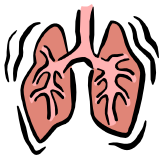
CEprn®
A PHARMACY CONTINUING EDUCATION PROGRAM
W-F Professional Associates, Inc. 400 Lake Cook Rd., Suite 207 Deerfield, IL 60015 847-945-8050

March 2003 "Review of COPD" 707-000-03-003-H01



THIS MONTH "COPD"

MISSING A LESSON? IT'S EASY TO GO TO OUR WEBSITE & DOWNLOAD WHAT YOU NEED. (www.wfprofessional.com)

LESSONS ARE USUALLY ON THE WEBSITE BY THE FIRST DAY OF EACH MONTH.

ALWAYS KEEP A COPY OF YOUR QUIZZES. YOU MAY MAIL QUIZZES TO US, FAX THEM (847-945-5037), OR SEND US A CONVENTIONAL EMAIL WITH YOUR ANSWERS. (info@wfprofessional.com)

HAVE YOU RECENTLY MOVED? PLEASE NOTIFY US.

What's the difference between emphysema, chronic bronchitis & asthma? What's COPD? In this lesson, we review Chronic Obstructive Pulmonary Disease. Our goal is to provide the most common therapeutic options. 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 707-000-03-003-H01.

Pharmacists completing this lesson by March 31, 2006 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.

Complete List of 2003 Topics: See Page 10.

If you have any comments, suggestions or questions, contact us at the above address, or call toll free 1-800-323-4305. (In Alaska and Hawaii phone 1-847-945-8050). **Please write your ID Number (the number that is on the top of the mailing label) in the indicated space on the quiz page** (for continuous participants only).

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

1. Relate the prevalence of COPD.
2. List the major causes of COPD.
3. Differentiate between chronic bronchitis, emphysema, & asthma.
4. State the main goals of COPD therapy.
5. Describe the mechanism of action, route of administration & adverse effects of medications used in the treatment of COPD.

BACKGROUND

Chronic obstructive pulmonary disease (COPD) is actually a heterogeneous group of relentlessly progressive, largely irreversible disorders leading to changes in lung functions, debility and mortality. It is characterized by the presence of persistent airflow obstruction in the lungs with related symptoms such as coughing, exertion and expectoration. The component of these heterogeneous disorders consists of **three distinct pulmonary disorders: emphysema, chronic bronchitis and asthma**. These disorders may coexist, and they show a significant degree of overlapping in symptoms and treatment. The distinguishing feature in asthma is that airflow limitation occurs as a result of reversible bronchospasm. Emphysema is accompanied by irreversible destruction of the connective tissue supporting the alveolar wall lending to air trapping and airway collapse. Chronic bronchitis causes irreversible structural changes in the mucous glands and bronchial mucous membrane. The chronic airway inflammation associated with emphysema and chronic bronchitis differs from that seen in asthma.

Prevalence:

COPD is a common, costly public health problem in both the USA and around the world. National surveys, hospital discharge records and reports published by the World Health Organization (WHO) indicate that COPD is the fourth leading cause of death in the USA, and accounts for 4% of all deaths. In order of frequency, the leading causes of deaths in the USA are: heart disease, cancer, cardiovascular disease (stroke), COPD, accidents, pneumonia and influenza, diabetes, suicide and liver disease. The USA ranks 12th among industrialized nations in COPD mortality in men and 7th in women. In 1996, the estimated number of individuals in the USA with COPD was over 10 million. This translates to be approximately 6% of the adult population. COPD is the leading cause of hospitalizations among adults, particularly in the elderly. Chronic bronchitis and emphysema are usually diagnosed between the ages of 55 and 65. They are more common among men than women with a male: female ration of 9:1. COPD is responsible for approximately 2% of all hospitalizations among adults. The percentage rises significantly in older individuals ranging in age from 55 to 75 years. The annual costs attributed to COPD in the USA are more than $14 billion. In spite of the fact that COPD is a leading cause of morbidity and mortality, its awareness as a major health problem has not stimulated attention, as has heart and cardiovascular disease. While the death rates from coronary diseases and strokes have declined, the rate for COPD has increased.

Definitions:

Emphysema is a chronic pulmonary disorder characterized by permanent, abnormal enlargement of the air spaces distal to the terminal bronchiole, with destructive
changes in the alveolar wall.

Chronic bronchitis is a chronic inflammation of the mucous membrane of the bronchial tubes, and is characterized by increased mucous secretion and certain structural changes in the bronchi. A productive cough is usually present for at least three months in each of two successive years, provided that the cough is not produced by other causes.

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 $90.00 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.
March 2003

Asthma is a respiratory disorder characterized by recurring dyspnea caused by reversible smooth muscle contraction that narrows the airway lumen, limiting airflow, and resulting in coughing, wheezing and expectoration. The main difference between asthma and other types of COPD is the reversibility of symptoms.

EMPHYSEMA AND CHRONIC BRONCHITIS

Etiology:

The incidence of emphysema and chronic bronchitis appears to occur as a result of individual susceptibility and exposure to provocative factors. Emphysema is believed to occur as a consequence of the action of the proteolytic enzyme neutrophil elastase on the elastic tissue of the lungs. The enzyme is normally inhibited by a_1-antitrypsin (AAT). Either deficiency or absence of AAT will allow the neutrophil elastase to cause structural changes in the lungs and the development of early onset emphysema and chronic bronchitis. The enzyme also is responsible for inducing mucus gland hyperplasia, mucus secretion, and a decrease in the ciliary beat frequency, which are 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 and chronic bronchitis are closely related to cigarette smoking, which is considered the most dominant risk factor for the development and progression of COPD. Oxidants that are present in smoke play an important role in the development of chronic bronchitis and emphysema. It has been postulated that the oxidant tends 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.

It appears that atmospheric pollution produced in heavily industrialized urban areas can contribute to the development of COPD. Chronic bronchitis is more common among workers whose occupation may expose them to bronchial irritants such as inorganic or organic dusts or noxious gases.

Familial and genetic factors may predispose the development of COPD. It has been shown that children of smokers may develop more severe respiratory disorders especially chronic ones. Children who are deficient in the protease inhibitor, AAT, most likely will develop an early onset of emphysema.

Pathophysiology:

Emphysema and chronic bronchitis are insidious in nature and may exist without noticing pulmonary obstruction. As the destruction and inflammatory processes progress, the patient begins to experience dyspnea. The most important aspect of emphysema is the continuous destruction of the walls of some of the millions of alveoli (air sacs) located at the end of the bronchioles in the lungs. Oxygen and carbon dioxide exchange takes

Statement of Ownership, Management & Circulation

1.	Publication Title: CE PRN®		
2.	Publication No. 0051-8730		
3.	Filing Date: 9/27/02		
4.	Issue Frequency: Monthly, January through November		
5.	No. of Issues Published Annually: 11		
6.	Annual Subscription Price: $90		
7.	Complete Address of Known Office of Publication:	400 Lake Cook Road Suite 207Deerfield, IL 60015	
8.	Complete Mailing Address of Headquarters or General Business Office of Publisher:	Same as #7 Above	
9.	Full Names & Complete Mailing Addresses of Publisher, Editor, & Managing Editor	William J. Feinberg	Same Address as #7
10.	Owner: W-F Professional Assoc., Inc.	William J. Feinberg, RPh, MBA, President	
14.	Issue Date for Circulation Data Below: September, 2002		

	Average No. of Copies Each Issue During Preceding 12 Months	Actual No. Copies of Single Issue Published Nearest Filing Date
Total No. Copies (Net Press Run)	4,200	4,200
Paid or Requested Mail Subscriptions	2,800	2,800
Total Paid and/or Requested Circulation	2,800	2,800
Total Distribution	2,800	2,800
Copies Not Distributed	1,400	1,400
Total	4,200	4,200
Percent Paid and/or Requested Circulation	100%	100%

place in the walls of the alveoli. In addition, the elasticity of the lungs is gradually destroyed, and the alveoli gradually rupture and manage to make fewer but larger alveoli. This results in 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 and into the bloodstream. In chronic bronchitis, there are widespread abnormalities and inflammation in the small bronchi and bronchioles. In the early stage, it is difficult to detect, but with repeated infection of the bronchi and bronchioles, the lining of these tubes thickens and becomes distorted. The lumen becomes narrow and impacted with mucus. The specific pathologic findings include hyperplasia and hypertrophy of the submucosal bronchial mucus glands, hyperplasia of bronchiolar goblet cells, chronic and acute inflammatory infiltrates in the bronchial mucosa, and profuse inflammatory exudates in the lumen of the bronchi and bronchioles.

Signs and Symptoms:

Emphysema and chronic bronchitis may begin early in life, but the symptoms and signs gradually intensify, and eventually become disabling by middle age. In emphysema, the age at the time of diagnosis is usually 60 years. Many patients do not seek medical assistance until the symptoms become significant. Patients see a physician mostly because of persistent cough, wheezing, recurring respiratory infection, weakness and weight loss resulting from energy expenditure. Dyspnea, which usually progresses gradually, becomes the chief complaint. Cough, which usually starts after dyspnea, produces scant and mucoid sputum. The patient is usually distressed and tends to utilize muscles of respiration to assist with inspiration. Expiration is usually prolonged with faint, high-pitched rale in the bronchial tubes. Physical findings vary depending on the stage of the disease at the time of examination. A common abnormality is obstruction to expiratory airflow. Other symptoms include hyperinflation of the lungs, with depressed diaphragm; hypertrophy of accessory muscle of respiration; pursed-lip breathing; and tachycardia. Carbon dioxide retention tends to occur in advanced cases.

The main symptom of chronic bronchitis is chronic cough with sputum that is often copious and purulent. As stated earlier, the definition of chronic bronchitis specifies that productive cough be present on most days for a minimum of 3 months in the year and in at least 2 consecutive years. The disease is encountered in smokers over the age of 35. Initially, cough and sputum production occur on awakening in the morning, but later on, the productive cough increases in frequency and intensity and symptoms of exertional dyspnea develop. Progression of the disease is usually accompanied by recurrent episodes of respiratory infection, resulting in exacerbation of the chronic bronchitis. The patient may experience increased cough with purulent sputum, fever and dyspnea as well as episodes of respiratory failure that leads to intubation and need for almost continuous ventilatory assistance. During an attack the patient may become cyanotic and experience tachypnea. Death usually occurs as a result of respiratory failure.

ASTHMA

Asthma is a reversible, obstructive lung disease characterized by increase 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. It is manifested by recurrent attacks of dyspnea, cough and mucoid sputum. Expiration is prolonged and accompanied by wheezing and rales.

Prevalence:

Asthma occurs in about 4 – 5% of the population in the USA. It can affect individuals of all ages, but occurs predominantly in childhood and early adulthood. About 50% of the cases occur before the age of 10, and another one-third before the age of 40. About 50% of asthmatic children "outgrow" the disease and become symptom-free. Only 5 - 10% continue to have symptoms throughout their life.

Etiology:

Asthma occurs in individuals with a nonspecific, hypersensitive, tracheo bronchial tree. Asthma may be classified according to the principle stimuli that incite the asthma attack. There are two main types of asthma: **allergic** and **non-allergic.** In allergic asthma, reaction to external factors such as dusts, grass, pollen, mold

spores, animal dander and pollutants are responsible for triggering asthma attacks. The characteristics of this type include: positive skin tests to intradermal injection of extract of airborne allergens, increased level of IgE, childhood or adult onset, presence of other allergies such as hay fever, and skin allergies. Initial exposure of a sensitive individual to one or more of the external allergens causes an increase in the production of the highly reaginic immunoglobulin antibody known as IgE. This antibody attaches itself to the surface of the pulmonary mast cells. Subsequent exposure results in generation and release of chemical mediators of hypersensitization such as histamine, slow-reacting substance of anaphylaxis (SRS-A) and bradykinin. These mediators are responsible for: 1) narrowing of small and large airways due to constriction of the smooth muscle of the bronchial tree; 2) excessive accumulation of mucoid secretions in the airways; and, 3) edema, and inflammation of the bronchial mucosa as a result of vasodilation.

Characteristics of nonallergic asthma are: no known external allergens, negative skin tests, IgE level is normal or low, onset is usually in adults, and absence of allergic disorders. The asthma attack occurs as a result of non-immunologic stimuli such as infection, cold air, sudden change in temperature and irritating pollutants. Furthermore, nonallergic asthma attack may be caused by factors that cannot be readily explained. The precise mechanism of this type of asthma is unknown.

Symptoms and Signs:

An asthma attack may occur abruptly within a short time following exposure to an allergen or stimuli. It may develop gradually following a respiratory infection. During the attack, the passageways of the bronchial tree are narrowed due to spasm of the bronchial tubes and swelling of their mucus membrane caused by edema and inflammation. The patient experiences dyspnea, accompanied by wheezing, coughing and expectoration. Due to constriction of the bronchial tree, the trapped inhaled air becomes saturated with high qualities of carbon dioxide, and fresh air with high oxygen content is prevented from reaching the air sacs where gas exchange takes place. The patient may become cyanotic and may find it necessary to free the air out of their lungs. The passage of the air through the narrowed passageways gives a wheezing sound that may be heard without a stethoscope. Asthma attacks may last for a few minutes to several hours. When the attack becomes prolonged, it is known as status asthmaticus.

TREATMENT

Emphysema and Chronic Bronchitis:

It is essential to realize that, since these disorders involve irreversible processes, therapy does not result in cure. However, the main goals are: 1) relief of symptoms, such as cough and bronchial secretion; 2) prevention of progression of the disease; 3) elimination and prevention of bronchial infection; 4) management of complications such as hypoxia and bronchospasm; and, 5) avoidance of bronchial and environmental irritants such as smoking and pollutants. Medications used include: bronchodilators, corticosteroids, cromolyn sodium and antibiotics.

Bronchodilators: This group of medications tends to relieve bronchospasm. Bronchodilation may be achieved by employing the following categories: **sympathomimetics, especially b_2-adrenergic stimulants (agonists), anticholinergics, and methylxanthines.** Selective b_2-adrenergic agonists including albuterol, terbutaline and metaproterenol. They act as bronchodilators with fewer cardiac side effects than experienced with adrenaline or isoproterenol. These three agents are given orally or administered by inhalation.

Albuterol is a synthetic sympathomimetic amine that is sparingly soluble in water, but the sulfate version is water soluble, and is used for nebulization. Albuterol is a selective b_2-adrenergic agonist with little or no effect on b_2-adrenergic receptors. It has been postulated that albuterol and other b_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. Albuterol sulfate is well-absorbed when administered orally. Peak blood level occurs within 2.5, 2 and 6 hours following the administration of conventional tablets, oral solution and extended-release tablets, respectively. The bronchodilation effect following the ingestion of oral conventional tablets peaks

within 2-3 hours and may last up to six hours. The onset of action is between 15 and 30 minutes. Extended-release tablets usually last up to 12 hours. Albuterol is absorbed from the respiratory tract following oral inhalation. It has been estimated that less than 20% of a single dose is absorbed when administered by nebulizer or by intermittent positive-pressure breathing (IPPB). The remaining 80% was in the expired air and the apparatus used. A significant amount of the inhaled dose is swallowed and absorbed from the GI tract. The onset of action of inhaled albuterol is 5 to 15 minutes, and the effect peaks in 1 to 1 ½ hours. The duration of action is 3 to six hours. Animal studies indicate that albuterol is capable of crossing the blood-brain barrier as well as the placenta. However, it is unclear if the drug is distributed in the milk.

Albuterol is used mostly for relief of bronchoconstriction in reversible, obstructive pulmonary disease, such as asthma. It is preferred over isoproterenol due to its longer duration of action following oral or orally inhaled dosage. Additionally, its cardiac stimulation is much less frequent and milder than that encountered with isoproterenol.

Adverse effects of conventional or extended-release tablets or syrup are usually transient and do not necessitate discontinuation of therapy. The adverse reactions associated with oral administration are more intense than those encountered with inhalation. When administered by inhalation, the side effects are minimal, and severe effects are uncommon, even if the patient self-administers an excessive dose. Unlike inhalation use, orally administered albuterol may produce some activation of the b_1–adrenergic receptors in the heart. Excessive oral doses may cause angina pectoris and tachycardia. The b_1–adrenergic stimulation caused by inhalation is minimal. Patients should be advised to report any chest pain or changes in heart rate. Some b_2-adrenergic agonists may cause tremor due to stimulation of the b_2-receptors on the skeletal muscle. Other adverse effects include nausea, increased anxiety and nervousness. Less encountered side effects include sedation, difficulty in urination, headache, dizziness, insomnia, increased sweating, and weakness.

Dosage and Administration: Albuterol may be given via oral inhalation by means of a metered-dose inhaler or nebulizer. Likewise, albuterol sulfate is administered by either metered-dose inhaler or a specifically designed oral inhaler that supplies powdered medication from a capsule placed in the device. It is essential that pharmacists fully explain the use of metered-dose inhalers, especially for first-time users. To achieve bronchodilation or prevent asthma attacks, the usual inhaled dosage 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 delivered by a special device is 20 mg every 4 to 6 hours for adults and children 4 years of age and older. Recommended maintenance therapy is 180 mg, 4 times daily. The oral adult dose is 2 to 6 mg, 3 to 4 times daily. In some cases, the oral dose may be increased to 8 mg four times daily. Extended-release tablets of 4 or 8 mg may be given every 12 hours.

Terbutaline is a synthetic sympathomimetic amine that can be administered both orally and by inhalation. It possesses a mechanism of action similar to albuterol. It stimulates the b_2–adrenergic receptors, but has little or no effect on the a–adrenergic receptors. One third to one half of an orally administered terbutaline dose is absorbed from the GI tract. It is well-absorbed via the subcutaneous route. The onset of action following inhalation is 5 to 30 minutes, and peak effects are achieved within one to two hours. Duration of action is 3 to 6 hours. Orally, the onset of action is 1 to 2 hours, peak effect is observed in 2 to 3 hours, and duration of action is 4 to 8 hours. Parenterally, the onset of action is within 15 minutes, and peak effects are experienced within ½ to one hour. This drug is partially metabolized in the liver and excreted into the urine.

The most common adverse reactions are dose related, and include tremors, increased anxiety, tachycardia, changes in blood pressure, nervousness, palpitation and dizziness. Less frequently reported are muscle cramps, nausea, vomiting, dry mouth or throat, and unusual taste in the mouth. The oral inhalation dose of terbutaline is 200 to 500 mg (2 metered inhalations) every 4 to 6 hours. The two inhalations should be administered one minute apart. Orally, an initial dosage of 2.5 mg, 3 to 4 times daily may be instituted. The maintenance adult dose is usually 5 mg, three times daily at approximately six-hour intervals. For children 12 to 15 years old, 2.5 mg 3 times daily is recommended. Subcutaneously, a 250 mg dose is administered, which may be repeated in 15 to 30 minutes.

Metaproterenol sulfate is a synthetic sympathomimetic amine that shares similarity with isoproterenol in chemical structure and in mechanism of action. It is a b_2-adrenergic receptor stimulant with little or no effect on a–adrenergic receptors. It is well absorbed from the GI tract, but less than half of the dose reaches systemic circulation as unchanged drug, because it is metabolized on first pass through the liver. Most of the metaproterenol in orally administered doses is eliminated in the urine. The adverse effects of this drug are dose related and are similar to those of other sympathomimetic agents. It may be administered orally or via oral inhalation by means of IPPB apparatus or nebulizer. For adults and children older than 9 years of age, the usual oral dose is 20 mg, 3 or 4 times a day. Metaproterenol by inhalation has an onset of action within 1 minute, and a peak effect within an hour. Adult bronchodilator dose by inhalation is 1.3 to 2.25 mg (2 or 3 inhalations) every 4 hours.

Anticholinergics:

Atropine is not used in COPD due to its side effects, especially desiccation of bronchial secretion. However, the anticholinergic agent **ipratropium bromide,** when administered in metered-dose inhaler, will act as a bronchodilator particularly in patients who suffer from chronic bronchitis. Ipratropium is a synthetic quaternary ammonium antimuscarinic agent. It is available in oral aerosol and in solution for nebulization. It acts as a nonselective competitive antagonist at muscarinic receptors in the bronchial tree and other tissues, and, consequently, it causes relaxation of the smooth muscle of the bronchi and bronchioles. It is considered a potent bronchodilator, and the inhalation of choice for patients with chronic bronchitis. When administered via inhalation, it causes no appreciable changes in heart rate, rhythm or blood pressure. Following inhalation, an insignificant amount of the dose reaches systemic circulation via the surface of the lungs or the GI tract. Because ipratropium bromide is administered by inhalation, its adverse effects are less frequent than other orally administered antimuscarinic agents. It is well tolerated and has a low incidence of side effects. 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. The 24 hour dose should not exceed 216 mg.

Xanthine Derivatives:

The Xanthine derivatives include caffeine, theophylline and theobromine. The main characteristics of these drugs are relaxation of the smooth muscles, in particular those of the bronchial tree, stimulation of the cardiac muscle and the central nervous system, and production of diuresis. The xanthine derivatives are methylated forms of xanthine and are usually known as methylxanthines. To be an effective bronchodilator, a methylxanthine must be converted to theophylline. The xanthines act by inhibiting mast cell degranulation and liberating of histamine and other chemical mediators. They also inhibit the action of the enzyme phosphodiesterase. The xanthine derivatives are used in both the treatment and prevention of asthma attacks, treatment of chronic bronchitis and emphysema. The main xanthine derivatives are aminophylline, dyphylline, oxtriphylline and theophylline. They are absorbed well from the GI tract, and the dose is usually personalized depending upon the medical circumstances. The main adverse effects include irritation of the gastric mucosa, CNS stimulation, cardiac palpitation, tachycardia, increased pulse rate, flushing, hypotension and transient increased urinary frequency. To minimize gastric irritation, it is recommended that the drug be taken after meals.

Corticosteroids:

The systemic use of corticosteroids in asthma may decrease airway obstruction. They cause stabilization of the membrane of lysosomes, thereby preventing the release of hydrolytic enzymes that trigger the inflammation. Inhaled corticosteroid aerosols produce a decrease in bronchial hyperactivity and prevention of symptoms. This route of administration may result in avoidance of systemic effects. A number of corticosteroids have been structurally modified to diminish systemic absorption from the bronchial tree. These include beclomethasone, flunisolide, and triamcinolone. The adult dose of beclomethasone is 2 oral inhalations, 3 to 4 times daily. Maximum dose is 840 mg (20 metered inhalations) daily. No dose has been established for children 6 years of age and younger. For children 6 to 12 years old, the dose is 1 to 2 metered sprays, 3 – 4 times daily to a maximum of 10 inhalations per day. For flunisolide, the dose is two oral inhalations twice a day, morning and night for children 4

years of age and older and adults. The adult dose of triamcinolone is 2 inhalations, 3 – 4 times daily. Up to 16 oral inhalations may be administered in severe cases. For children 6 to 12 years of age, 1 – 2 inhalations, 3 – 4 times daily is the norm. Maximum daily dose for children is 12 inhalations. Side effects of corticosteroids for oral inhalation include tachycardia, anorexia, cough and dizziness.

Cromolyn Sodium:

Cromolyn sodium and nedocromil sodium are antiasthmatic agents that act locally on the lung mucosa. They prevent the release of chemical mediators from sensitized mast cells in response to antigen-antibody reactions. After prolonged use, cromolyn sodium may result in hyposensitization. It has been postulated that this drug may block calcium channels in the mast cells. Cromolyn sodium and nedocromil sodium have no bronchodilator effect, nor do they have any effect on mediators that have already been released in the body. Thus, they do not relieve an asthma attack already in progress. Cromolyn sodium is used to prevent asthma attacks from taking place. It is administered by oral inhalation. The drug is poorly absorbed from the GI tract. Only 0.5 – 2% of an oral dose is absorbed. Approximately 8 – 10% of an oral inhalation dosage is absorbed in the lungs. About 98% or more of an orally administered cromolyn sodium dose is excreted in feces unchanged and 0.5% is eliminated in the urine. By inhalation, the adult prophylactic dose is 2 inhalations (1.6 mg), 4 times daily. A dose has not been established for children less than 5 years of age. The main side effects include dryness of the throat, cough, bad taste, wheezing and nausea.

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. The course may be repeated at the first sign of recurrent bronchial infection or sputum purulence. In England, antibiotics are given routinely during the winter months as prophylactic therapy.

CONCLUSION

COPD is a common disorder that it is the fourth leading cause of death in the U.S. Even though genetic factors may play a role in causing COPD, smoking is considered the most dominant risk factor for its development and progression. Sustained smoking cessation improves lung function, and it may retard progression of the disease. The use of bronchodilators and anti-inflammatory agents may provide symptomatic relief of bronchospasm.

REFERENCES

1. Hill, A., Gompertz, S., and Stockley, R. Thorax, 55:970 (2000)
2. Ferguson, G., Chest, 117:23S (2000)
3. Singh, J.M., Palda, V.A., Stanbrook, M.B., and Chapman, K.R., Arch Intern Med., 162:2527 (2002)
4. Hurd, S., Chest, 117:1S (2000)
5. McKenry, L.M., and Salerno, E., "Mosby's Pharmacology in Nursing," 19th Ed., Mosby, St. Louis, 737 (1995).
6. Lehne, R.A., "Pharmacology for Nursing Care," 2nd Ed., W.B.Saunders, Philadelphia, 819 (1994).

TOPICS FOR 2003	
Jan: Pharmacy Law Update 2003	Feb: Osteoporosis
Mar: COPD	Apr: Cystic Fibrosis
May: Lipid Management	Jun: Acne
Jul: Hepatitis	Aug: Ulcer Management
Sep: Psoriasis	Oct: Essential Hypertension
Nov/Dec: Anxiety	

MISSING ANY LESSONS? GO TO OUR WEBSITE (www.wfprofessional.com) & DOWNLOAD THEM.

Fill in the information below, answer questions and return **Quiz Only** for certification of participation to: CE PRN®, 400 Lake Cook Road, Suite 207, Deerfield, IL 60015.

NAME_____(1st line on label)_____

ADDRESS_____CITY_____STATE_____ZIP_____

CHECK IF NEW ADDRESS ❑ COMPANY WHERE EMPLOYED_____

LESSON EVALUATION

Please fill-out this section as a means of evaluating this lesson. The information will aid us in improving future efforts. Please rate each of the following from 1 to 7. Circle your choices. (1 is the lowest rating; 7 is the highest).

	Poor			Average			Excellent
1. Relevance of topic to practice.	1	2	3	4	5	6	7
2. Author's ability to communicate.	1	2	3	4	5	6	7
3. Author's knowledge of topic.	1	2	3	4	5	6	7
4. Appropriateness of topic.	1	2	3	4	5	6	7

5. Do you have any further comments about this lesson? _____

_____

Please Select the Most Correct Answer

1. COPD:
   A. Is the 4th leading cause of death in the US
   B. Is more common among women than men
   C. Is responsible for annual costs of $100 million in the US
   D. Death rate has decreased significantly in recent years
2. Emphysema is defined as a chronic pulmonary disorder characterized by permanent abnormal enlargement of the air spaces distal to the bronchiole.
   A. True B. False
3. Which of the following is NOT a causative factor of emphysema?
   A. Smoking
   B. Atmospheric pollutants
   C. Genetic factors
   D. Inhibition of ±-antitrypsin by the enzyme, neutrophil elastase
4. A characteristic of allergenic asthma is:
   A. Decreased level of IgE
   B. Irreversibility of the bronchospasm
   C. Increased level of IgE
   D. Absence of dyspnea
5. Ipratropium is:
   A. An anti-inflammatory agent
   B. The expectorant drug of choice
   C. Administered only by parenteral route
   D. An antocholinergic bronchodilator
6. Release of chemical mediators results in:
   A. Narrowing of small & large airways due to constriction of smooth muscle of the bronchial tree
   B. Bronchodilation
   C. Absence of mucoid secretion in the airways
   D. Vasoconstriction of the bronchial mucosa
7. Albuterol:
   A. Is a potent ±$_1$-adrenergic stimulant
   B. Is a selective ²$_2$-adrenergic agonist
   C. Is available only in tablet form
   D. Should not be used for symptomatic relief of asthma
8. Which of these is not considered a chemical mediator of hyposensitization?
   A. Histamine
   B. Adrenaline
   C. Slow-acting substance of anaphylaxis
   D. Bradykinin
9. Xanthine derivatives cause:
   A. CNS depression
   B. Bronchoconstrictor
   C. Diuresis
   D. Depression of cardiac muscle
10. Cromolyn sodium:
    A. Is a bronchodilator
    B. Prevents the release of chemical mediators from mast cells
    C. Is absorbed well from the GI tract
    D. Has antibacterial activity

Contributing Author
Farid Sadik, PhD, Dean
Univ. of South Carolina
College of Pharmacy
Columbia, SCL

Executive Editor
William J. Feinberg,
BS Pharm, MBA

CE PRN® is a publication of W-F Professional Associates, Inc.
This program is in printed format.

W-F Professional Associates, Inc. is approved by the American Council on Pharmaceutical Education (ACPE) as a provider of continuing pharmaceutical education.

Providers who are approved by ACPE are recognized by the following states: Alaska, Arizona, Arkansas, California, Connecticut, Delaware, District of Columbia, Florida, Georgia, Hawaii, Idaho, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Mississippi, Missouri, Montana, Nebraska, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, North Dakota, Ohio, Oregon, Pennsylvania, Puerto Rico, Rhode Island, South Carolina, South Dakota, Tennessee, Texas, Utah, Vermont, Virginia, Washington, West Virginia, Wisconsin and Wyoming.

Pharmacists completing this course by March 31, 2006 may receive full credit.
This program has been approved by the State Boards of Pharmacy in Alabama and Oklahoma.

This lesson furnishes 1.25 hours (0.125 CEUs) of credit.

Program ID #707-000-03-003-H01.