A NATIONAL EPIDEMIC

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Purpose and Goals
The goal of the enclosed course is to familiarize you with a variety of aspects of COPD, using an integrative approach. Nurses are important facilitators in coordinating treatments and teaching the patient in areas that range from nutrition, breathing exercises, medication usage/adherence, pathophysiology of the disease process, and alternative therapy modalities. You will also briefly learn about the four major diseases involved, including A/P, etiology, and pathogenesis. A review of ABGs, V/Q ratio, clinical features and treatments of COPD will be discussed. The goal is to provide you with optimum, accurate information including a lengthy section of Bonus Information that introduces you to natural methods, home remedies, and nutrient plans to give your patient the opportunity to incorporate complementary therapies along with their medical protocol.

Instructional Objectives
Upon completion of this course, the learner will be able to:
1. Summarize chronic bronchitis, emphysema, asthma and bronchiectasis.
2. Define the normal control of ventilation in order to identify abnormal pathologies like hypoxic drive and CO2 narcosis.
3. Differentiate auscultation of adventitious and vesicular breath sounds.
4. Define normal ventilation / perfusion ratios and relate abnormalities in disease states.
5. Outline normal blood gas values as a review of what disease states change these normalities.
6. Distinguish COPD abnormalities in roentgenograms objective symptomology and EKG’s and incorporate them with disease states.
7. Outline treatment options for COPD clients, by identifying areas researchers are working on to improve COPD’s effects.
8. Outline integrated material and synthesize to complete critical thinking segment.

Introduction
Chronic Obstructive Pulmonary Disease (COPD) is largely preventable. Yet it affects over 15 million Americans and is the third leading cause of death in the U.S. Some sources estimate the true numbers may be as high as 24 million. Although many diseases have seen a gradual decline in their associated mortality, COPD rates have increased. The term COPD is a broad one used to describe a set of symptoms, referring to persistent, slowly progressive obstruction of airflow and dyspnea, that is predominantly irreversible. It may be caused by chronic bronchitis, emphysema, or bronchiectasis.

People with COPD experience deterioration in functional status; therefore, improving function is a major goal of treatment. Nurses are often facilitators and coordinators of pulmonary rehabilitation. Evaluation of the effects of treatment is an essential aspect of providing quality care. Although some effects of COPD are permanent, you can do plenty to assist your patient in managing it, by educating him on how to gain back control of his health. This will help to decrease the depression, hopelessness, and pessimism that are commonly seen in patients with COPD.

Your specific nursing measures can help ease your patient’s breathing and prevent further deterioration, thus protecting the remaining lung function and offering a better quality of life.

Definitions, Anatomy and Pathology Review of COPD
Chronic obstructive pulmonary disease (COPD) is a term that applies to patients with chronic bronchitis, bronchiectasis, emphysema and, to a certain extent, asthma. A brief review of normal functional anatomy will provide a background for the discussion of pathology.

The airway down to the bronchioles normally is lined with ciliated pseudo-stratified columnar cells and goblet cells. Mucus derives from mucus glands that are freely distributed in the walls of the trachea and bronchi. The cilia sweep mucus and minor debris toward the upper airway.

Low humidity, anesthesia gases, cigarette smoking and other chemical irritants paralyze the action of these cilia. The mucociliary action starts again after a matter of time. This is why people awaken to “smokers cough.”

Bronchi run in septal connective tissue, but bronchioles are suspended in lung parenchyma by alveolar elastic tissue. The elastic tissue extends throughout alveolar walls, air passages, and vessels, connecting them in a delicate web. Bronchiolar epithelium is ciliated, single-layered and columnar or cuboidal. Beyond the bronchioles the epithelium is flat and lined with a film of phospholipid (surfactant), which lowers surface tension and thereby helps to keep these air spaces from collapsing. Remember that the phospholipid develops during later gestation in utero. This is the reason why premature infant’s lungs cannot stay inflated without the addition of surfactant therapy. Macrophages are found in alveolar lining. Smooth muscles surround the walls of all bronchi, bronchioles, and alveolar ducts and when stimulated they shorten and narrow the passages. Cartilage lends rigidity and lies in regular horse-shaped rings in the tracheal wall. Cartilage is absent in bronchi less than 1 mm in diameter.

The terminal bronchiole is lined with columnar epithelium and is the last purely conducting airway. An acinus includes a terminal bronchiole and its distal structures. Five to ten acini together constitute a secondary lobule, which is generally 1 to 2 cm in diameter and is partly surrounded by grossly visible fibrous septa. Passages distal to the terminal bronchiole include an average of three but as many as nine generations of respiratory bronchioles lined with both columnar and alveolar epithelium. Each of the last respiratory bronchioles gives rise to about six alveolar ducts, each of these to one or two alveolar sacs, and finally each of the sacs to perhaps seventy-five alveoli. Alveolar pores (pores of Kohn) may connect alveoli in adjacent lobules.
Two different circulations supply the lungs. The pulmonary arteries and veins are involved in gas exchange. The pulmonary arteries branch with the bronchi, dividing into capillaries at the level of the respiratory bronchiole, and supplying these as well as the alveolar ducts and alveoli. In the periphery of the lung, the pulmonary veins lie in the interlobular septa rather than accompanying the arteries and airways. The bronchial arteries are small and arise mostly from the aorta. They accompany the bronchi to supply their walls. In some cases of COPD, like bronchiectasis, extensive anastomoses develop between the pulmonary and bronchial circulations. This can allow major shunting and recirculation of blood, therefore contributing to cardiac overload and failure. Lymphatics run chiefly in bronchial walls and as a fine network in the pleural membrane. The lumina of the capillaries in the alveolar walls are separated from the alveolar lining surfaces by the alveolar-capillary membrane, consisting of thin endothelial and epithelial cells and a minute but expansive interstitial space. This interface between air and blood, only 2 microns in thickness, is the only place where gases may be exchanged effectively.

**Disease Specific Review**

**Chronic Bronchitis**

Chronic bronchitis is a clinical disorder characterized by excessive mucus secretion in the bronchi. It was traditionally defined by chronic or recurrent productive cough lasting for a minimum of three months per year and for at least two consecutive years, in which all other causes for the cough have been eliminated. Today’s definition remains more simplistic to include a productive cough progressing over a period of time and lasting longer and longer. Sometimes, chronic bronchitis is broken down into three types: simple, mucopurulent or obstructive. The pathologic changes consist of inflammation, primarily mononuclear, infiltrate in the bronchial wall, hypertrophy and hyperplasia of the mucus-secreting bronchial glands and mucosal goblet cells, metaplasia of bronchial and bronchiolar epithelium, and loss of cilia. Eventually, there may be distortion and scarring of the bronchial wall.

**Asthma**

Asthma is a disease characterized by increased responsiveness of the trachea and bronchi to various stimuli (intrinsic or extrinsic), causing difficulty in breathing due to narrowing airways. The narrowing is dynamic and changes in degree. It occurs either spontaneously or because of therapy. The basic defect appears to be an altered state of the host, which periodically produces a hyperirritable contraction of smooth muscle and hypersecretion of bronchial mucus. This mucus is abnormally sticky and therefore obstructive. In some instances, the illness seems related to an altered immunologic state.

Histological changes of asthma include an increase in the size and number of the mucosal goblet cells and submucosal mucus glands. There is marked thickening of the bronchial basement membrane and hypertrophy of bronchial and bronchiolar smooth muscle tissue. A submucosal infiltration of mononuclear inflammatory cells, eosinophils and plugs of mucus blocks small airways. Patients who have had asthma for many years may develop cor pulmonale and emphysema.

**Emphysema**

Pulmonary emphysema is described in clinical, radiological and physiologic terms, but the condition is best defined morphologically. It is an enlargement of the air spaces distal to the terminal non-respiratory bronchiole, with destruction of alveolar walls.

Although the normal lung has about 35,000 terminal bronchioles and their total internal cross-sectional area is at least 40 times as great as that of the lobar bronchi, the bronchioles are more delicate and vulnerable. Bronchioles may be obstructed partially or completely, temporarily or permanently, by thickening of their walls, by collapse due to loss of elasticity of the surrounding parenchyma, or by influx of exudate. In advanced emphysema, the lungs are large, pale, and relatively bloodless. They do not readily collapse. They many contain many superficial blebs or bullae, which occasionally are huge. The right ventricle of the heart is often enlarged (cor pulmonale), reflecting pulmonary arterial hypertension. Right ventricular enlargement is found in about 40% of autopsies of patients with severe emphysema. The distal air spaces are distended and disrupted, thus excessively confluent and reduced in number. There may be marked decrease in the number and size of the smaller vascular channels. The decrease in alveolar-capillary membrane surface area may be critical. Death may result from infection that obliterates the small bronchi and bronchioles. There is often organized pneumonia or scarring of the lung parenchyma due to previous infections.

Classification of emphysema relies on descriptive morphology, requiring the study of inflated lungs. The two principal types are centrilobular and panlobular emphysema. The two types may coexist in the same lung or lobe.

**Centrilobular emphysema** (CLE) or centriacinar emphysema affects respiratory bronchioles selectively. Fenestrations develop in the walls, enlarge, become confluent, and tend to form a single space as the walls disintegrate. There is often bronchiolitis with narrowing of lumina. The more distal parenchyma (alveolar ducts and sacs and alveoli) is initially preserved, then similarly destroyed as fenestrations develop and progress.

The disease commonly affects the upper portions of the lung more severely, but it tends to be unevenly distributed. The walls of the emphysematous spaces may be deeply pigmented. This discoloration may represent failure of clearance mechanisms to remove dust particles, or perhaps the pigment plays an active role in lung destruction. CLE is much more prevalent in males than in females. It is usually associated with chronic bronchitis and is seldom found in nonsmokers.

**Panlobular emphysema** (PLE) or panacinar emphysema is a nearly uniform enlargement and destruction of the alveoli in the pulmonary acinus. As the disease progresses, there is gradual loss of all components of the acinus until only a few strands of tissue, which are usually blood vessels, remain. PLE is usually diffuse, but is more severe in the lower lung areas. It is often found to some degree in older people, who do not have chronic bronchitis or clinical impairment of lung function. The term senile emphysema was formerly applied to this condition. PLE occurs as commonly in women and men, but is less frequent than CLE. It is a characteristic finding in those with homozygous deficiency of serum alpha-1 antitrypsin. It has also been found that certain populations of IV Ritalin abusers show PLE.

Bullae are common in both CLE and PLE, but may exist in the absence of either. Air-filled spaces in the visceral pleura are commonly termed blebs, and those in the parenchyma greater than 1 cm in diameter are called bullae. A valve mechanism in the bronchial communication of a bulla permits air trapping and enlargement of the air space. This scenario may compress the surrounding normal lung. Blebs may rupture into the pleural cavity causing a pneumothorax, and through a valve mechanism in the bronchopleural fistula a tension pneumothorax may develop.

Paracicatricial emphysema occurring adjacent to pulmonary scars represents another type of localized emphysema. When the air spaces distal to terminal bronchioles are increased beyond the normal size but do not show destructive changes of the alveolar walls, the condition is called pulmonary overinflation. This condition may be obstructive, because of air trapping beyond an incomplete bronchial obstruction due to a foreign body or a neoplasm. Many lung lobules may be simultaneously affected as a result of many check-valve obstructions, as in bronchial asthma. Pulmonary overinflation may also be nonob-
Asthma Chronic Obstructive Pulmonary Disease Overlap Syndrome (ACOS)

This term is now used to classify 15 to 25% of all patients who have worse outcomes and have a tendency to be younger with more acute exacerbations. The term is newly recognized mainly because of lack of clinical trials addressing both conditions. ACOS as a diagnosis, takes the place of the older term asthmatic bronchitis.

The patients are usually over 40 years of age. Most commonly they are over 40 years of age, age 50 to 60 and were past or current smokers. AsthriTy is present as In asthma. They appear with rhinosinusitis, GERD and limited exercise tolerance. The hallmark symptoms are frequent nocturnal awakenings and exacerbations.

Pathological findings are eosinophils, neutrophils, CD4+T-lymphocytes, CD8+T-lymphocytes, alveolar macrophages, smooth muscle hyperplasia/hypertrophy and emphysema. Peribronchial fibrosis may be present, as well as IgE, IL-4, IL-5, IL-13, IL-beta, IL-8, IL-6, TNF-a, etoxin and proteases.

Pharmacological intervention is usually a mix of both treatments. Other emerging treatment is bronchial thermoplasty. In bronchial thermoplasty, a controlled radiofrequency is applied to the wall of the airway, removing the smooth muscle and replacing it with connective tissue that allows for less spasms to occur. It has shown to be effective for up to five years. Initially the insurance companies were reluctant to cover this procedure, but as with all new technology, as more people are finding it useful, that has improved. The procedure is done much like a bronchoscopy with a device called Alair that has a temperature controlled catheter.

Bronchiectasis

Bronchiectasis means irreversible dilation and distortion of the bronchi and bronchioles. Saccular bronchiectasis is the classic advanced form characterized by irregular dilatations and narrowing. The term cystic is used when the dilatations are especially large and numerous. Cystic bronchiectasis can be further classified as fusiform or varicose.

Tubular bronchiectasis is simply the absence of normal bronchial tapering and is usually a manifestation of severe chronic bronchitis rather than of true bronchial wall destruction.

Repeated or prolonged episodes of pneumonia, inhaled foreign objects or neoplasms have been known to cause bronchiectasis. When the bronchietatic process involves most or all of the bronchial tree, whether in one or both lungs, it is believed to be genetic or developmental in origin.

Mucoviscidosis (cystic fibrosis), Kartagener’s syndrome (bronchiectasis with dextrocardia and panasal sinisitis), and agammaglobulinemia are all examples of inherited or developmental diseases associated with bronchiectasis. The term pseudobronchiectasis is applied to cylindrical bronchial widening, which may complicate a pneumonia but which disappears after a few months. Bronchiectasis is true saccular bronchiectasis but without cough or expectoration. It is located especially in the upper lobes where good dependent drainage is available. A proximal form of bronchiectasis (with normal distal airways) complicates aspergillus mucus plugging.

Advanced bronchiectasis is often accompanied by anastomoses between the bronchial and pulmonary vessels. These cause right-to-left shunts, with resulting hypoxemia, pulmonary hypertension and cor pulmonale.

Etiology and Pathogenesis

Etiology

By far the most common etiological cause of COPD remains smoking. Even after the client quits smoking, the disease process continues to worsen. Air pollution and occupation also play an important role in COPD. Smog and second-hand smoke contribute to worsening of the disease.

Occupational exposure to irritating fumes and dusts may aggravate COPD. Silicosis and other pneumonoconioses may bring about lung fibrosis and focal emphysema. Exposure to certain vegetable dusts, such as cotton fiber, molds and fungi in grain dust, may increase airway resistance and sometimes produce permanent respiratory impairment. Exposures to irritating gases, such as chlorine and oxides of nitrogen and sulfur, produce pulmonary edema, bronchiolitis and at times permanent parenchymal damage.

Repeated bronchopulmonary infections can also intensify the existing pathological changes, playing a role in destruction of lung parenchyma and the progression of COPD.

Hereditary or biological factors can determine the reactions of pulmonary tissue to noxious agents. For example, a genetic familial form of emphysema involves a deficiency of the major normal serum alpha-1 globulin (alpha-1 antitrypsin). A single autosomal recessive gene transmits this deficiency. The homozygotes may develop severe panlobular emphysema (PLE) early in adult life. The heterozygotes appear to be predisposed to the development of centrilobular emphysema related to cigarette smoking. The other better known cause of chronic lung disease is mucoviscidosis or cystic fibrosis, which produces thickened secretions via the endocrine system and throughout the body.

Aging by itself is not a primary cause of COPD, but some degree of panlobular emphysema is commonly discovered on histopathologic examination. Age related dorsal kyphosis with the barrel-shaped thorax has often been called senile emphysema, even though there is little destruction of interalveolar septa. The morphologic changes consist of diluted air spaces and pores of Kohn.

Pathogenesis

The pathogenesis of COPD is not fully understood despite attempts to correlate the morphologic appearance of lungs at necropsy to the clinical measurements of functioning during life. Chronic bronchitis and centrilobular emphysema do seem to develop after prolonged exposure to cigarette smoke and/ or other air pollutants. Whatever the causes, bronchiolar obstruction by itself does not result in focal atelectasis, provided there is collateral ventilation from adjacent pulmonary parenchyma via the pores of Kohn.

It has been proposed that airway obstruction at times may result in a check-valve mechanism leading to overdistension and rupture of alveolar septa, especially if the latter are inflamed and exposed to high positive pressure (i.e. barotrauma). This concept of pathogenesis of emphysema is entirely speculative. Airflow obstruction alone does not necessarily result in tissue destruction. Moreover, both centrilobular and panlobular emphysema may exist in lungs of asymptomatic individuals. It has been reported that up to 30% of lung tissue can be destroyed by emphysema without resulting in demonstrable airflow obstruction. Normally, radial traction forces of the attached alveolar septa support the bronchiolar walls. With loss of alveolar surface in emphysema, there is a decrease in surface tension, resulting in expiratory airway collapse. Additional investigative work continues in an effort to link disease states to pathogenesis.

So much research work is being done currently to identify biomarkers and gene expression. For example, iron has been identified in the IRP2 (Iron-responsive element binding protein) gene and has been associated in two studies as tied to inflammation and emphysema after exposure to cigarette smoke. Although the study was not in humans, it does seem to mean that looking into excessive iron buildup could play a role in developing COPD.
Other studies are looking into the role of Fibulin-5 (FBLN5) in elastogenesis. For example, could regulation of these protein processes provide cures?

Control of Ventilation

A brief description of respiratory control mechanisms will help the nurse better understand how the progression of COPD results in pathophysiologic changes. The respiratory centers impart rhythmicity to breathing. The sensory-motor mechanisms provide fine regulation of respiratory muscle tension and the chemical or humoral regulation that maintains normal arterial blood gases. This will help the nurse to understand why hypercapnia (increased PaCO2) results in the COPDers' extreme reliance on the hypoxic drive.

The reticular formation of the medulla oblongata constitutes the medullar control center responsible for respiratory rhythmicity. The mechanism whereby rhythmicity is established is not clear, but it may be the end result of the interaction of two oscillating circuits, one for inspiration and one for expiration, which inhibit each other. Although medullar centers are inherently rhythmic, medullar breathing without pontine influence is not well coordinated; therefore, pontine as well as medullar centers participate in producing normal respiratory rhythm.

In the pons, a neural mechanism has been identified as the pneumotaxic center. Stimulation of this center leads to an increase in respiratory frequency with an inspiratory shift, whereas ablation of the center leads to a slowing of respiration. The pneumotaxic center has no intrinsic rhythmicity but appears to serve by modulation of the tonic activity of the apneustic center. The latter is located in the middle and caudal pons. Stimulation of the apneustic center results in respiratory arrest in the maximal inspiratory position, or apneusis.

Respiratory muscles, like other skeletal muscles, possess muscle spindles, which, by sensing length, form a part of a reflex loop that assures that the muscle contraction is appropriate to the anticipated respiratory load and required effort. This servo-mechanism facilitates fine regulation of respiratory movements and may stabilize the normal respiration in spite of changes in mechanical loading. Breathing is automatic when the respiratory load is constant or when changes in load are subconsciously anticipated. Thus, because it is anticipated, we are not consciously aware of the increase in expiratory resistance during phonation. Under such circumstances the increase in effort is not sensed because it is appropriate to the expected load.

It has been suggested that signals from respiratory muscle and joint mecano-receptors are integrated to produce a sensation that may reach consciousness when there is this "length tension appropriateness."

Humoral regulation of the medullar centers is mediated by chemosensitive areas in the medulla and through peripheral chemoreceptors. Peripheral chemoreceptors are primarily responsible for the hypoxic drive. These receptors are highly vascular structures located at the carotid bifurcation and arch of the aorta. A diminution of oxygen supply results in anaerobic metabolism in cells of these carotid and aortic bodies. The resulting locally produced metabolites stimulate receptor nerve endings and, through signals conveyed to medullar control centers, lead to increased ventilation. The extremely high blood flow of the chemoreceptors and their almost immeasurable arterial-venous difference make them sensitive to reduced arterial oxygen tension (PaO2) but not to a reduction in oxygen content alone. However, a decrease in blood flow to these chemoreceptor organs, by permitting accumulation of metabolites, results in their stimulation and an increase in ventilation. Very high PaCO2 minimizes receptor stimulation regardless of blood flow.

A decrease in arterial pH also stimulates these peripheral chemoreceptors. The stimulation resulting from an increase in arterial carbon dioxide tension (PaCO2) is probably secondary to the increase in pH. The effect of pH has been attributed to dilatation of arteriovenous anastomoses in the periphery of the chemoreceptor bodies, with resulting reduction in blood flow to the chemosensitive cells. However, the effect of carbon dioxide and pH on respiration is mediated only to a limited extent by peripheral chemoreceptors. Denervation of these receptor organs abolishes the hypoxic drive to respiration but has little effect on the influence on ventilation of carbon dioxide or pH.

Changes in PaCO2 have a profound effect on central chemoreceptors located in the medulla. These are primarily responsible for mediating the hypercapnic respiratory drive. The precise location and characteristics of these central chemoreceptor sites nor their neural connections with the medullar respiratory control centers have been established. The chemosensitive areas appear to be directly responsive to hydrogen ions rather than to carbon dioxide.

Central chemoreceptors are sensitive to changes in pH, and through this mechanism they appear to be specifically responsive to PaCO2. Hydrogen ions themselves do not readily traverse the blood-brain barrier. Under normal circumstances, CO2 plays the primary role in chemical control of ventilation while PaO2 and extracellular pH have lesser roles. Normal subjects increase their ventilation more than two-fold while breathing 5% CO2 gas mixture.

Chronic elevation of PaCO2 (hypercapnia) is found in patients having COPD. The respiratory response to CO2 is markedly diminished in these clients and they become markedly sensitive to their diminished PaO2 (hypoxemia). An exuberant use of oxygen may have dire consequences by removing the dominant respiratory stimulus in these clients.

Applied Abnormalities in Cardiopulmonary Physiology

The normal lung is capable of receiving and distributing a large flow of air and blood to its alveoli. In emphysema, the elastic recoil of the lung decreases with loss of alveolar septa, presumably because the reduced alveolar surface area exerts a lower surface tension. Inspiration lowers alveolar pressure, allowing air to flow into the lungs; the bronchiole dilates when the pressure in the surrounding alveoli is less than that within the lumen of the bronchiole. Conversely, in expiration, the airways are compressed because the alveolar pressure surrounding the bronchiolte exceeds that within the bronchiolar lumen. There is a greater tendency for airflow obstruction during expiration. In emphysema, bronchiolar obstruction due to loss of alveolar structure is irreversible.

The bronchial glands and goblet cells may be hypertrophied, producing excessive amounts of mucus, which frequently obstructs bronchiolar lumina. One aspect of therapy focuses on increasing the fluidity and mobility of mucus. Submucosal edema and cellular infiltration cause a thickening of the bronchiolar wall and narrowing of the lumen. Because vasodilatation often leads to edema, another aspect of treatment is to cause vasoconstriction by means of alpha-adrenergics. The smooth muscle may be hypertrophied in bronchiitis or asthma, narrowing the lumen. Adrenergic drugs are used to smooth the muscle. COPD is usually insidious, existing in an asymptomatic unrecognized form for years prior to the appearance of noticeable dyspnea on exertion. With mild to moderate COPD, bronchiolar obstruction is found in a patchy distribution throughout the lungs. This results in uneven ventilation/perfusion ratios, which will be discussed at the end of this section. The less involved, better-ventilated lung units become insufficient to compensate for the more involved, poorly ventilated units in cases of advanced COPD or superimposed viral or bacterial infections.

Severe arterial hypoxemia is likely to increase production of erythropoietin, which
stistrates the bone marrow causing erythropoiesis. This erythropoiesis may be either useful or harmful. The higher hemoglobin associated with increased O2 capacity is good; but the increased blood volume in the presence of a failing heart is not. Increased blood viscosity causes a harmful resistance to blood flow through the lungs and coronary vessels. Early medicine utilized phlebotomies to treat hypoxia instead of O2. This resulted in a stimulus for increased erythropoiesis causing a snowball effect.

Patients with severe bronchitis have mismatched ventilation/perfusion. This leads to arterial hypoxemia, secondary erythropoiesis, and cor pulmonale with congestive heart failure. They are called blue bloaters due to their cyanosis and edema, or anasarca. A patient with severe emphysema may have decreased cardiac output and a relatively small heart, but as long as he/she can effectively hyperventilate and match ventilation/perfusion, he/she will not develop hypoxemia. They are called pink puffers because they maintain a near normal PaO2 and are hyperpneic.

Auscultation

Auscultation of the lungs provides information about the airflow through the tracheobronchial tree and the presence of fluid, mucus or obstruction of the airway. Vesicular breath sounds are normally heard over the chest. They are soft and low in pitch. Bronchovesicular breath sounds are medium in intensity and pitch and heard over the large, main stem bronchi. Bronchial breath sounds are loud and high in pitch and normally heard over the trachea. One type of bronchial breath sound rarely heard is the amorphic breath sound heard over a thick walled cavity that communicates freely with a large sized bronchus. The sound resembles blowing over the top of a wine bottle. Vesicular breath sounds last longest on inspiration and when airflow to an area is diminished, they may be decreased or absent. Bronchial breath sounds are longest on expiration. Consolidation of lung tissue, as occurs in pneumonia, blocks the passage of air through the affected area and prevents the exchange of sound quality.

Remember that a patient with particularly severe asthma may have a rather quiet chest on auscultation. This is probably because airflow is so slow that it can no longer generate much sound. Breath sounds will also be absent or decreased in COPD. This is caused by lung distention and poor transmission of sound to the chest wall.

Abnormal breath sounds (adventitious or “added”) include rales, rhonchi, wheezes and pleural friction rubs. Rales are noisy murmurs caused by passage of air through liquid.

Moisture causes a sound like soda fizzing, ce-lolophane crinkling, or the sound you hear when you roll your hair between your fingers near your ears. Rales are usually heard on inspiration. Coarse rales may clear after a cough but fine rales are near the bases of long fields rarely do. Rales are sometimes called “crackles.” The crackles of interstitial lung disease, such as fibrosing alveolitis, are typically heard on late inspiration as opposed to crackles from secretions.

Rhonchi are rumbling, snoring or rattling sounds caused by obstruction of a large bronchus or the collection of secretions in a large bronchus. They are most prominent on expiration. Another name for rhonchus is a “wheeze.” Snoring sounds are called sonorous rhonchi, and high pitched musical sounds are called sibilant rhonchi. Wheezes may be audible without a stethoscope.

Pleural friction rubs occur when the pleural fluid that normally lubricates the pleura is decreased or absent. The membranes rub together causing a loud creak or a soft click that resembles a grating sound. They are heard on inspiration and expiration and are associated with pain and splinting.

Ventilation/Perfusion (V/Q) Ratio

Effective gas exchange depends on uniform distribution of function throughout the lung. Ventilation must be distributed to 300 million alveoli through 23 generations of branching airways along with blood distribution through a myriad of capillaries. Even in normal lung function, distribution is not uniform. There is a gravity-dependent gradient of pleural pressure in the upright lung of about 0.3 cm H2O pressure/cm vertical distance. The pleural pressure over a normal adult lung 30 cm in height is about 9 cm H2O more negative at the apex than at the base. Lung units near the lung apex are distended by a greater transpulmonary pressure and are more fully inflated than those at the base.

Blood flow, like ventilation, is least at the apex and increases down the lung. However, alveolar ventilation and perfusion are not evenly matched, so the gradient of perfusion is steeper than that of ventilation. The average V/Q (Ventilation/Perfusion Ratio) is 0.8.

In regions of the lung where the V/Q ratio is increased above normal, wasted ventilation occurs. This has the effect of adding a space that is ventilated but does not participate adequately in gas exchange. An extreme example can occur when perfusion is virtually eliminated, by a blood clot or following ligation of a pulmonary artery.

Ventilation of regions of the lung with high V/Q ratios is partly wasted and contributes to alveolar dead space ventilation. In decreased states, this is not uncommon. It results in hyperventilation and increased work of breathing.

When ventilation is impaired without decreased blood flow or when perfusion continues to non-ventilated regions of the lung, as in atelectasis, there is a decreased V/Q. Gas exchange is extremely impaired or absent and perfusing blood is poorly oxygenated. Hyperventilation can help hypercapnia, but not hypoxemia. The addition of poorly oxygenated blood from areas of low V/Q to normally oxygenated blood acts like a shunt. This “physiologic shunting” must be differentiated from true venous admixture produced by an “anatomic” shunt.

A shunt study can be performed by having the patient breathe 100% O2 for 20 minutes and then obtaining arterial blood gases. True venous admixture will not be changed by breathing 100% O2. Use extreme caution in some patients, however, making sure hypoxic drive is what is keeping them ventilated.

Brief Arterial Blood Gas (ABG) Review

Ventilatory impairment produced by alveolar hypoventilation in COPD leads to various abnormalities of blood gases and pH. Compensatory mechanisms, including increased ventilatory effort, result in normal values in COPD when disturbances of ventilation, perfusion and diffusion are not severe. As the disease progresses, a normal PaO2 may be maintained at rest, but not with exercise. In severe COPD, the PaO2 is low and PaCO2 high at rest, with compensatory increase of serum bicarbonate. It should be noted that the normal ranges for PaO2 decline with age, whereas PaCO2 does not. In practice, it is helpful to know baseline ABGs and compare them with results obtained during acute distress.

It is important to note that renal compensation for acidosis resulting from increased carbonic acid takes five to seven days. Although initial increases in CO2 increase respiratory drive, a further increase in CO2 may profoundly depress the entire central nervous system, including the respiratory center (CO2 narcosis). Carbon dioxide is a powerful cerebral vasodilator, and thus may cause cerebral edema, increased cerebrospinal fluid pressure, and papilledema. (This is the primary reason why hyperventilation is necessary in brain injuries, if COPDers were to receive sedatives while in this state of CO2 narcosis, the result could be fatal.)
The following box shows an example of what happens in Respiratory Acidosis:

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<td>&lt; 7.35</td>
<td>&gt; 45</td>
<td>normal</td>
</tr>
<tr>
<td>Partly Compensated</td>
<td>&lt; 7.35</td>
<td>&gt; 45</td>
<td>&gt; 26</td>
</tr>
<tr>
<td>Compensated</td>
<td>normal</td>
<td>&gt; 45</td>
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Clinical Features of COPD

**History and Physical Findings**

Patients with COPD have at least one symptom in common: undue breathlessness on exertion. Chronic bronchitis is unusual in nonsmokers and is more common in men than in women. Cough is often worse on arising due to accumulation of secretions while sleeping. Wheezing and exercise intolerance are often present and tend to worsen during acute infections of the lower respiratory tract. The sputum may become mucopurulent or purulent. Unless the patient has a hobby or job that requires strenuous exertion, the disease may go unnoticed until quite extensive.

In general, the COPDer appears anxious and malnourished, and complains of lost appetite, use of accessory muscles, muscle atrophy, jugular engorgement, cyanosis, and digital clubbing.

The COPDer’s chest will have increased AP diameter, barrel chest, or hyper-resonant chest, with decreased breath sounds and adventitious breath sounds. Their ventilatory pattern may include paradoxical movement of the abdomen, prolonged expiratory time, active expiration, and pursed lip breathing. In advanced disease, peripheral edema may be present.

Asthmatics who show some degree of persistent airway obstruction and exertional dyspnea are classified as COPD. The accompanying cough is often paroxysmal, and wheezing is severe. Asthma can be brought on by intrinsic or extrinsic factors. An example of an intrinsic factor would be an emotional upset that brings on an attack; extrinsic factors would include specific allergens, etc. Usually by the time an emphysema patient reaches the fifth decade, dyspnea is the primary complaint. Hyperventilation may be present if the patient becomes anxious, but true orthopnea is uncommon unless heart failure is present.

The history may be helpful to distinguish other conditions like chronic pulmonary fibrosis, recurrent pulmonary thrombembolism, polycythemia vera, the diseases of hyperventilation, and myxedema. Aerophagia with gastric distension causes early satiety.

Patients often complain of upper abdominal soreness, distention, and fullness, or even epigastric pain. It is important to note that 20 to 25% of emphysema patients develop ulcers at some stage of their disease.

With deteriorating blood gases, there will be gradual impairment of mental acuity, memory, and judgment, along with headache and insomnia. Patients with cor pulmonale complain of easy fatigability, and may have anterior chest pain and palpitation on exertion. With right heart failure, ankle edema appears and liver enlargement with or without ascites develops.

Clinical features of bronchiectasis principally include a chronic, loose cough with mucopurulent, foul-smelling sputum. In advanced cases, the mucus settles out into three layers: cloudy on top, clear saliva in the middle, and cloudy, purulent material on the bottom. It is frequently associated with chronic paranasal sinusitis. Hemoptysis, occasionally severe, occurs in at least a half of all cases. Advanced cases result in chronic malnutrition, sinusitis, clubbing, cor pulmonale and right heart failure. Physical signs are variable; rales may be present at times. A plain chest film may not be helpful if dilatations of air fluid levels are not present.

(Figure 2 goes here)

Bronchoscopy in bronchiectasis often reveals a deep velvety red mucosa with pus swelling up from areas of involvement. Gram stains may show fusospirochetal organisms and cultures will reveal common mouth flora and anaerobic streptococci or others. Microscopic exam of sputum may show necrotic tissue, muscle fibers and epithelial debris.

Air bronchograms may be noted via ultrasound technology to aid in diagnosis. This shows tubular outlines of the airway caused by surrounding alveolar fluid or inflammatory exudates.

Although pulmonary function tests (PFT’s) are the hallmark for diagnosis of emphysema, experts say that current criteria for airflow obstruction call for a lowering of the normal lower limits.

**Roentgenologic Features**

Correlation among symptoms, physical findings, and the appearance of chest x-rays is often poor in COPD. Films of moderately advanced disease can be read “essentially normal,” but at least they can be used to rule out other complications. In acute asthma, hyperlucency may mask emphysema, but will clear after attack. Emphysema patients will show attenuation of the peripheral pulmonary vasculature. Those with alpha-1-antitrypsin will have scarcity of vascular markings in bases, and hilar shadows present.

Increased prominence of the basal vascular markings is often seen in patients with severe chronic bronchitis or bronchiectasis, with or without emphysema. In patients with pulmonary hypertension and right ventricular enlargement, classically there is prominence of the main pulmonary artery segment, bulging of the anterior cardiac contour into the retrosternal space, and enlargement of the right and left pulmonary artery shadows. In combined right and left ventricular failure, the transverse diameter of the heart is widened, and the basal vascular markings show increased prominence. Comparison with x-rays previously taken may show progressive flattening of the diaphragm, increased radiolucency of the lung fields, increased size of bullous areas, and increased heart size.

The best radiologic criteria for the presence of emphysema is a flattened diaphragm, as seen in lateral view, and an increased depth of the retrosternal space of more than 3 cm between the anterior wall of the origin of the ascending aorta and the sternum. Fluoroscopy in COPD may be helpful because radiolucency of the lung bases tend to persist during forced expiration, in contrast to the increased density seen in normal subjects. Expiratory films should be obtained four or five seconds after the command to exhale is given, to allow time for the full effects of airway obstruction to be registered. CT Scans and modern MRI’s have replaced most need for older lung laminagrams to demonstrate size and location of bullae.

High resolution computed tomography (HRCT) is more sensitive than chest x-ray (CXR). It can be more precise (even at low resolution) on early onset emphysema even before it becomes apparent on pulmonary function tests. Typically the diaphragmatic flattening noted above does not show until advanced stages. One study even said that up to 41% of those with moderate and severe emphysema did not show up on standard films.

CT scans may not be first choice, but they are the definitive choice for diagnosing subclinical and pathologic manifestations of COPD.

Ventilation/Perfusion Scans, a type of scintigraphy using Xenon or tagged albumin, are used to rule out pulmonary embolisms.

**EKG Aspects**

The electrocardiogram is often normal in...
early or moderate emphysema. One of the most frequent changes in COPD is a shift of the P-wave axis toward the right, often greater than +80 degrees in the frontal plane. Observing the P wave in a V1, easily assesses this; it is isoelectric at the +60 degree axis and becomes increasingly negative as its axis moves further to the right, greater than +60 degrees. The Pwaves frequently are symmetrically peaked in leads II, III, and aVF; and when their height is 2.5 mm or more they are classified as “P pulmonale.”

The QRS complexes often show low voltage in both the limb leads and the precordial leads, especially leads V5-6. The mean QRS axis is displaced posteriorly and superiorly and shifted toward the left (clockwise rotation). The frontal electrical axis is often vertical, frequently more than +70 degrees. Superior rotation of the electrical vector manifested by a late R-wave in a VR ABG gives rise to a SI, SII, SIII pattern with an indeterminate mean axis. With more severe rotation, axes greater than minus 30 degrees (left axis deviation) may be seen.

When right ventricular hypertrophy develops as a result of increased pulmonary vascular resistance and pulmonary hypertension, the QRS vector shift anteriorly and to the right. R-waves then appear in the right precordial leads. Complete right bundle branch block is rare. Right axis deviation is associated with advanced right ventricular hypertrophy.

The QRS abnormalities may sometimes simulate those of myocardial infarction, particularly of the inferior portion of the heart. The presence of abnormal pulmonale-type P waves suggests that emphysema is the sole cause of the EKG abnormality.

Treatment of COPD

By far the best ways to treat COPD are to catch it early and to stop smoking. The physician-patient relationship requires realistic expectations to keep the client from becoming too depressed or discouraged. The aim of treatment is to improve or at least to preserve existing lung function and to help the client to adapt to the limitations imposed by his illness. The physician needs to tell the client that the signs of acute infection or respiratory distress. Pulmonary function tests allow the physician to monitor hypoxia non-invasively.

The nurse-patient relationship develops as well, with the nurse often the liaison between the physician and the patient. In early stages, cardiopulmonary rehabilitation is of utmost importance to help the client to understand how to pace himself, control his diet/weight, control climate and avoid irritants. It also helps clients learn about medications (including steroids), breathing exercises, and oxygen therapy. The nurse should teach the client to be aware of symptoms of bronchial infections:

- treatment of cough and sputum retention;
- how to recognize cor pulmonale and congestive heart failure;
- and how to recognize a spontaneous pneumothorax, peptic ulcers, arteriosclerotic and hypertensive heart disease, and pulmonary thromboembolic disease.

The psychological and economic problems of COPD patients call for sympathy as well as wisdom. Suggestions for treatment are sedated work often cause resentment. Many times the impairment of mental acuity and judgment force the work issue. The patient needs to learn new habits in walking and pacing his activities. Mild sedation may be needed to keep the dyspeptic patient from getting more anxious.

Frequent small meals are recommended. Eating usually results in dyspnea and the resultant air hunger and chewing difficulties can exhaust the COPDer. Mental depression may cause anorexia; sometimes drugs such as theophylline or digitalis may be the offender. The recommended low salt diet to reduce edema can make food less palatable. A 3 to 4 g sodium restriction is recommended. Serum zinc tends to run low in many COPDers. Protein is the single, most important nutrient for COPDers on steroids, as they break down more protein than was previously thought.

Healthy individuals consume 36 to 72 calories per day in the energy expenditure of breathing. COPD patients consume an estimated 430 to 720 calories per day, a tenfold increase. They require an average of about 500 calories per day more than people without COPD do. Somewhere between 25 to 65% of COPD patients are plagued with significant weight loss. Of course, steroid use can also cause weight can in some instances.

As mentioned above, mental depression is one of the emerging areas where the impact of COPD is continuing to be studied. A five-year surveillance study recently reported that in a 21 states confusion, memory loss and functional limitations were reported to be greater among those with COPD.

It should also be noted that moving to a warm dry climate is usually of no benefit. It is better to live at sea level because at higher elevations there is reduced oxygen tension. Sensitization to allergens seems to work better in younger patients. Of course, inhaled irritants should be avoided; for example, smoking, fumes, extreme cold or hot air, industrial dusts, etc.

For those bothered with seasonal allergies, the following antihistamines may also be prescribed. They occupy the histamine receptors. There are two types: H1 and H2 receptors. Commonly used drugs are: Cetirizine (Zyrtec), Dimenhydrinate (Dramamine), Diphenhydramine hydrochloride (Benadryl), Fexofenadine (Allegra), Loratadine (Claritan) and Promethazine (Phenergan).

The most common intranasal corticosteroids are Becloethasone (Budesonide (Rhinocort), Ciclesonide (Omnaris), fluticasone (Flonase, Veramyst), mometasone (Nasonex) and triamcinolone (Nasocort AQ). Some of these are available now OTC.

For those suffering from Alpha-1-Antitrypsin deficiency, alpha 1 proteinase Inhibitors may help. Examples are alpha-1-antitrypsin, Aralast, Prolastin, Glassia and Zemaira in the United States, and Trysone in Spain.

After flunisolide was phased out December 31, 2013, many of the breathing medications were replaced by HFA MDI’s (hydrofluoralkane metered dose Inhalers), soft mist Inhaler devices, and DPI’s (“Dry Powder Inhalers”.

List of Commonly Seen Respiratory Medications

**Short Acting Beta 2 Agonists (SABA)**

- Albuterol (Ventolin, Proventil, Proair) available In HFA [HydroFlouroAlkane] MDI [Metered Dose Inhalers] - In UK, you may hear the term Salbutamol used.

- » Xopenex (Levabuteral) HFA MDI
- » Nebulizer Solutions: Albuterol (generic)
- » Albuterol (Accuneb)

**Short Acting Anticholinergics (aka Muscarinic Antagonist)**

- Ipratropium (Atrovent) HFA MDI
- Ipratropium bromide 0.02% (Atrovent) nebulizer solution

**SABA/Short Acting Anticholinergic Combinations**

- Nebulizer Solution: Albuterol & Ipratropium (Duoneb)
- Albuterol & Ipratropium (Combivent Respimat) SMI [soft mist inhaler] spring-loaded

**Phosphodiesterase-4 Inhibitors (PDE-4)**

- Roflumilast (Daliresp) tablets

**Mast Cell Stabilizers**

- Cromolyn Sodium: nasal spray or nebulizer solution

**Methyloxanthines**

- Theophylline (Slow-bid, Theo-Dur) time-released caps or elixir

**Anti-IgE Therapy**

- Omalizumab (Xolair) powder for SQ Injection

**Mucolytics (Breaks the disulphide bond of mucus)**

- Acetylcysteine (Mucomyst) liquid for Inhalation

**Anti-Leukotriene**

© National Center of Continuing Education
• Montelukast (Singulair) tablet, chewable tablet
• Zileuton (Zyflo) tablet
• Zafirlukast (Accolate) tablet

**Systemic Corticosteroids**
• Prednisone tablets, syrup
• Methylprednisone tablets

**Smoking Cessation**
• Nicotine patches, gum
• Buproprion (Zyban, Wellbutrin) tablet
• Varenicline (Chantix) tablet

**Long Acting Beta Agonists (LABA)**
• Formoterol
  » (Performist) Inhalation Solution
  » (Foradil Aerolizer) Dry Powder Inhaler (DPI)
• Arformoterol (Brovanna) Inhalation Solution
• Salmeterol (Serevent Diskus) DPI
• Indacaterol (Arcapta) DPI
• Oladaterol (Steriverdi Respimat) SMI (Soft Mist Inhaler spring device)

**Long Acting Muscarinic Antagonists (LAMA)**
• Ipratropium (Atrovent) MDI (Mist Inhaler spring device)
• Neohaler) DPI device
• Umeclidinum (Incruse Ellipta) DPI
• Oladaterol (Steriverdi Respimat) SMI (Soft Mist inhaler spring device)

**ICS/LABA Combinations**
• Umeclidinum bromide/vilanterol (Anora Ellipta) DPI
• Tiotropium/olodaterol (Stiolto Respimat) DPI device
• Indacaterol/glycopyrrolate (Tudorza Pressair) Breath Actuated DPI
• Oladaterol/glycopyrrolate (Bevespi MDI) DPI device

**Inhaled Corticosteroids (ICS)** *Remember to rinse mouth after use*
• Beclomethasone (QVAR) HFA MDI
• Budesonide (Pulmicort) DPI
• Ciclesonide (Alvesco) HFA MDI
• Fluticasone propionate (Flovent) HFA MDI
• Fluticasone/salmeterol (Advair) HFA MDI or DPI Diskus
• Fluticasone/vilanterol (Breo Ellipta) DPI
• Flunisolide (Maxair) DPI device
• Fluticasone propionate (Flovent) DPI
• Ciclesonide (Alvesco) DPI
• Budesonide (Pulmicort) DPI
• Beclomethasone (QVAR) DPI
• Zileuton (Zyflo) tablet
• Buproprion (Zyban, Wellbutrin) tablet
• Varenicline (Chantix) tablet

Here is a list of discontinued drugs that may sound familiar:
• Tilade
• Intal
• Alupent
• Azmacort
• Aerobid
• Combivent (changed to Respimat)
• Maxair
• Nasalade

Up to 70% of terminally ill patients experience dyspnea. Morphine nebulization has proven to be safe in treating dyspnea associated with end-stage COPD, CHF and lung cancer. It’s effectiveness is believed to be caused by opioid receptors in the lungs and loosening of secretions. Nebulization is not recommended as a route for analgesia, primarily because current administrative technologies result in very small amounts of analgesic being absorbed. Nebulized morphine has a viable bioavailability of 9.35%. There is no clear benefit though over other methods if able to tolerate.

Other new inhalation medications include Tobramycin [TOBI] an aminoglycoside antibiotic. This aerosolized antibiotic is frequently used in cystic fibrosis patients and requires a specialized nebulizer to insure proper particle size. Side effects are voice alteration and tinnitus. All aminoglycosides have potential to cause tubular necrosis, renal failure, deafness due to cochlear toxicity, vertigo due to damage to vestibular organs and rarely neuromuscular blockade. That is why monitoring peak and trough Tobramycin blood levels are so important.

## Assessment Data and Possible Nursing Diagnosis Systems Review

- Patient may report fatigue, exhaustion, malaise, inability to perform basic ADL’s, dyspnea on exertion or at rest, inability to sleep, and the need to sleep sitting up.
- Patient may exhibit swelling of the lower extremities, elevated blood pressure, tachycardia, distended neck veins, faint heart sounds due to increased AP chest diameter, cyanosis, clubbing, or pallor if anemia is present.
- Patient may be anxious, fearful or irritable and complain of poor appetite or weight loss. There may also be reports of decreased sexual libido.
- The COPD patient may appear to have poor hygiene or body odor due to increased shortness of breath with ADL’s.
- They may report air hunger and increased spumt production.
- They may have dependent relationships or insufficient social support.

### Nursing Diagnoses

The following nursing diagnoses are most common for COPD:

1. **Airway Clearance, Ineffective** Related to: Bronchospasm, Viscous secretions, Fatigue
2. **Gas Exchange, Impaired** Related to: Altered O2 supply (obstruction by secretions, bronchospasm, air trapping), Alveolar destruction
3. **Altered Nutrition, Less Than Body Requirements** Related to: Dyspnea, Fatigue, Medication side effects, Sputum production, Anorexia, nausea/vomiting
4. **Infection, High Risk for** Related to: Inadequate primary defenses (decreased ciliary action, stasis of secretions); inadequate acquired immunity (tissue destruction, environmental exposure), Chronic disease process, Malnutrition
5. **Knowledge Deficit Regarding Treatment** Related to: Lack of information or resources, Information misinterpretations, Lack of recall/cognitive limitations

Despite the limitations that the disease of COPD causes, the advent of new therapies and treatment modalities is allowing patients to live more normally and travel more than ever before. Airlines have special ways to help COPD patients by providing special diets, oxygen, wheelchairs and terminal transportation if needed.

As health care professionals, we should be there for support for the COPD patient and his family. While improved survival time is an important goal of therapy; there is growing recognition that improving the length of an individual’s life may not be the only goal. For some, improving the quality of life is much more important. Intimacy is only one small part of the entire quality of life issue. Researchers favor the restrictive term health related quality of life (HRQL), and the term functional status to describe an individual’s ability to function in such diverse realms as physical, social and emotional. HRQL instruments vary from disease-specific measures assessing the severity of symptoms, such as dyspnea or cough, to a global assessment including mood changes, family and social role functioning, activities of daily living, and leisure pursuits. These instruments include the following:

- **Sickness Impact Profile (SIP)**: Â136 items (30 min)
- **Medical Outcomes Study (MOS)**: Â20 items (3 min)
- **Quality of Well Being (QWB)**: Â50 items (12 min)
- **Nottingham Health Profile (NHP)**: Â45
Confusion in COPD

Greenlund, Kurt J., PhD

Biomarkers for COPD Topic

Suggested Readings by Reference and

patients with COPD! scored lower on measures of mental health than prove that this includes memory loss. Only prominent role that depression and emotional issues. For many, it is the social event where they give one another helps with the depression the patients teach each other. The support that airborne Obstruction, Dyspnea and Exercise.

be used. BODE stands for Body-mass Index, airflow Obstruction, Dyspnea and Exercise.

Education is key to rehabilitation. Not only is it what we teach that is important, but what the patients teach each other. The support that they give one another helps with the depression issues. For many, it is the social event where they are totally accepted, oxygen and all!

Lastly and most importantly, one of the most striking findings of HRQL research is the prominent role that depression and emotional dysfunction play in COPD. Recent studies also prove that this includeds memory loss. Only patients with chronic gastrointestinal disorders scored lower on measures of mental health than patients with COPD!

Reference and Suggested Readings by Topic


Environmental Protection Agency (EPA) EPA Safer Choices Program. www.EPA.gov/safer choices.


Vapor Ablation “Segmental Vapor Ablation For Emphysema Improves Pulmonary Function” April 2016, www.lungdiseasenews.com

Bonus: Patient Education

Given the numbers of patients with COPD and a larger aging population, your teaching skills will be challenged.

In acute stages of the disease, the patient’s chronic dyspnea, fear, and exhaustion may all interfere with learning. Proceed steadily but slowly with your teaching. This vital information can make a tremendous difference in the patient’s quality of life, even though progresssion of the disease can’t be halted.

Devote time to coaching your patient in making necessary lifestyle changes. For example, you’ll need to convince him to take preventive measures, participate in prescribed regular exercise, and maintain a proper diet. You may need to encourage him break well-established habits, such as smoking. Getting these points across effectively will help your patient control the dyspnea and avoid exacerbating his condition.

You will also need to clarify various diagnostic tests and treatments, including chest x-rays and physiotherapy, oxygen and drug therapy, and pulmonary function tests.

Included in your teaching strategies your patient will benefit from advise on daily disease management including: recognizing early symptoms of a flare up, avoiding exposure to toxic inhalants and the opportunity of choosing complementary therapies and home remedies to make their symptoms more bearable.

Overview

To begin with, COPD is defined as a group of diseases that reduce the ability to breathe, including chronic bronchitis and emphysema. The most common cause of COPD is smoking, and it has been estimated that anywhere from 15 to 50% of smokers become stricken by COPD. Those who are exposed to environments containing high levels of heavy particulate for extended periods of time are also at risk.

“Chronic obstructive pulmonary disease (COPD) is a term that applies to patients with chronic bronchitis, bronchiectasis, emphysema and, to a certain extent, asthma.”

In the early stages, COPD is characterized by a gradual loss of lung function. The condition can be reversed in the early stages, but most people do not realize they have COPD until lung tissue damage and loss occurs. By this time the condition becomes irreversible, which is why recognizing the symptoms during the early stages is critical.
The symptoms of COPD include:
- Years of smoking, or exposure to heavy pollution such as chemical fumes, grains, cotton, wood, mining dust.
- You suffer bouts of asthma or bronchitis.
- Chronic coughing / heavy phlegm.
- Morning smoker’s cough.
- Colds last weeks instead of days, and you suffer at least one prolonged bout of bronchitis every winter.
- You’re often breathless during ordinary activity.
- Your cough has changed, shallow, as if something is lodged inside your chest.
- You are over the age of 40.
- Additional phlegm is being produced, and your cough becomes moist.

The most important remedy for most people who develop COPD is to quit smoking. Other than that, there are a variety of other natural methods that can help minimize the effects of COPD.

**Natural Methods**

Communicate to the patient the importance of a well-balanced, nutritious diet to compensate for the extra calories expended just to breathe. Because dyspnea and increased sputum can discourage eating and lead to weight loss, help the patient maintain caloric intake. Encourage frequent oral hygiene to stimulate his appetite and enjoyment of food.

If the patient has severe dyspnea, advise him to chew slowly and to eat small, frequent meals to reduce fatigue and swallowing air. Recommend nutritious snacks, including fruit juices and liquid enteral supplements for added calories between meals. Including fresh fruit, vegetables and bran will help prevent constipation.

Consume antioxidants, such as Vitamin C and E, carotene, and selenium. Deriving these nutrients through what you actually eat is most effective. Beta-carotene is found in dark green, orange, red, and deep yellow fruits and vegetables. Beta-carotene offers little protection for those that are still smoking. Foods high in selenium include seafood, organ meats, lean meats, poultry, low-fat dairy products, and whole grains. Selenium offers strong protection for smokers.

Build up energy. This is important since it requires about 10 times as much energy for an individual with COPD to breathe. One way to do this is to stay in good shape by exercising. Another is to eat properly.

Eat 5 or 6 small meals a day instead of 2 or 3 larger ones. This helps prevent bloating and shortness of breath.

Drink plenty of water to keep air passages moist to allow phlegm to be easily coughed up. Soda will not help, since carbonation results in shortness of breath.

**Other Care Measures**

**Avoid pollutants**, which can cause further damage and tissue loss to your already vulnerable lungs. An air purifier will be helpful, since it is difficult to avoid airborne pollutants altogether, especially when inside.

Steering clear of products that may cause lung problems does not mean you can’t have a home that’s fresh and clean. Contact the American Lung Association 1-800-LUNGUSA or go to their website http://www.lung.org to find more information about healthy air.

To get information about specific “current” products for cleaning, please refer to the Environmental Protection Agency website at http://www.EPA.gov. You can click on the “safer choices” tab for cleaning products and also look for the “safer choices” labeling on your products. Please note that not all insurances will pay for environmental air testing in homes.

Here is a list of product lines that are less likely to cause irritation because they are most often free of solvents and are available at most health food stores:
- Seventh Generation, cleaning solutions and laundry detergents
- Ecover, home cleaning products
- Earth Rite, home cleaning products
- Citra Solve, makers of a citrus-based cleaning solution

**Avoid Infections** Avoid contact with people who have colds. They are easily passed through droplets in the air as well as on eating utensils. Wash hands frequently, since cold germs can linger on items he handles or from shaking hands with others. Get extra rest to increase resistance to infections. Ask your healthcare practitioner about a flu vaccine each Fall.

It has also been documented that there is a connection between periodontal disease and increases in acute exacerbations so patient education should also include this topic. COPD falls under the high risk category of those needing the pneumonia vaccine if they are age 19 or older. The CDC website (www.cdc.gov/vaccines) posts yearly schedules for recommendations of vaccinations.

If you are in the high risk category, it is recommended the Prevnar (PCV 13) vaccine be given and follow-up with the Pneumovax shot (PPSV23) a year later. It can be given at the same time as the flu shot, but as a separate injection. The Pneumovax dose my be IM or SQ, Prevnar must be IM. The vaccines are covered by Medicare Part B.

The reason they want the Prevnar first is because of a proven greater immune response.

Patients who were vaccinated with one or two doses of PPSV23 at age 64 or younger still need to have the dose at 65 if it has been five years since the last dose. PCV 13 should be given and then the PPSV23 can be given eight weeks later. If you have previously had the Pneumovax shot and you are at high risk, you need to be revaccinated every five years.

**Fight the Flu** Two drugs, oseltamivir (a pill, Tamiflu) and paramivir (Rapivar) are available. If you suspect that you have already contacted the flu. Rapivar is a once only dose given IV over 15 to 30 minutes. Do not give within two weeks after or 48 hours before the flu vaccine. Most common side effect is diarrhea. Relenza is NOT for people with breathing problems or asthma. Ask your doctor about them, but remember they do not replace the influenza virus vaccine. Be sure to get your shot every fall.

**Learn to Cope** Help your patient explore the impact of COPD on his life and family. Encourage steps that will help him deal with chronic anxiety and depression. Allow him to express his fears about the illness. If he is anxious, suggest relaxation exercises or biofeedback. Help family members deal with the additional stress in their lives. Encourage patient/family to join a support group. As mentioned before, pulmonary rehab is an excellent place for patients to learn how to gain this support.

**Breathing Exercises**

- Hold your breath and breathe deeply frequently, which allows your blood to take in more oxygen. By doing this, make sure you are in an environment with clean air. If the air has many pollutants, this will actually cause you to take in more impurities, which can cause more harm than good.
- Concentrate on pushing out your diaphragm when you breathe. The diaphragm is the muscle between your rib cage and abdomen. Expanding it when breathing allows you to take in more air. Place your hand on your diaphragm to make sure it is expanding when you breathe.
- Use controlled coughing to remove infection-causing phlegm in your lungs. To do this, breathe deeply, hold your breath, and then cough twice. First, loosen the phlegm, and the second to bring it to the back of your throat. Immediately cough up this phlegm to spit it out.
- Avoid anxiety, which results in muscle tightening and shallow breathing.

**Inspiratory Muscle Training (IMT)** This method has recently taken the spotlight for those who present with respiratory muscle weakness. A 6-minute walk or shuttle test should routinely be administered in patient predisposed to respiratory muscle weakness and subjective
Research Information

The ECLIPSE (Evaluation of COPD Longitudinally to Identify Predictive Surrogate Endpoints) study was a large multi-center study that has resulted in more than 50 publications and 75 communications that have helped to identify cellular, blood protein and other blood biomarkers and other genetic factors.

Gene studies were also included involving smoking history, COPD susceptibility, COPD subtypes, emphysema, cachexia and blood biomarkers. Information can be found at http://www.eclipse-copd.com.

Research is continuing to evolve from these studies. In the International Journal of COPD, they noted that microparticles (MP’s) found in the sputum could help reveal the pathogenesis of the disease. This MP phenotype was analyzed and found in all subjects enrolled.

The ECLIPSE study revealed that low levels of Vitamin D are related to emphysema and a low 6-minute walk test (6MWD). The study also indicated low vitamin D also causes increased airway activity and decreased CC16 plasma protein levels, which are known to be associated with lung function decline, emphysema and depression.

Sample Simple Nutrient Plan

- Vitamin C 1,000-2,000 mg.
- Beta-carotene 15,000-25,000 IU
- Vitamin A 5,000-10,000 IU
- Zinc 15-30 mg.
- Selenium 200-300 mcg.
- Vitamin E 400-800 IU

Based on previous studies that indicated a correlation between high lung function levels and a diet rich in various antioxidant vitamins, researchers continue to investigate the relationship between lung function and the intake of magnesium, and vitamins C, E and A with those diagnosed with asthma and COPD.

Dietary Recommendations

No support program for smokers will be as effective as stopping and then working to regain the health lost by smoking. A wholesome diet and nutritional supplements are essential; however, the best program cannot offer immortality to cigarettes.

While the diet is, of course, important, some practitioners believe that for smokers, taking supportive, protective nutrients is even more essential. Many smokers tend to eat more meat, fatty and fried foods, and refined foods than nonsmokers. It is important for smokers to avoid other addictions. Sugar, coffee, alcohol, and meats should be minimized or avoided if possible.

A basic, wholesome diet helps to at least reduce some of the risks of smoking addiction, which may be influenced by nutritional deficiencies. This plan, especially with adequate fruits, vegetables, and whole grains, will help to provide some of the necessary, protective antioxidant nutrients, beta-carotene, vitamins A, C, and E, and selenium, all of which will help lower rates of cancer and other smoker’s maladies. In addition, some raw seeds and nuts, legumes, sprouts, and other proteins should be consumed. Water is an essential nutrient to balance out the drying effect of smoking. A daily intake of two to three quarts is suggested, depending on how much high water content fruits and vegetables, salads, and soups are consumed. Caffeine beverages increase the need for water, as they are also dehydrating. Smoking usually generates a mild acid condition in the body, and an alkaline diet is helpful to balance this. A high-fiber diet also helps in detoxification, maintaining bowel function, and reducing the risks of smoking.

An alkaline diet is even more important during the cigarette withdrawal and detoxification periods. Studies have shown, increased blood alkalinity that results from a diet high in fruits and vegetables, even mainly raw food consumption, helps reduce the craving for and interest in smoking. The alkaline diet is not necessarily a lifelong program, although, it is wise for our diet generally to be more alkaline than acid. During cigarette withdrawal, a vegetarian or raw food diet may be sufficient for the average person to help reduce nicotine craving. This can be used for three to six weeks to aid in the detoxification process. Fasting has also been employed by some smokers to help eliminate their habit. It allows for rapid transitions, but it can also be somewhat intense. It might be reserved for the more durable and strong willed or the overweight or hypertensive smoker.

The vegetarian diet is high in chlorophyllic (green) vegetables and sprouts, grains, fruits, and liquids, such as water, juices, soups, and herbal teas. The raw foods diet is similar, with more seeds and nuts. Eating whole, unsalted sunflower seeds (or carrot or celery sticks) can help replace that hand-to-mouth addiction that is common in smokers; however, we must be careful not to replace nicotine addiction with food addiction.

The diet for detoxification is also low in fat and high in fiber. It is important to keep the energy and bowels moving. The raw foods (and vegetarian) diet helps with both. This includes several salads of leafy greens daily, and some snacks of fruits, vegetables, nuts, or seeds. Some of the high protein algae, such as spirulina and chlorella, also help during withdrawal and detox.

Supplements

Antioxidants help reduce the toxicity of smoke in primary and secondary smokers and also help lessen the free-radical irradiation during the detox period. Vitamin E, 400,Â²I800 IU daily, specifically helps stabilize the cell membranes and protects them and the tissue membranes from the free radical and chemical irritations generated by cigarette smoke. Selenium, as sodium selenite or selenomel, at a level of 200,Â²I300 mcg., supports vitamin E and also reduces cancer potential, which is so much higher with chronic smoking.

Vitamin A reduces cancer risk and supports tissue health, and beta-carotene specifically protects against lung cancer in smokers. Smoking clearly depletes vitamin C levels, probably by increasing antioxidant demands and reducing absorption. Therefore, smokers need regular vitamin C intake to help neutral-
ize the toxins. Supplementing 500–12,000 mg. four or five times daily is recommended. (Note: Both vitamin C and niacin are mild acids, which may increase ulcer risk, as well as nicotine elimination and craving in smokers. Extra zinc, 30–160 mg. a day, like vitamin A, helps protect the tissue and mucous membrane health.

There are many other helpful nutrients needed during smoking and detox. First, we need to support the B vitamins that are more easily depleted in smokers, mainly thiamine (B1), pyridoxine (B6), and cobalamin (B12). The B12 may also help to decrease the cellular damage caused by tars and nicotine. Niacin (B3) helps in opening up the circulation that is constricted with nicotine. It also lowers cholesterol, which may reduce the risk of atherosclerosis. Pantothenic acid may reduce the aging of the skin and support the generally stressful lifestyle. Folic acid should be taken in higher amounts, such as 1,2 mg. daily. Coenzyme Q10 is also helpful in dosages of 30–160 mg. daily. Extra choline may support the brain and memory.

Besides zinc and selenium, other minerals also are important. Magnesium and molybdenum are needed in higher amounts than usual. Magnesium, important for nerve function, could benefit COPD patients by relaxing bronchial muscles. Taking 400 mg/day eased symptoms in adult asthmatics. Copper is needed at levels of 3,4 mg. daily, when used along with a higher zinc intake (60,1 mg.). Zinc also helps reduce cadmium absorption and toxicity. Vitamins C and E, selenium, and L-cysteine also help to reduce cadmium toxicity.

L-cysteine is very helpful to smokers and during detoxification. Along with thiamine and vitamin C, it protects the lungs from smoking damage and from acetaldehyde generated by smoke. It helps reduce smoker’s cough. Glutathione, formed from L-cysteine, is part of the protective antioxidant enzyme system. Heavy smokers might use 250–500 mg. of glutathione, up to 1,500 mg. (500–1,750 mg. more usually) of L-cysteine, with 5,1 g. of vitamin C, 150 mg. thiamine, and the total B vitamins and amino acids to balance the specific ones used.

To prevent obesity, it is very important to be aware of eating properly when stopping smoking. Smoking reduces appetites and the taste for foods and probably increases metabolism as well as nervous energy. It is natural to want to eat more and enjoy food more when not smoking. Over half of ex-smokers will gain weight, and this is more common in heavier (use) smokers. If weight gain is undesirable (many smokers are overweight), a weight-control diet should be instituted as smoking is stopped. Research has shown that smokers crave and eat fewer sweets than nonsmokers. This changes with smoking cessation (the taste buds come alive again), so new nonsmokers need to watch out for this.

The alkaline, high-fiber, low-fat diet is helpful in maintaining weight. Another amino acid, L-phenylalanine, can help reduce the appetite if taken before meals in amounts of 250,500 mg. Because it has a mild tendency to raise blood pressure, this should monitored if blood pressure is of concern. Often, however, the blood pressure drops somewhat with smoking cessation. More choline may improve fat utilization and maintain weight, as may the amino acid L-carnitine. Regular exercise, walking, and getting used to breathing deeply of the fresh air are also part of the new plan. (Adapted from Staying Healthy with Nutrition by Elson M. Haas, M.D. on Healthy.net)

**Home Remedies Can**

“By far the best ways to treat COPD are to catch it early and to stop smoking.”

**Compliment COPD Medications**

The purpose of this section is to give information so that your patients can incorporate natural methods of treatment in conjunction with prescribed medication(s) to alleviate COPD symptoms. Hopefully, these ideas will help reduce suffering, and prevent the need to increase medication dosage.

Take a look at the vast number of bottles of Echinacea, ginkgo, and St. John’s Wort in any supermarket or drug store, and it’s obvious that alternative medicine has gone mainstream. An estimated one-third of US adults use herbal remedies each year. Many people want the best of both worlds: herbal remedies that have been used for centuries, and state-of-the-art, modern medications to help their lungs function at their best.

*Note: Complementary therapies may be used with, but never instead of, the treatments that the physician has prescribed. The “recipes” on the following page may give an extra sense of comfort and well being.*

**On The Horizon**

One of the procedures researchers are looking into now is bronchial thermal vapor ablation (BTVA). With installation of heated water vapor, a local inflammatory reaction is induced, leading to fibrosis and shrinkage with the purpose of targeted lung volume reduction (TLVR). It seems to have been found to improve Forced Expiratory Volume in 1 second (FEV1) and respiratory questionnaire scores in emphysema patients.

The Bronchoscopic Lung Volume Reduction for patients with Heterogenous emphysema and intact Fissures study (BeLieVeR-HiFi) was formed in 2012-2013. It has found that unilateral lobar occlusion with endobronchial valves in patients with heterogeneous emphysema and intact interlobar fissures produce significant improvements in lung functions but there is a significant rate of further complications.

Previously the National Emphysema Treatment Trial (NETT) had reported a rate of 5% morbidity although recent studies have reported lower rates. NETT found the process was most successful when the disease was predominantly in the upper lobe.

Studies are continued to be done on a polymer sealant (manily Aer-Sea) emphysematous lung sealant (ELS) that is being used in lung volume reduction (LVR). Results are varied from improved lung function to deterioration after 6 months. This study needs to be refined. Studies are also being done using coils. Europe had previously done studies but the Medical University of South Caroline (MUSC) is involved in a study called RENEW. They used a RePneu LRVC Implant using a bronchoscopic implant. Ten coils are placed in one lung, then the other lung is treated four months later. The study is continuing.

Targeted Lung Denervation (TLD) is also under study. It works on the premise that the parasympathetic nerves release acetylcholine to induce smooth muscle constriction, so if you disrupt that, breathing will improve.

Lastly, Yale scientists are trying to explore a shared network of genes that exist in COPD and pulmonary fibrosis. If they can find out how this works, maybe it could lead for a way to treat the progression of the diseases.
## Resources

**American Lung Association**  
1840 York Road--Suite M  
Timonium, MD 21093-5156  
1-800-LUNG-USA  
(1-800-586-4872)  
www.lungusa.org

**The National Emphysema Foundation**  
HealthOne Center  
1719 East 19th Ave.  
Denver, CO 80218  
http://emphysemafoundation.org

**Centers for Disease Control and Prevention**  
www.cdc.gov/nccdphp/osh/resource.htm

**Agency for Health Care Policy and Research**  
Office of Health Care Information  
Executive Office Center  
Suite 501  
2101 East Jefferson St.  
Rockville, MD 20852  
1-800-358-9295  
www.ahcpr.gov/clinic/toolskit.htm

**Office of the Surgeon General**  
http://www.surgeongeneral.gov/tobacco/

**Global Institute for Chronic Obstructive Lung Disease**  
www.goldcopd.com

**Indoor Purification Systems**  
http://www.indoorpurifiers.com/sources.htm

**COPD-Support, Inc.**  
http://copd-support.com

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## Stop Smoking Diet

<table>
<thead>
<tr>
<th>Increased Alkaline Foods</th>
<th>Reduced Acid Foods</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Fruits</td>
<td>• Figs</td>
</tr>
<tr>
<td>• Vegetables</td>
<td>• Raisins</td>
</tr>
<tr>
<td>• Greens</td>
<td>• Carrots</td>
</tr>
<tr>
<td>• Lima Beans</td>
<td>• Celery</td>
</tr>
<tr>
<td>• Millet</td>
<td>• Almonds</td>
</tr>
<tr>
<td>• Baked Goods</td>
<td></td>
</tr>
</tbody>
</table>

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### Stop Smoking Brew

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Parts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lemon grass</td>
<td>3 parts</td>
</tr>
<tr>
<td>Dandelion root</td>
<td>3 parts</td>
</tr>
<tr>
<td>Raspberry</td>
<td>2 parts</td>
</tr>
<tr>
<td>Red Clover</td>
<td>2 parts</td>
</tr>
<tr>
<td>Alfalfa</td>
<td>2 parts</td>
</tr>
<tr>
<td>Peppermint</td>
<td>2 parts</td>
</tr>
<tr>
<td>Mullein leaf</td>
<td>2 parts</td>
</tr>
<tr>
<td>Valerian root</td>
<td>1 part</td>
</tr>
<tr>
<td>Catnip</td>
<td>1 part</td>
</tr>
</tbody>
</table>

Simmer dandelion and valerian in water for 10 minutes, then pour into a pot containing other herbs and steep for 15 minutes. Use about 1 teaspoon of root and 1 tablespoon of leaves and flowers per cup. Drink one cup several times daily or as needed for cravings.