": With Cor Pulmonale present, patient has right heart failure with peripheral fluid retention (dependent edema) and is more likely to appear cyanotic.

V. **Treatment for both emphysema and chronic bronchitis: See SOP**

- A. Goal is to maintain optimal pulmonary function by relieving hypoxia and reversing bronchospasm.
- B. **IMC** special considerations:
 1. Evaluate ventilation/oxygenation, work of breathing, accessory muscle use, degree of airway obstruction/ resistance, speech, cough, cerebral function, fatigue, hypoxia, CO₂ narcosis, and cardiac status.
 2. Obtain Hx of current meds: time and amount of last dose; duration of this attack
 3. **If wheezing without Hx of COPD/Asthma:** consider FB aspiration, pulmonary embolus, vocal cord spasm, HF/pulmonary edema. **If probable cardiac cause** (PMH: CVD): treat per Cardiac SOPs.
 4. Assess for pneumonia, atelectasis, pneumothorax or tension pneumothorax. **If tension pneumothorax** (↓ BP, absent breath sounds): Needle decompress affected side.
 5. Place in a seated or semi-Fowler's position to assist the accessory respiratory muscles.
 6. **Airway/Oxygen:** Oxygen delivery is based on SpO₂, work of breathing, and adequacy of tidal volumes.
 - a. **Mild to moderate distress:** O₂ 2-6 L/NC. May supplement with O₂ 6 L/NC if patient is hypoxic (SpO₂ < 94%) & using a HHN. If SpO₂ does not improve to 92%, switch to a NRM. Do not overcorrect their pO₂ and pCO₂ status too quickly. These have been chronic changes for them with compensatory adjustments. Overcorrection too quickly can cause major acid-base and electrolyte imbalances.
 - b. **Severe SOB, orthopnea, use of accessory muscles, speaks in syllables, tachypnea, breath sounds diminished or absent; exhausted (HR & BP may be dropping)**
 Assess need for DAI if near apnea, coma or depressed mental status, exhaustion, severe hypoxia (SpO₂ < 90); hypercapnia (EtCO₂ > 60 mmHg); hemodynamic instability, impending respiratory failure or arrest, or failure to improve with maximal initial therapy.

Prepare resuscitation equipment; anticipate rapid patient deterioration. If immediate intubation not needed:

C-PAP: Start FiO₂ at 60% with 5 cm PEEP if unit is adjustable; May increase to FiO₂ 95% and PEEP not to exceed 10 cm by SOP to achieve SpO₂ ≥ 94%. If SBP falls under 90: Remove C-PAP.

- c. If assisted ventilation/intubation required: **ventilate at 6 - 12 BPM** [slower rate, smaller tidal volume (6-8 mL/kg), shorter inspiratory time & longer expiratory time to allow complete exhalation]
“In patients with severe obstructive pulmonary disease and increased resistance to exhalation, providers should try to prevent air trapping that may result in inadvertent generation of intrinsic positive end-expiratory pressure (PEEP), so-called "auto-PEEP." In patients with hypovolemia, auto-PEEP may substantially reduce cardiac output and blood pressure. To prevent this, **use lower respiratory rates in these patients, allowing more time for complete exhalation**”.
- d. If cardiac arrest occurs, brief disconnect from BVM may be considered and compression of the chest wall to relieve air-trapping can be effective (Class IIa)
- e. Despite the fact that they may be breathing from an oxygen-dependent drive, **do not** withhold oxygen if in hypoxic distress! Hypoxic drive is not a prevailing consideration in cyanotic patients or those in ventilatory distress. Although of all persons with COPD, chronic bronchitis patients are most likely to be in this category.
- f. Monitor respirations continually and assist as needed with a BVM if depth or rate diminishes.

7. **Vascular access**

- a. **Mild distress:** No IV usually necessary
- b. **Moderate to severe distress:** Vascular access & NS titrated to maintain hemodynamic stability. If dehydration evident, provide small fluid challenge and reassess. This may also aid in loosening thick mucus secretions.
- c. **Monitor ECG:** Bradycardia signals deterioration of patient status.

C. Drug interventions

ALBUTEROL 2.5 mg & IPRATROPIUM (Atrovent) 0.5 mg via HHN or mask

- Supplement w/O₂ 6 L/NC if patient is hypoxic (SpO₂ < 94%) & using a HHN
- **Begin transport as soon as neb is started.** Do not wait for a response.
- Continue nebulizer therapy while enroute to hospital. May repeat **ALBUTEROL 2.5 mg/HHN**.

- D. They usually need supportive therapy and antibiotics at hospital as they are candidates for septic shock

VI. **Asthma** - see Asthma handout

TERMINOLOGY

APNEA	Cessation of respiration
ATELECTASIS	Collapse of alveoli from obstruction or decrease in surfactant. This prevents oxygen from being picked up by the circulating blood.
AUDIBLE/ NOISY BREATHING	Breath sounds heard without a stethoscope, indicating partially obstructed airways. Examples: Snoring (partial oropharyngeal obstruction from tongue); gurgling -obstruction from liquids as blood, vomitus; stridor (partial obstruction from FB or edema in upper airways).
BRADYPNEA	Decreased respiratory rate, less than 12/minute.
CO₂	Carbon dioxide is a byproduct of metabolism eliminated in ventilation. Fever (\uparrow metabolic rate) = \uparrow CO ₂ ; Sleep (\downarrow metabolic rate) = \downarrow CO ₂ .
COMPLIANCE	The ease of distensibility or resistance of the lung and chest wall.
CLUBBING	Broadening and thickening of the soft tissues of the ends of fingers or toes. Frequently seen in COPD patients.
COR PULMONALE	Primary failure of the R ventricle due to \uparrow pulmonary artery pressures resulting from lung disease (COPD). Signs: JVD, dependent edema.
CYANOSIS	Bluish discoloration of skin due to \uparrow carboxyhemoglobin in blood. Late sign of hypoxia. See hypoxia.
DEAD SPACE	Volume of air in conducting airways; space is that ventilated/not perfused (wasted ventilation). Ave. adult = 150 ml (1 cc/kg).
DIFFUSION	Transport of particles from area of higher concentration across a semipermeable membrane to area of lower concentration.
DYSPNEA	Difficult or labored breathing usually associated with serious disease of the heart or lung.
EXPECTORATION	Coughing up sputum.
FiO₂	Percentage of oxygen in inspired air. Room air FiO ₂ = 21%.
HEMOPTYSIS	Blood in sputum due to bronchitis, pulmonary embolism, TB and lung cancer.
HYPERCAPNIA/ HYPERCARBIA	Increased CO ₂ in arterial blood.
HYPERPNEA	Deep inhalation.
HYPOXEMIA	Decreased oxygen in blood.
HYPOXIA	Insufficient oxygen to meet tissue needs.

Comments on hypoxia & cyanosis

Cyanosis is a late and unreliable sign of hypoxemia. It takes five grams of desaturated hemoglobin to produce cyanosis. Normal oxygen levels are 95 mmHg or above on room air.

The human brain accounts for about 20% of total oxygen consumption is very sensitive to hypoxia. Mental status is a more sensitive and earlier indicator of hypoxia than cyanosis.

<u>Symptoms</u>		<u>Signs</u>	<u>\uparrow CO₂ Effects</u>
Dyspnea	\uparrow RR	\uparrow RR	
Restlessness		\uparrow HR	Headache
Impaired judgment		\downarrow HR late	Tremor
Personality change		Arrhythmias	Diaphoresis
Headache		\uparrow BP	Asterixis: impaired motor function
		\downarrow BP late	Delirium
		Central cyanosis late	
Confusion		Heart, kidney failure	Seizures, coma

HYPOVENTILATION	Decreased rate and depth of breathing, resulting in increased CO ₂ levels causing respiratory acidosis.
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ORTHOPNEA	Difficulty breathing except in an upright position. Charted as the number of pillows the patient uses in order to sleep.
PERFUSION	Blood flow to tissues. Is affected by pump, vessel, and volume factors.
PLEURISY	Inflammation of the pleurae, frequently caused by pneumonia, TB, tumors or trauma. Causes pleuritic chest pain and pleural friction rubs.
PLEURITIC CHEST PAIN	Chest pain which occurs with or increases with respiration. Often occurs with conditions as pulmonary embolus, pneumothorax, pneumonia and pleurisy.
SINGULTUS	A hiccup. Sudden inhalation due to spasmodic contraction of diaphragm cut short by glottic closure.
TACHYPNEA	Increased respiratory rate, greater than 20/minute.
ALVEOLAR VENTILATION	Air flow to alveoli with each breath. Decreases can be from: obstruction by secretions (bronchitis/pneumonia); obstruction from muscle spasm (asthma); obstruction from collapse of alveoli (atelectasis); obstruction from fluid filled airways (pulmonary edema). Calculated by measuring tidal volume and subtracting the dead space.
TRACHEAL TUGGING	Thyroid cartilage is pulled upward during labored inhalation.
VOLUMES - AVERAGE ADULT	<p>Total Lung Capacity (TLC) = $\pm 6,000 - 7000$ cc Vital capacity + residual volume.</p> <p>Tidal Volume (V_T) = ± 500 cc Volume of air inspired or expired with each normal breath.</p> <p>Vital Capacity (VC): 5000 cc Maximal inspiration followed by a maximal expiration. Exhaled volume is VC.</p> <p>Inspiratory Reserve Volume (IRV) = $\pm 1,200$ cc Maximum amount of air one can inspire after inspiration of TV.</p> <p>Functional Residual Capacity (FRC): = $\pm 2000 - 3000$ cc Gas remaining in lung after normal expiration.</p> <p>Residual Volume (RV) = $\pm 1,200$ cc Volume of air left in lungs after maximum expiration.</p> <p>Minute Volume - amount of gas leaving the lungs per minute. Determined by multiplying the tidal volume by respiratory rate.</p> <p>Ex: $500 \text{ cc } (V_T) \times 15 \text{ (RR)} = 7,500 \text{ cc/minute} = \text{Minute volume}$</p> <p>The volume will \uparrow or \downarrow if either changes. The patient in respiratory distress may \uparrow his rate, but often the breaths will be more shallow - resulting in a \downarrow minute volume.</p> <p>Ex: $200 \text{ cc } (V_T) \times 22 \text{ (RR)} = 4,400 \text{ cc/minute} = \text{Minute volume}$</p> <p>$\text{CO}_2$ elimination is directly related to the minute volume. The higher the minute volume, the more CO_2 is eliminated.</p>

Chronic Respiratory Disorders**Chronic Respiratory Homework questions**

1. Chronic obstructive pulmonary diseases share the common characteristic of increased airway resistance resulting in obstruction to inspiratory / expiratory (circle one) airflow.
2. What is the primary cause of most COPD diseases?
 - A. Allergies
 - B. Cigarette smoking
 - C. Genetic predisposition
 - D. Ineffective cardiac output
3. Which IS NOT a common obstructive lung disease?
 - A. Asthma
 - B. Pneumonia
 - C. Emphysema
 - D. Chronic bronchitis
4. Which COPD condition is characterized by destruction of terminal bronchioles and alveolar walls?
 - A. Asthma
 - B. Pneumonia
 - C. Emphysema
 - D. Chronic bronchitis
5. Which patient with COPD is known as the "pink puffer" because they are not usually cyanotic?

6. What compensatory mechanism allows these patients to stay "pink"?

7. What is an overdistended, destroyed alveolus called? _____
8. When the distal lung segments collapse with air trapping, what gas is trapped?
 - A. N₂
 - B. O₂
 - C. CO₂
 - D. CO
9. Air trapping leads to an increase of this gas in the body called: _____
10. The air trapping leads to lung _____
11. Patients with breathing difficulty tend to
 - A. prefer a supine position.
 - B. display cold extremities.
 - C. assume a "tripod" position.
 - D. become hypotensive early in the disease.
12. Why do COPD patients breathe through pursed lips?

What oxygen delivery device can be applied to mimic the same physiological function as pursed lip breathing?

Chronic Respiratory Disorders

13. What change occurs to the anterior-posterior chest diameter in COPD?

Why does this happen?

14. What lung sound changes should be normally anticipated in a patient with emphysema?

What change in lung sounds is likely if there is increased bronchoconstriction due to an infection?

15. Which physical finding indicates the presence of chronic hypoxemia when looking at the patient's extremities?

16. Which patient with COPD is known as the "blue bloater" because they are chronically hypoxic and often develop primary right heart failure?

17. Which chronic condition results from an increase in the number of mucus-secreting cells in the respiratory tree and is usually associated with a productive cough and copious sputum production for three consecutive months over two or more consecutive years?

- A. Asthma
- B. Emphysema
- C. Chronic bronchitis
- D. Pulmonary embolism

18. Are alveolar walls severely affected in chronic bronchitis? Yes / No

19. Is diffusion across the respiratory membrane normal or abnormal in chronic bronchitis? Why or why not?

20. Why is a patient with chronic bronchitis often hypoxic?

21. What is the name of the primary right heart failure that often occurs in patients with chronic bronchitis?

22. What pathophysiologic change in the lungs causes the primary right heart failure?

23. What clinical S&S should lead a-PM to suspect primary right heart failure?

24. What is orthopnea?

25. What lung sounds should be anticipated in a patient with chronic bronchitis?

26. An adult with a chronic respiratory condition is c/o difficulty breathing. What is usually the most reliable indicator of the severity of the patient's present condition?

- A. Tachycardia
- B. Pallor and diaphoresis
- C. One or two word dyspnea
- D. Patient's description of severity

27. Which clinical finding is present in chronic bronchitis but usually absent in emphysema and asthma?

- A. Cough
- B. Wheezing
- C. Resistance to airflow
- D. Daily excessive mucus production

28. When is a nasal cannula indicated for oxygen administration in a patient with COPD in mild distress?

29. If a patient with COPD presents with spontaneous ventilations with good effort, severe respiratory distress, an SpO₂ reading of 86% and wheezing, what oxygen delivery device and liter flow should be applied first?

If a high FiO₂ is administered, what might happen to the patient's ventilatory drive?

What signs should be assessed to determine if this is happening?

What action should a paramedic take if ventilatory failure becomes evident?

30. What drugs (name, dose, route, timing) should be given to patients with emphysema or chronic bronchitis if they are wheezing with mild to moderate respiratory distress?
