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June 2012 "Chronic Obstructive Pulmonary Disease"

THIS MONTH
June 2012
"COPD"

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COPD is an often misunderstood term. In this lesson we review the concept and discuss methods of treatment.

This lesson provides 1.25 hours (0.125 CEUs) of credit, and is intended for pharmacists in all practice settings. **The program ID # for this lesson is 707-000-12-006-H01-P. Pharmacists completing this lesson by June 30, 2015 may receive full credit.**

To obtain continuing education credit for this lesson, you must answer the questions on the quiz (70% correct required), and return the quiz. Should you score less than 70%, you will be asked to repeat the quiz. Computerized records are maintained for each participant.

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The objectives of this lesson are such that upon completion the participant will be able to:

1. List the various parts of the respiratory tract and their functions.
2. Describe the symptoms of COPD.
3. Differentiate between chronic bronchitis, emphysema and bronchial asthma.
4. Discuss the medications used in treating COPD.
5. Discuss the prevalence, causes and prognosis of COPD.

REVIEW OF ANATOMY & PHYSIOLOGY OF THE RESPIRATORY SYSTEM

Respiration is the act of inhaling and exhaling during which oxygen from air is taken to be used in body tissue and carbon dioxide is given off. The lungs are the primary organs of the respiratory system. They are located in the thorax. The respiratory system includes all organs that facilitate breathing, including: the pharynx, larynx, trachea, bronchi, bronchioles, alveoli and the lungs. The main function of the respiratory tract is to act as the place where passive gas exchange takes place between the circulatory system and atmospheric air. Oxygen is absorbed from the inhaled air and diffuses into the circulation. Subsequently it flushes out carbon dioxide from the body via the exhaled air. The inhaled air enters the upper respiratory tract (nasal cavity, pharynx and larynx), then passes through the lower respiratory tract (trachea, bronchi, bronchioles, alveoli). Inhalation occurs when the external intercostal muscles contract causing the thorax to expand. Simultaneously the diaphragm moves down. This movement creates a negative pressure within the thorax as well as within the lungs. Inhalation is followed by expiration. The mechanics are the opposite of inhalation.

The respiratory tract consists of three parts: the upper respiratory tract (URT), respiratory passages and the lungs. **The upper respiratory tract** includes: the nasal cavity, pharynx and larynx. The nasal cavity is a space located behind the nose forming the roof of the mouth. It is the first part of the URT that receives the inhaled air. Due to its large surface area and its abundance of capillaries the nasal cavity warms up the entering air in order to be received in proper condition by the lower respiratory tract. Furthermore, the mucus that covers the inside walls tends to humidify the air and trap dust and other foreign particles which eventually are pushed up by the cilia of the epithelium toward the pharynx and into the esophagus where it may be swallowed.

The pharynx is a musculomembranous tube that is considered a part of the throat located behind the mouth and the nasal cavity and above the larynx where it connects with the esophagus. It serves as a passageway for air flowing from the nasal cavity to the larynx and food from the mouth to the esophagus.

The larynx is the enlarged upper end of the trachea, commonly known as the voicebox, which plays a role in breathing, sound production and prevention of food from entering the trachea. **The respiratory passageways consist of the trachea, main bronchi, bronchioles, and alveolar ducts and sacs**.

The trachea (windpipe) is a cylindrical cartilaginous tube that connects the larynx with the lungs. It is lined with a mucus membrane, ciliated epithelium and goblet cells that release mucus which traps foreign particles that ultimately reach the larynx. These are either swallowed or are expectorated as phlegm. The trachea contains about 15 to 20 C-shaped cartilaginous rings to reinforce its walls and prevent collapse. The epiglottis is a flap located at the entrance of the larynx and is made of an elastic cartilaginous membrane. During the swallowing process, the glottis closes the opening of the larynx to prevent the swallowed materials from passing into the trachea (choking).

The bronchi are cartilaginous tubes made & covered with mucosa and are located at the left and right of the lower end of the trachea. Both penetrate the lungs and eventually branch out into smaller tubes known as bronchioles.

The bronchioles contain no cartilaginous tissues or glands. They constitute the last division of the bronchial tree. They terminate into the alveolar ducts that lead to the alveolar sacs (alveoli).

The alveoli are tiny sacs that form the dead-end of the bronchial tree and are the place where gas exchange with the blood takes place. The average adult lung contains 300 – 400 million alveoli. Oxygen reaches the alveoli and diffuses through the capillaries into arterial blood. The waste rich blood from the veins releases carbon dioxide into the alveoli, and it is ultimately exhaled. This gas exchange that takes place in the alveoli occurs simultaneously. The alveoli are made of collagen and elastin allowing them to stretch and spring back during the breathing process. The huge surface area of these sacs makes gas exchange possible. The diaphragm assists in getting rid of carbon dioxide from the lungs through exhalation.

The lungs are the main organ of the respiratory tract and are housed in the thoracic cavity. They are soft, elastic and spongy and are protected from physical damage by the ribcage. There are a number of ways

CE PRN® (ISSN 0199-5006) is owned and published by W-F Professional Associates, Inc. 400 Lake Cook Road, Suite 207, Deerfield, Illinois 60015. William J. Feinberg, President. *CE PRN*® is published eleven times per year, monthly, January through November. Subscription rate is $110.00 per year. Second-Class Postage paid at Deerfield, Illinois 60015 and at additional mailing offices. *POSTMASTER: Send all address changes to W-F Professional Associates, Inc., 400 Lake Cook Road, Suite 207, Deerfield, IL 60015.*
June 2012

by which the lungs are protected from irritants and foreign particles. The nose traps relatively large particles of pollutants from entering the lungs. The mucus (sputum) that lines the bronchial mucosa also eliminates smaller particles by adhering to them. The approximately 3 ounces of mucus produced daily in a healthy person are pushed by the cilia toward the mouth where it is swallowed or expelled. Excessive production of sputum is abnormal and occurs during respiratory infections as well as COPD. Additional physical protection for the lungs includes coughing which tends to alleviate irritation and eliminate accumulated phlegm. Sneezing tends to relieve irritation and tickling inside the nose by removing irritants and pollutants. Sneezing is initiated by the sneezing centers in the brain.

COPD

Chronic Obstructive Pulmonary Disease is an umbrella term describing a disorder that affects the lungs and is characterized by inflammation and narrowing of the bronchial tubes, thereby reducing the amount of air that flows in and out of the alveolar sacs. The disease is accompanied by coughing episodes, shortness of breath, increased production of sticky sputum and wheezing.

These types of COPD may show a significant degree of overlapping in symptoms and treatment. The distinguishing feature in **asthma** is that airflow obstruction occurs as a result of a reversible bronchospasm. **Chronic bronchitis** causes irreversible structural changes in the mucus glands and bronchial mucosa. **Emphysema** is accompanied by irreversible loss of elasticity and eventual destruction of the connective tissue supporting the alveoli sacs leading to air trapping and airway collapse. COPD is a debilitating and generally fatal disease, but it can be managed, controlled and its progression can be slowed. COPD is one of the most common lung diseases. In general, asthma is not considered a true COPD since it is reversible, but will be included in this discussion

EPIDEMIOLOGY

COPD is a very common disorder both in the U.S. and worldwide. Surveys, hospital discharge records and World Health Organization (WHO) records indicate that COPD is the third leading cause of death in the U.S. It accounts for 4% of all deaths. WHO estimates that in 2000, 2.74 million people died of COPD worldwide. The U.S. ranks 12th among industrialized nations in COPD mortality in men and 7th in women. It is estimated that there may currently be 16 million people in the U.S. diagnosed with COPD. Furthermore, it is estimated that there may be as many as an additional 14 million or more in the U.S. who are undiagnosed and whose symptoms are in the early stages. COPD is the leading cause of hospitalizations among the elderly. About 762,000 hospitalizations due to COPD occurred in 2000, and the total estimated cost of COPD in 2002 was $32.1 billion. The Centers for Disease Control (CDC) reported that there were 124,816 deaths in the U.S. in 2002. Evidence indicates that in most of the world COPD prevalence and mortality are on the rise in response to an increase in smoking particularly by women and adolescents. Although smoking is considered the main cause of COPD, WHO estimates that there are 400,000 annual deaths worldwide as a result of exposure to biomast fuels, that are derived from plants and other organic waste.

FORMS OF COPD

Types of COPD include: 1) chronic bronchitis, 2) emphysema, and 3) bronchial asthma.

CHRONIC BRONCHITIS

Acute bronchitis is a common respiratory disease that results in inflammation of the bronchial tubes and is accompanied by excessive secretion of mucus within the tubes. This results in expectoration and coughing. The condition may last up to two weeks, and the symptoms disappear without any complications.

On the other hand, chronic bronchitis is an ongoing inflammation and swelling of the mucus membranes of the bronchial tree characterized by increased production of thick sputum and persistent cough that occurs daily for at least three months over a two year period. The disease is associated with structural changes in the bronchial mucosa and hypertrophy of the mucus producing glands. Gradually, obstruction of the air passages occurs and the flow of air is reduced. The cells of the bronchial mucosa appear irritated, hyperemic, edematous and the mucociliary function becomes impacted. As a result, the thick mucus accumulating in the bronchial branches increases the irritation and intensifies the characteristic cough. The bronchial mucosa releases inflammatory mediators such as interleukin 8 and other pro-inflammatory cytokines in response to inflammatory stimuli such as smoking and pollutants. Chronic bronchitis may occur as a sequence to a series of acute bronchitis attacks.

Causes

Cigarette smoking is the most dominant risk factor for the development and progression of chronic bronchitis. It accounts for 85-90% of chronic bronchitis and COPD. It has been postulated that the oxidants present in smoke play a role in triggering the initial and later phases of chronic bronchitis. The oxidants tend to inactivate a_1-antitrypsin (AAT) and cause polymorphonuclear leukocytes to release proteolytic enzymes and to initiate a low-grade inflammation. Eventually smoking inactivates the ciliary movement and causes hypertrophy and hyperplasm of the mucus-secreting glands. Long term exposure to bronchial irritants such as organic and inorganic dust or fumes may play an important role in causing chronic bronchitis. Familial and genetic factors may also predispose the development. It has been shown that children of smokers may develop severe respiratory disorders such as COPD.

Allergens may cause mucus hypersecretion, thus leading to symptoms that resemble those of bronchitis.

Diagnosis

Physical examination as well as studying medical history and identifying symptoms experienced by the patient are important first steps toward diagnosis of this disease. In addition the following tests will assist in confirmation of the disorder:

Pulmonary Function Test (PFT): This involves a series of breathing attempts that record the air flow and volume of air in the respiratory tract. This process assists in determining how well the lungs are functioning.

High Resolution Computed Tomography (HRCT): This is a CT scan that shows the images of the lungs and any irregularities that may be present.

Chest X-Ray: Its usefulness is that it helps in ruling out other lung disorders.

Sputum Examination to confirm the presence of neutrophil granulocytes.

Signs and Symptoms

Signs and symptoms of chronic bronchitis develop slowly and gradually intensify if the patient fails to seek medical advice. The main symptoms are:

a) Cough and increased sputum production: The initial sign of chronic bronchitis is a mild morning cough that may be dismissed as normal smoker's cough. With time the cough becomes more severe and the production of sputum increases, especially upon arising. The sputum may be clear, yellowish, green or may include a small amount of blood. Production of sputum decreases as the day progresses.

b) Dyspnea: Difficulty in breathing begins in the early stages of the disorder, but it gradually increases in severity. At the beginning, dyspnea occurs with daily activity, but later on it may be experienced at rest.

c) Wheezing: Occurs as a result of partial obstruction of the airways resulting from the presence of sputum or inflammation.

Other symptoms include fatigue, muscle aches, nasal congestion, headaches and fever, which indicates the presence of an infection.

Prognosis

Even though chronic bronchitis is a progressive disorder, early diagnosis, cessation of smoking and avoidance of exposure to airborne dust and pollutants may result in good prognosis for a significant number of years. Improvement level depends on the degree of damage present as well as compliance with therapy. Patients who continue to smoke and live in an environment where pollutants are unavoidable have fair to poor prognosis.

Treatment

Treatment of chronic bronchitis is generally aimed at alleviation of symptoms. Since the damage to the bronchial mucosa of the bronchial tubes is irreversible, treatment does not lead to cure. Medications commonly used in the management of chronic bronchitis include: **bronchodilators**, **corticosteroids**, and **expectorants**.

Bronchodilators

The main action of these medications is to relieve bronchospasm. This activity may be achieved by using sympathomimetics, especially beta$_2$-adrenergic stimulants (agonists), anticholinergics and methylxanthines. Selective beta$_2$-adrenergic agonists include **albuterol, terbutaline and metaproterenol**. This group

achieves bronchodilation with fewer cardiac side effects than are experienced with adrenaline and isoproterenol. These three groups may be given orally or by inhalation. All have similar mechanisms of action.

Albuterol Sulfate

It has been postulated that albuterol and other beta$_2$-adrenergic agonists stimulate the production of cyclic adenosine-3, 5-monophosphate (AMP) by activation of the enzyme, adenyl cyclase. Ultimately, the increased level of AMP enhances the activity of cyclic AMP-dependent protein kinase A, which reduces the intracellular calcium concentration, resulting in relaxation of the bronchial smooth muscle, and in inhibition of the release of the chemical mediators from the mast cells in the bronchial tree. Albuterol has lesser effects on the b$_1$-adrenergic receptors of the heart. It is well absorbed from the GI tract and is absorbed from the respiratory tract following oral inhalation.

The side effects associated with oral albuterol sulfate are dose related and are more intense than those encountered with inhalation, which are usually minimal. Unlike inhalation use, orally administered albuterol may produce some activities of adrenergic receptors. Patients should be advised to report any chest pain or changes in heart rate. Other adverse effects are tremor, nausea, anxiety, nervousness, headache, insomnia, increased sweating and fatigue.

Albuterol may be given orally or via oral inhalation by means of a metered-dose nebulizer or a specially designed oral inhaler that supplies powdered medication from capsules placed in the device. The usual inhaled dose for children 4 years of age and older and adults is 180 mg (2 inhalations) every 4 to 6 hours. It is recommended that there be a one minute time lag between the two inhalations. The usual dose for powdered albuterol is 20 mg every 4 to 6 hours for adults and children 4 years of age and older. Recommended maintenance dose is 180 mg 4 times daily.

Terbutaline

This drug is a fast-acting synthetic sympathomimetic amine that can be administered both orally and by inhalation in order to dilate the bronchial branches. It has a mechanism of action similar to albuterol. In addition to its bronchial activity terbutaline has been used to delay premature labor for 48 hours to allow for the lungs to mature. However, the labor delay should not exceed 48 to 72 hours. In 2011 the FDA ordered that the drug label should state that pregnant women should not be given injections of terbutaline for the prevention of preterm labor or for long term (beyond 48-72 hours) management of preterm labor. Additionally, oral terbutaline should not be used for any type of prevention or treatment of preterm labor due to the potential for serious internal heart problems and death.

The most common adverse reactions are dose related and include tremors, increased anxiety, tachycardia, changes in blood pressure, nervousness, palpitations and dizziness. The oral inhalation dose is 200 - 500 mg (2 metered inhalations) every 4 to 6 hours.

Metaproterenol Sulfate

This is a sympathomimetic amine that acts as a beta$_2$-adrenergic receptor stimulant. The adverse effects of this drug are similar to other sympathomimetic agents. It may be administered orally or via oral inhalation. Adult dose by inhalation is 1.3 to 2.25 mg (2 or 3 inhalations) every 4 to 6 hours.

Formoterol

This drug is a long-acting beta$_2$-agonist with duration of action that lasts up to 12 hours. Short-acting beta$_2$ agonists usually last from 4 to 6 hours. This drug is usually administered with corticosteroids such as fluticasone. Formoterol as well as other long-acting beta$_2$-agonists are not recommended for the treatment of acute asthma. Adverse effects are similar to other sympathomimetics. In 2005 the FDA issued a warning to the public informing them that the use of long-acting beta$_2$-adrenergics may exacerbate wheezing symptoms in certain patients.

Salmeterol

Salmeterol is also a long-acting beta$_2$-agonist which is available as a dry powder inhaler. It is mostly used for severe persistent asthma that failed to respond to short-acting beta$_2$-agonists. It is normally adminis-

tered with corticosteroids such as beclomethasone. The most encountered side effects are dizziness, sinus infection, tachycardia, elevation of blood pressure and migraine headaches.

ANTICHOLINERGICS

Ipratropium Bromide

The main anticholinergic used in the management of chronic bronchitis and other COPD's is ipratropium bromide.

Atropine is not used due to its side effects, especially desiccation of bronchial secretions.

When administered in a metered-dose inhaler, ipratropium bromide relaxes the bronchial tubes. It is available in oral aerosol and in solution for nebulization. Ipratropium acts as a nonselective competitive antagonist at muscarinic receptors in the bronchial tree and other tissues. As a result, it causes relaxation of the smooth muscles of the bronchi and bronchioles. When administered via inhalation, it causes no appreciable changes in heart rate, rhythm and blood pressure. Because it is administered by inhalation, ipratropium bromide's adverse effects are less frequent than other orally administered antimuscarinic agents. The usual, initial oral inhalation dose when administered alone or with albuterol sulfate in adults and children 12 years of age and older is 36 mg (2 inhalations) 4 times daily.

XANTHINE DERIVATIVES

Aminophylline is the xanthine derivative used as a bronchodilator. It is a methylated form of xanthine. To be effective it must be converted to theophylline. It acts by inhibiting mast cell degranulation and liberating of histamine and other chemical mediators. It inhibits the action of the enzyme phosphodiesterase. It is used mainly in the treatment and prevention of asthma attacks, and COPD. Other xanthine derivatives used in COPD are dyphilline, oxtriphylline, and theophylline. The dose is usually personalized depending on the medical need. The main adverse effects are irritation of the gastric mucosa, CNS stimulation, cardiac palpitation, tachycardia, flushing, hypotension and INCREASED DIURESIS.

CORTICOSTEROIDS

Corticosteroids are potent anti-inflammatory agents and cause stabilization of the membrane of the lysosomes, thereby preventing the release of hydrolytic enzymes that trigger inflammation. Due to the seriousness of the adverse effects of corticosteroids, their prolonged systemic use is not recommended. Inhaled corticosteroid aerosols produce a decrease in bronchial hyperactivity and prevention of symptoms. This route of administration may result in avoidance of adverse effects following systemic use. A number of corticosteroids have been structurally modified to diminish systemic absorption from the bronchial tree. These include **beclomethasone, flunisolide, budesonide, fluticasone, mometasone and triamcinolone**. Inhaled corticosteroids are usually delivered using a metered-dose inhaler, but are also often available for dry powder inhalers. They may be used to treat stable symptoms of COPD or symptoms that are increasingly getting worse, particularly patients with chronic bronchitis and frequent exacerbation. The dosage is dependent on age of the patient, severity of the case, and other medication used in the treatment. The adult dose of beclomethasone is 2 oral inhalations, 3 to 4 times daily. For flunisolide, the dose is 2 oral inhalations twice daily, morning and night for children 4 years and older and adults. Generally, the adult dose of flunisolide inhalations is once or twice daily. The adult dose for fluticasone is usually twice a day. The adult dose of triamcinolone is 2 inhalations, 3-4 times daily.

ANTIBIOTICS

Individuals with chronic bronchitis and emphysema are prone to bronchial infections. A broad spectrum antibiotic, especially one that is effective against lactamase-producing organisms, may be given for a 7-10 day course. This may be repeated at the first sign of recurrent bronchial infection or sputum purulence.

EMPHYSEMA

Emphysema is a chronic pulmonary disease characterized by permanent abnormal enlargement of the air spaces distal to the terminal bronchioles, with destructive changes in the alveolar wall. The incidence of emphysema appears to occur as a result of individual susceptibility and exposure to provocative factors. Emphysema is believed to occur as a result of a consequence of the action of the proteolytic enzyme neu-

trophil elastase on the elastic tissue of normal lungs. The enzyme is normally inhibited by alpha$_1$-antitrypsin (AAT). Either deficiency or absence of AAT allows the neutrophil elastase to cause structural changes in the lungs and the development of early onset emphysema. The enzyme also is responsible for inducing mucus glands, hyperplasia, mucus secretion, and a decrease in the ciliary beat frequency. These are also features of chronic bronchitis. Furthermore, the enzyme, neutrophil elastase, has a detrimental effect on the epithelial cells causing inactivation of immunoglobulin, impairment of mucociliary clearance, and reduction in the ability of the airways to remove bacteria and retain its sterility. In the presence of congenital deficiency of AAT, emphysema may appear by age 40 among cigarette smokers and by age 55 to 60 in non-smokers.

Emphysema, like chronic bronchitis, is closely related to cigarette smoking. Oxidants present in the smoke have harmful effects and trigger the development of emphysema. As in chronic bronchitis, oxidants tend to inactivate AAT and cause polymorphonuclear leukocytes to release proteolytic enzymes acutely and to form a low-grade inflammation. The ultimate result of smoking is impairment of ciliary movement and formation of hypertrophy and hyperplasia of mucus-secreting glands. In addition, smokers tend to show a decline in lung function throughout their adult lives. Familial and genetic factors may play a role in the development of emphysema. Children who are deficient in the protein inhibitor, AAT, most likely will develop an early onset of emphysema.

Emphysema, like chronic bronchitis, is insidious in nature and may begin its development without noticeable pulmonary obstruction. As the destruction and inflammatory processes progress, the patient begins to experience dyspnea. The most important feature of emphysema is the continuous destruction of the walls of millions of alveolar sacs. Additionally, the elasticity of the lung is gradually destroyed, and the alveoli begin a slow rupture, resulting in fewer but larger alveoli. The result is reduced lung surface area and diminished number of capillaries in the remaining alveoli, as well as reduced level of oxygen that crosses the alveolar wall into the blood stream.

The symptoms of emphysema may begin early in life. In the absence of taking the necessary precautions and failure to treat the condition, the signs and symptoms will intensify and eventually become debilitating. Many patients do not seek medical assistance until the symptoms become obvious and interfere with daily activities. The major complaints are cough, wheezing, difficulty in breathing, recurring respiratory infection and weakness. Dyspnea becomes the chief complaint. Cough that follows dyspnea produces scant and mucosal sputum. The patient becomes distressed and tends to utilize muscles of respiration to assist with inspiration. Expiration is usually prolonged with faint, high-pitched rales in the bronchial tubes. Carbon dioxide retention and decreased supply of oxygen tends to cause cyanosis, especially in advanced cases.

Treatment, prevention and prognosis are similar to those stated earlier under chronic bronchitis.

BRONCHIAL ASTHMA

Some clinicians do not consider bronchial asthma as COPD since it is a reversible disorder, while others consider it as an obstructive pulmonary disease. Bronchial asthma is a reversible, obstructive lung disease characterized by increased responsiveness of the trachea and bronchi to various stimuli. Unlike emphysema and chronic bronchitis the pulmonary obstruction that usually occurs as a result of narrowing of the airways is reversible, either spontaneously or following treatment. Asthma is manifested by recurrent attacks of dyspnea, cough and expectoration. Expiration is prolonged and usually accompanied by wheezing and rales. Asthma occurs in about 4-5% of the population in the U.S. It can affect individuals of all ages, but occurs predominately in children and young adults. The disorder occurs in individuals with a nonspecific, hypersensitive, tracheobronchial tree. The attacks are usually incited by stimuli such as dust, grass, pollen, mold spores, animal dander and pollutants. Such asthma is known as allergic asthma. Non-allergic asthma is not triggered by allergens, but rather as a result of non-immunologic stimuli such as infection, cold air, sudden change in temperature or emotional stress. Furthermore, non-allergic asthma attacks may be caused by factors that cannot be explained.

Asthma attacks may occur abruptly within a short time following exposure to an allergen or stimuli. During the attack, the bronchial tree is narrowed due to spasm of the smooth muscle of the respiratory tract and swelling of the mucus membrane caused by edema and inflammation. The patient experiences dyspnea, wheezing, coughing and expectoration. The patient may become cyanotic due to lack of oxygen. The attacks may last from a few minutes to several hours. Status asthmaticus is a term used when the attack becomes prolonged.

Treatment of bronchial asthma is identical to that of chronic bronchitis. Cromolyn sodium and its derivatives are used only for asthma.

SUMMARY

COPD is a common and financially costly debilitating disease which is responsible for a significant number of fatalities. The predominant cause is cigarette smoking, but other factors may play a role in the development of COPD. There is no cure, but it can be managed to improve the quality of life and reduce its progression. There are a number of medications used mainly for symptomatic relief.

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Please fill out this section as a means of evaluating this lesson. The information will aid us in improving future efforts. Either circle the appropriate evaluation answer, or rate the item from 1 to 7 (1 is the lowest rating; 7 is the highest).

1. Does the program meet the learning objectives?

List the various parts of the respiratory tract and their functions.	YES	NO
Describe the symptoms of COPD.	YES	NO
Differentiate between chronic bronchitis, emphysema and bronchial asthma.	YES	NO
Discuss the medications used in treating COPD.	YES	NO
Discuss the prevalence, causes and prognosis of COPD.	YES	NO

2. Was the program independent & non-commercial YES NO

	Poor			Average		Excellent	
3. Relevance of topic	1	2	3	4	5	6	7

4. What did you like most about this lesson?_____

5. What did you like least about this lesson?_____

Please Select the Most Correct Answer(s)

1. The alveoli contain C-shaped cartilaginous rings.
   A. True
   B. False
2. The 2002 estimated costs of hospitalizations due to COPD was:
   A. $15 billion
   B. $20 billion
   C. $32.1 billion
   D. $48.7 billion
3. Chronic bronchitis is confirmed:
   A. If symptoms occur abruptly
   B. In presence of fever
   C. Wheezing that lasts for 1 month
   D. Daily persistent cough for at least 3 months for 2 successive years
4. A side effect of aminophylline is:
   A. Bradycardia
   B. Dilation of pupil of the eye
   C. CNS depression
   D. Gastric distress & irritation
5. Which of these is NOT used to diagnose chronic bronchitis?
   A. Skin test
   B. HRCT
   C. PFT
   D. Sputum examination
6. Which of these is a beta2-adrenergic agonist?
   A. Adrenaline
   B. Terbutaline
   C. Isoproterenol
   D. Ipratropium
7. Cigarette smoking:
   A. Stimulates release of globulin
   B. Eventually triggers immunity against inflammation
   C. Causes desiccation of mucus
   D. Inactivates ciliary movement in the bronchial tree
8. Deficiency or absence of AAT allows the enzyme, neutrophil elastase, to cause structural changes in the lung.
   A. True B. False
9. Asthma has no cure & it may result in death.
   A. True B. False
10. Which of the following symptoms is absent in asthma?
    A. Cyanosis
    B. Cough
    C. Permanent destruction of respiratory mucosa
    D. Dyspnea

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