Chronic Obstructive Pulmonary Disease

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Abstract:
Chronic obstructive pulmonary disease (COPD) is the fourth-leading cause of death in the United States, and the mortality rate continues to rise. Cigarette smoking is the major cause. COPD is preventable and treatable. Early recognition is important to decrease morbidity and mortality.

Introduction

Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease characterized by airflow limitation that is generally progressive, though it may be partially reversible. It is associated with an inflammatory response of the lung to noxious particles or gases and may be accompanied by airflow hyper-reactivity. It also causes significant extrapulmonary effects. Pathologically, there is a mixture of small airway disease (obstructive bronchiolitis) and parenchymal destruction (emphysema). COPD is manifested clinically as emphysema, chronic bronchitis or both.[1]

Chronic bronchitis is classically defined as the presence of a chronic productive cough on most days for three consecutive months in two successive years. Emphysema is defined morphologically as permanent enlargement of airspaces distal to the terminal bronchioles due to destruction of alveolar walls.

Epidemiology

COPD is the fourth-leading cause of death in the United States, and the mortality rate continues to rise.[2] The prevalence of the disease is significantly higher in those over 40 years of age, in men and in smokers and ex-smokers.[1] The prevalence of COPD has increased in women as smoking rates in women have increased.[3] In 2005, within racial groups, the prevalence was higher in women than men with the exception of the 65 years and older age group.[2] In 2000, the mortality rate from COPD in women eclipsed that in men.[3] The predominant form of COPD is chronic bronchitis.

Pathophysiology and Risk Factors

Cigarette smoking is the major cause of COPD, accounting for greater than 80 percent of cases, though not all smokers develop clinically significant COPD. This suggests that genetic factors modify risk.[4,5] Age of onset of smoking, total pack-years and current smoking status are predictive of mortality from COPD. The role of passive smoking (secondhand smoke) is unclear, though it may contribute to COPD.

Occupational exposure to hazardous airborne substances (e.g., dusts, gases, fumes), when intense or prolonged, is an independent risk factor for COPD and increases the risk of disease when associated with smoking. Indoor pollution from biomass cooking and heating in poorly ventilated dwellings is a risk factor. Urban air pollution is harmful to persons with lung disease, but its role in the etiology of COPD is uncertain.[1]

$Alpha_1$-antitrypsin deficiency is a rare genetic abnormality found in 1 to 2 percent of patients with COPD. Individuals who inherit the Z allele have markedly reduced levels of $Alpha_1$-antitrypsin.[6] Screening for this problem is appropriate in patients who present with COPD prior to age 45 or who have a strong family history of the disorder,[1] and screening may also be considered in patients with unremitting asthma or unexplained hepatic cirrhosis.

Symptoms and Signs

Dyspnea is the cardinal symptom of patients with emphysema and the reason most patients with COPD seek medical care. Chronic productive cough is the key symptom of patients with chronic bronchitis and is often the first symptom of COPD. Initially, it may be intermittent. Sputum is usually mucoid except during exacerbations, when it may become purulent.

Other symptoms of COPD include wheezing, chest tightness and recurrent respiratory infections. Anorexia and weight

loss are common in advanced emphysema. Anxiety and depression also are frequent problems in severe COPD.

COPD is characterized by acute exacerbations manifested by a change from baseline in a patient's dyspnea, cough and/or sputum production with shorter intervals between episodes as the disease progresses.[7] The physical examination is usually normal with early disease. With later-stage disease, hyperinflation is manifested by a barrel-shaped chest, hyperresonance to percussion, decreased breath sounds and distant heart sounds. Crackles and intermittent wheezing may be heard, particularly in chronic bronchitis. In advanced disease, there is dyspnea at rest and there may be cyanosis. The patient often uses pursed lip breathing during expiration, and the accessory muscles of respiration are employed. While sitting, the patient may lean forward and rest on his or her elbows to increase use of the accessory muscles.

Physical findings unrelated to heart failure may include a palpable but normal-sized liver due to chest hyperexpansion and neck vein distention due to increased intrathoracic pressure. When right heart failure is present, increased jugular venous distention, tender hepatomegaly and peripheral edema are typical.

The primary differential diagnoses for COPD are asthma, bronchiectasis, congestive heart failure and other less common chronic inflammatory lung diseases. A discussion of these conditions is beyond the scope of this article.

Diagnostic tests

Spirometry is necessary to make the diagnosis, assess disease severity and monitor response to treatment. In addition, it is indicated in COPD patients who will be undergoing surgery. The most important measurements of airflow include forced vital capacity (FVC), forced expiratory volume in 1 second (FEV_1) and the calculated FEV_1/FVC ratio. Lung volume measurements and diffusing capacity are important in diagnosing restrictive lung disease but are not routinely used in the office management of COPD. It is logical that an active lung infection, active hemoptysis, unstable angina, a recent myocardial infarction and severe debilitation are among the contraindications to spirometry.

Spirometry results are generally compared to reference values based on race, gender, age, and height. An FEV_1/FVC ratio of less than 70 percent indicates airflow limitation. If initial testing indicates airflow limitation (particularly Stage II COPD or greater) a short-acting inhaled bronchodilator should be administered and the testing repeated. A 12 percent or greater increase in the FEV_1 indicates a significant component of reversibility.[1] The spirometric classification of COPD from the Global Initiative for Chronic Obstructive Lung Disease (GOLD) is depicted in Table 1.

Table 1: Spirometric Classification of COPD Severity Based on Post-Bronchodilator FEV_1

Stage I: Mild	FEV1/FVC < 0.70 $FEV_1 \geq 80\%$ predicted
Stage II: Moderate	FEV1/FVC < 0.70 $50\% \leq FEV_1 < 80\%$ predicted
Stage III: Severe	FEV1/FVC < 0.70 $30\% \leq FEV_1 < 50\%$ predicted
Stage IV: Very Severe	FEV1/FVC < 0.70 FEV1 < 30% predicted or $FEV_1 < 50\%$ predicted plus chronic respiratory failure

FEV1: forced expiratory volume in one second; FVC: forced vital capacity; respiratory failure: arterial partial pressure of oxygen (PaO2) less than 8.0 kPa (60 mm Hg) with or without arterial partial pressure of CO2 (PaCO2) greater than 6.7 kPa (50 mm Hg) while breathing air at sea level.

From the Global Strategy for the Diagnosis, Management and Prevention of COPD, Global Initiative for Chronic Obstructive Lung Disease (GOLD). 2007. Available from http://www.goldcopd.org

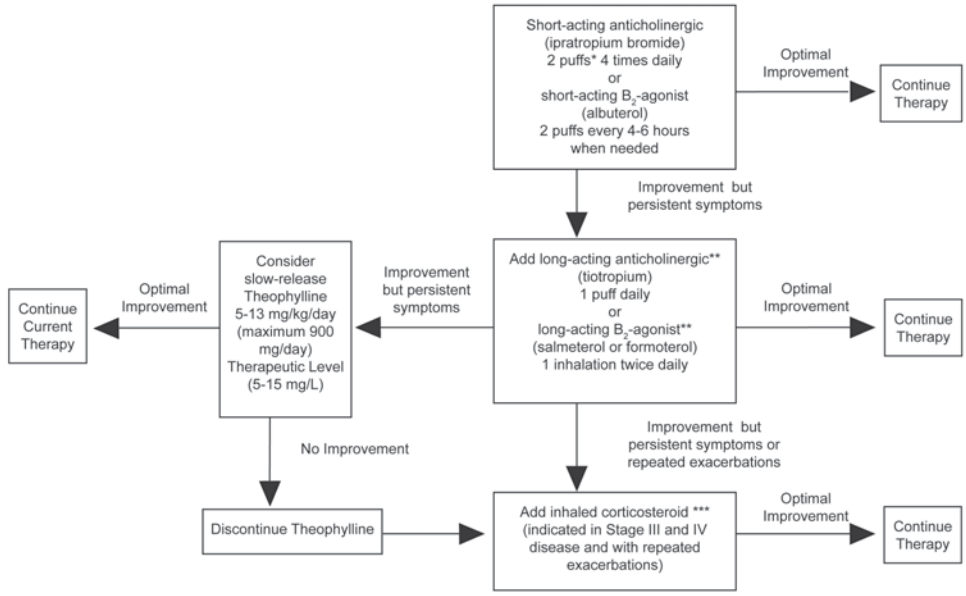
Arterial blood gas measurements are usually normal in early or mild COPD. They should be obtained in patients with an FEV_1 of less than 50 percent of predicted, findings suggestive of respiratory or right heart failure,[1] polycythemia, dysrhythmias or an altered mental state.

Complete blood counts are indicated to screen for polycythemia and when the patient is febrile or a superimposed infection is suspected. Eosinophilia suggests atopy and possibly an element of reversible bronchospasm (i.e., allergic asthma).

Chest X-rays are unremarkable in early disease, but abnormalities are apparent with advanced disease. Findings include hyperexpansion characterized by a low and flat diaphragm, increased anteroposterior diameter of the thorax, hyperlucency, increased retrosternal airspace and a vertically positioned, narrow heart shadow. Bullae may or not be seen and, if present, may only indicate focal severe disease, not necessarily diffuse disease. High-resolution computerized tomographic scans of the chest have a much greater sensitivity and specificity but are not part of routine care unless the diagnosis is in doubt or if a surgical procedure (e.g., bullectomy) is considered.[1]

Electrocardiograms (EKGs) are not routinely indicated, though in patients with long-standing COPD, EKGs may show low voltage, right axis deviation, poor R wave progression, and – when cor pulmonale is present – may

Figure 1: A typical drug protocol for treating chronic obstructive pulmonary disease



* Sometimes higher total daily dosage necessary. Do not exceed 12 puffs in 24 hours.
** May use both long-acting anticholinergic and B_2-agonist if sub-optimal response with single agent
*** If good response, consider combination of a B_2-agonist with a corticosteriod

demonstrate right atrial enlargement (P pulmonale) and right ventricular hypertrophy with strain.

Treatment

Treatment goals include prevention of disease progression; correction of any reversible component; increasing respiratory muscle function; improving exercise tolerance; controlling symptoms (optimum therapy); minimizing and treating exacerbations; treating complications; improving overall health status; and reducing mortality. The various components of treatment are discussed below. A typical drug regimen for treating COPD is outlined in Figure 1. No drugs alter the decline of lung function or modify the natural history of COPD.[1]

Nutrition

Patients with COPD are prone to nutritional deficiencies, and low body weight is associated with reduced pulmonary function, lower exercise capacity and a higher mortality rate, compared to COPD patients with normal nutritional status.[8] Weight loss and the loss of fat mass is primarily the result of inadequate caloric intake relative to energy expenditure, whereas muscle wasting is due to protein breakdown in excess of protein synthesis.[9]

Unfortunately, a meta-analysis showed that appetite enhancing agents and nutritional support had no effect on anthropometric measures, lung function or exercise capacity in patients with stable COPD.[10] Caloric supplementation, however, should be considered when the patient's body mass index is less than 21 or there has been involuntary weight loss of more than 10 percent during the preceding six months or more than 5 percent in the past month.[11,12]

Liquid high-calorie protein supplements can enhance caloric intake. Sodium restriction is appropriate in patients with cor pulmonale or congestive heart failure. Fluid intake should be adequate to maintain good hydration. Consultation with a nutritionist may be helpful in developing a plan tailored to the patient.

Exercise

Exercise has both physiologic and psychological benefits. A combination of muscle strength and endurance (aerobic) training is beneficial.[1,12] Programs should relate to daily activities such as walking and use of the arms. Recommendations for frequency, intensity and duration vary based on baseline functional status, needs and goals. Regular lower extremity exercise improves exercise tolerance, reduces dyspnea and enhances quality of life.[12] Walking, jogging, bicycling, stair climbing and swimming are examples of aerobic exercise. Treadmills, exercycles and stair steppers also are effective devices, as well as cross-country skiing and rowing machines, which provide the added benefit of upper extremity exercise.

Resistance training (e.g., weightlifting) is the mainstay of strengthening and has proven benefits.[12] It is particularly indicated for patients with significant muscle atrophy and weakness.[11] Breathing retraining and respiratory muscle training are discussed below.

Bronchodilators

β-adrenergic agents, anticholinergics, and methylxanthines are the principle bronchodilators used in COPD. β_2-agonists produce sympathetic-mediated bronchodilation, whereas anticholinergic agents reduce parasympathetic-mediated

bronchoconstriction. The methylxanthine, theophylline, is a non-selective phosphodiesterase inhibitor. Bronchodilators are given either on a routine basis to prevent or decrease symptoms or as needed for relief of acute or persistent symptoms. Both β_2-agonists and anticholinergic agents improve exercise capacity and reduce symptoms.[1] Inhaled bronchodilators are preferred. Side effects are less common and resolve more quickly upon cessation than with oral preparations such as theophylline. The potential for theophylline toxicity also is a concern. Though more expensive, regular use of long-acting agents is more convenient and effective (i.e., decreased dyspnea, improved exercise capacity and health-related quality of life) than short-acting bronchodilators.[1,13,14] Clinicians must keep in mind that some patients are slow to respond to long-acting bronchodilators and may require weeks or months on the medications before feeling better.[14] Combining drugs with different mechanisms of action may improve pulmonary function and health status but also can increase cost.[13-18] Therefore, increasing the dose of one agent is an appropriate strategy, as this may offer a similar benefit, assuming side effects are not a problem. Ultimately, the choice of bronchodilator(s) relates to availability, patient compliance, patient response and cost consideration.[1] Patient education is imperative for successful and proper use of an inhalation agent.

β-adrenergic agents are available in inhaled (via MDI = Metered-Dose Inhaler, DPI = Dry Powder Inhaler or nebulizer), oral and subcutaneous preparations. Selective β_2 agents are preferred to minimize the likelihood of β_1 (cardiac) side effects. Inhalation via MDI, DPI or nebulizer-delivered aerosol is preferential to oral preparations, due to their ability to present a high concentration of drug to target receptors with minimal systemic effects. Short-acting (albuterol, levalbuterol) and long-acting (salmeterol, formoterol) agents are available. The usual regimen for short-acting agents given via an MDI is two puffs four times daily, though they can be used on an as needed basis for stable COPD. Salmeterol and formoterol are both inhalation powders given as one inhalation twice daily. They must not be given more often due to their duration of action. Salmeterol and formoterol are excellent choices for the patient with nocturnal symptoms if there is no impairment of sleep.[15] The short-acting agents are required for "rescue" in the scenario of acute bronchospasm with dyspnea. In severe exacerbations, a nebulized aerosol can deliver a high concentration of medication with greater systemic absorption; however, the repeated use of an MDI has equivalent effects when used properly, particularly, when a spacer is employed. Use of a nebulizer is prudent in patients who are in extremes from dyspnea or have altered consciousness.[6] Routine use of a spacer with an MDI is preferable, but particularly in the setting of an acute exacerbation or for patients who are not facile with use of an MDI. The potential for cardiac dysrhythmias necessitates careful monitoring in patients with known cardiac disease, though complications are rare with usual doses. Levalbuterol is the *R*-isomer of racemic albuterol and is usually administered as two puffs every eight hours but may be given every four to six hours on an as-needed basis. Though promoted as safer than albuterol and with less effect on heart rate, its use is controversial among clinicians, given its considerably higher cost. Tachyphylaxis is a potential side effect of β_2 agonists.[14] Oral β_2 drugs should only be prescribed for patients unable to use inhaled agents, and, when employed, should be administered in small doses that are gradually increased. A subcutaneous preparation of terbutaline is available but infrequently used.

Anticholinergic agents are at least as effective as β-adrenergic agents in improving the pulmonary function of patients with COPD. Side effects (dry mouth, metallic taste, urinary retention and glaucoma secondary to mydriasis) have been reported, though such side effects tend to be less troublesome. They are supplied as MDIs, DPIs or aerosols for use with nebulizers. Ipratropium bromide is the only short-acting agent and tiotropium the only long-acting agent currently available in the United States. Anticholinergics have fewer side effects even at high doses and do not cause tachyphylaxis. Some consider them to be the initial agents of choice for maintenance therapy. However, a Cochrane database systematic review found any advantage to be small.[19] Ipratropium has a slower onset of action than short-acting β_2-agonists and is not suited for "rescue" when a rapid response is required. The standard dosage of an ipratropium MDI is two to four puffs four times daily, but higher doses may be required and tolerated in selected patients with severe exacerbations. A combined preparation of ipratropium and albuterol is available as an MDI or as a solution for nebulization, though the same systematic review noted above showed little advantage of the combination drug over a β_2-agonist alone.[19] Tiotropium – a long-acting anticholinergic – is used as one inhalation daily. It has proven to be more effective than ipratropium in improving symptoms and quality of life, as well as decreasing exacerbations and related hospitalizations.[20] A disadvantage is the markedly higher cost. A recent meta-analysis associated anticholinergics with an increased risk of cardiovascular death, myocardial infarction or cerebrovascular accident in patients with COPD.[21] Another study reported its safety, leading to some controversy.

Theophylline is effective in COPD. However, it is not a first-line drug and carries the risk of significant toxicity. It is appropriate to consider adding it in patients who remain symptomatic despite optimum bronchodilator inhalation therapy and in patients who have difficulty with or are non-compliant with MDIs.[22]

Corticosteroids

Corticosteroids given by inhalation are indicated in patients with Stage III and IV COPD. Although they do not modify the natural history of the disease, they do reduce the rate of exacerbations.[1] Treatment of exacerbations with oral or parenteral agents improves dyspnea, reduces treatment failure and the need for additional treatment.[23] There is no evidence to support long-term use of glucocorticoids in stable patients.[1]

For acute exacerbations, oral prednisone can be started in doses of 30 to 40 mg daily for 7-10 days. Long-term treatment is not recommended. Parenteral corticosteroids are often prescribed for acute exacerbations in the hospital setting. In the Veterans Affairs Cooperative Study on the effect of systemic corticosteroids in COPD exacerbations, methylprednisolone 125 mg was given intravenously every six hours for 72 hours followed by oral prednisone tapered for up to 54 days. This clinical trial demonstrated that patients treated with systemic steroids had fewer treatment failures, improved spirometry and shorter hospital stays.[24]

Inhalant preparations include beclomethasone, flunisolide, fluticasone, triamcinolone, budesonide and mometasone. Oral candidiasis and dysphonia are the most common side effects reported with use of a steroid inhaler and can be reduced by use of a spacer and by rinsing the mouth with water after use.

Combination therapy with a long-acting β_2-agonist and a corticosteroid improves symptoms, spirometric indices and quality of life and decreases mean rates of exacerbations.[1,15,18] Fluticasone plus salmeterol as a dry powder inhaler is given as one inhalation twice daily. The newer aerosol preparation is given as two puffs twice daily, as is the combination medication budesonide plus formoterol.

Antibiotics

Antibiotics are usually indicated for acute exacerbations of COPD. Increased dyspnea is the main symptom of an exacerbation of COPD. Although viral infections may cause an exacerbation, increased sputum volume and purulence suggests a bacterial etiology. Influenza, parainfluenza, coronavirus and rhinovirus are common viral pathogens. Bacteria may be either the cause of an infection or represent a secondary infection of what originated as a viral syndrome. The GOLD Program recommends antibiotics in patients presenting with the three cardinal symptoms (increased dyspnea, increased sputum volume, and purulence), in patients with increased sputum purulence and one other cardinal symptom and in patients requiring mechanical ventilation.[1] Antibiotics reduce short-term mortality and treatment failures for patients with an acute exacerbation.[25] The emergence of drug resistance also demands thoughtful consideration in antibiotic selection. The most likely bacterial pathogens include: *Haemophilus influenzae*, *Streptococcus pneumoniae*, *Moraxella catarrhalis* and *Pseudomonas aeruginosa*. *Mycoplasma pneumoniae* and *Chlamydia pneumoniae* also have been reported. First-line, less expensive antibiotics include amoxicillin, sulfa-trimethoprim, tetracycline, doxycycline and erythromycin. Second-line agents offer broader coverage but are more expensive. There is no evidence to support prophylactic or continuous use of antibiotics.[1]

Other Agents

Mucolytic agents are associated with varied results in long-term studies and their widespread use cannot be recommended.[1] Hydration (oral, intravenous) and moisture administered by nebulization may be useful.

Antitussives must be used judiciously as cough has a significant protective role.[1] If prescribed, a nonnarcotic agent such as dextromethorphan should be tried. Codeine and other narcotics should be used cautiously because they may cause respiratory depression and worsen hypercapnia.

Respiratory stimulants are not currently recommended. Nebulized opioids, antioxidants (such as N-acetylcysteine), nedocromil, and leukotriene antagonists have not been sufficiently tested to determine if they are effective and are not currently recommended.[1] Phosphodiesterase-4 (PDE_4) inhibitors are being studied. The role of these selective agents is yet to be determined.[8,26] Alpha-1 antitrypsin therapy is expensive and not available in most countries. It should only be considered in patients with radiographic evidence of emphysema and abnormal lung function related to a severe deficiency of this protease inhibitor.[6]

Psychoactive agents to treat depression, insomnia, or anxiety can be helpful to improve the quality of life for patients with COPD. Benzodiazepines are best avoided due to the potential for suppressing ventilatory drive. Nortriptyline, sertraline, and buspirone are reported to reduce anxiety in COPD patients and buspirone decreased dyspnea in one small study.[27] The antidepressant bupropion

may be of assistance in smoking cessation.[16]

Surgery

Surgery can be considered in carefully selected patients. Bullectomy, which allows greater lung expansion, is effective in reducing dyspnea and improving pulmonary function.[28] In a large multicenter trial of lung volume reduction surgery (LVRS), patients with upper lobe emphysema and low exercise capacity had a greater survival rate at 4.3 years and improved maximal work capacity and health-related quality of life compared with similar patients who received medical therapy.[29] This palliative operation, however, is expensive and associated with significant morbidity and an early mortality rate of approximately 5 percent.[6,8] Lung transplantation is an alternative for patients meeting specific criteria.[1,17,18]

Management Strategies

The physician should develop an individualized plan for the patient and coordinate the care given by others.

Smoking Cessation

Smoking cessation is often the most difficult challenge faced by the patient with COPD but also the most important, as only smoking cessation modifies the natural history of the disease by decreasing the rate of pulmonary function decline.[15-17,30] Please refer to other articles in this special issue on smoking cessation techniques.

Environmental Control

Patients should avoid exposure to secondhand tobacco smoke and remain indoors when air quality is poor. Patients who are sensitive to extremes of humidity and temperature may find that use of a humidifier in the winter and a dehumidifier or air conditioner in the summer improves symptoms. Air cleaners, whether directed against indoor or outdoor generated air contaminants, are ineffective.[1]

Hospitalization

Hospitalization for the patient with COPD should be considered under the following circumstances:

1. Acute exacerbations failing to respond to outpatient treatment, frequent exacerbations, marked increase in intensity of chronic symptoms, or onset of new physical findings (e.g., cyanosis, peripheral edema).[1,9]
2. Changes in mental status.[9]
3. Significant comorbidities including pulmonary (e.g., pneumothorax, pneumonia), cardiac (congestive heart failure, new onset dysrhythmias), and other conditions that may seriously aggravate the patient's baseline status (e.g., rib or vertebral body fracture, severe steroid myopathy.[1,9]
4. Lack of sufficient home support either by family or supplementary home care services.[1,9]
5. Planned surgical or diagnostic procedure requiring analgesics or sedatives that may adversely affect pulmonary function.

Home Oxygen Therapy

The administration of oxygen for 15 hours or more per day to patients with a PaO_2 less than 60mm Hg increases survival.[1,6,31] Continuous oxygen administration is associated with lower mortality.[32] It positively impacts "hemodynamics, hematologic characteristics, exercise capacity, lung mechanics, and mental state."[1] Avoidance of hypoxemia is the goal of supplemental oxygen.[6] Maintaining the PaO_2 of, at least, 60mm Hg and/or an oxygen saturation of, at least, 90 percent is the target.[1] Reversal of hypoxemia supersedes concerns about CO_2 retention, which is rare.[12]

Guidelines for the use of home oxygen therapy are as follows:[1]

1. PaO_2 ≤55 mm Hg or SaO_2 ≤88 percent (waking values), with or without hypercapnia; *OR* PaO_2 between 55 mm Hg and 60 mm Hg or SaO_2 of 88 percent with pulmonary hypertension, peripheral edema suggesting congestive heart failure, or polycythemia (hematocrit >55 percent).
2. The goal is to increase baseline PaO_2 to at least 60–65 mm Hg or SaO_2 > 90 percent at rest, with exertion and during sleep using the lowest liter flow rate possible. Start with 1 L per minute by nasal cannula. The flow rate should be adjusted upward over baseline for exercise and sleep if required to prevent desaturation.

There are several sources and delivery systems for oxygen, and they all have their advantages and disadvantages. A discussion is beyond the scope of this article.

Prior to institution of oxygen therapy, a patient should initially be assessed via arterial blood gases. Monitoring may then be accomplished by either arterial blood gases or by pulse oximetry done periodically.[12] This is particularly important in the patient with worsening symptoms. Pulse oximetry should also be checked with typical exertion (e.g., walking in the hallway) and sleep, as a patient's resting oxygen saturation may be normal. Flow rates should be adjusted accordingly.[1,17,18]

Pulmonary Rehabilitation

Pulmonary rehabilitation is a multidisciplinary, multidimensional approach to care of patients with chronic

respiratory diseases with goals of reducing symptoms, preventing complications, improving quality of life, and achieving the individual's maximum level of independence and functioning in the community.[1,11,12,18] Major components include: education, nutrition counseling, exercise training, breathing re-training, and inspiratory muscle training.

Patients at all stages of disease appear to benefit from exercise training by demonstrating less dyspnea, less fatigue, and improved exercise tolerance independent of a direct effect on pulmonary function.[1,11] It is unknown whether repeated rehabilitation courses enable patients to sustain benefits gained in an initial program.

Breathing retraining is often part of a rehabilitation program with the aim of helping the patient relieve and control dyspnea, and of counteracting physiologic abnormalities such as hyperinflation. Pursed-lip and diaphragmatic breathing slow the respiratory rate,[33] increase tidal volume and diaphragmatic excursion[34] and inhibit premature expiratory flow.[35] Leaning forward and resting the arms on one's thighs or on a table may also help relieve dyspnea.[1] Inspiratory muscle training increases inspiratory muscle strength and endurance, but its effect on symptoms and exercise capacity is not clearly established.[36]

Immunization

Annual influenza vaccination is recommended for all COPD patients. It can reduce serious illness and death by approximately 50 percent. The value of pneumococcal immunization has also been demonstrated.[1] The vaccine covers more than 88 percent of pneumococcal strains. In patients who received the vaccine prior to age 65, a booster should be given after age 65 but at least 5 years after the initial vaccination.[37]

Patient Education

Personalized patient education should address the disease process and provide information about medications (e.g., rationale, side effects, inhaler technique, and home oxygen, if prescribed). Patient education and patient-physician agreement on short- and long-term goals improves adherence to the therapeutic regimen. This should include helping identify a way for the patient to monitor progress toward goals. Sensitive issues such as sexual activity should be addressed. Education of family members, particularly those involved in the patient's care, is also important and, given the considerable adaptation they must make in their own lives, caregivers may need supportive therapy themselves.

Prognosis

Although FEV_1 declines with normal aging, this decline is greater in males than in females. Smokers have a greater progressive decline in pulmonary function proportionate to their cigarette smoking history. Dyspnea with moderate exertion is usually noticeable when FEV_1 falls to about 1.5 L. Dyspnea with any exertion is usually present at a FEV_1 of 1 L. Patients with a FEV_1 of 0.5 L or less are limited to sedentary activity. Besides age, FEV_1 is the best predictor of mortality. The BODE Index, incorporating body-mass index, airway obstruction (FEV_1), dyspnea (Modified Medical Research Council dyspnea scale), and exercise capacity (six-minute walk), is also an excellent prognostic tool for COPD patients.[38] Other indicators of poor prognosis include resting tachycardia, severe hypoxemia, severe arterial hypoxia, severe hypercapnia, hypoalbuminemia, and cor pulmonale.

In severe COPD, episodes of hypoxia lead to the development of pulmonary vascular hypertension and cor pulmonale. Acute respiratory failure, severe pneumonia, pneumothorax, pulmonary embolism, and cardiac dysrhythmias are medical complications often responsible for death. The mortality rate 10 years after diagnosis is >50 percent.

Ongoing Monitoring and Assessment

Regular follow-up is important to the successful management of the patient with COPD. The purpose of such visits includes supporting the patient in smoking cessation, monitoring and modifying as necessary the therapeutic regimen, monitoring changes in pulmonary function, early identification of complications, identification and management of comorbidities, ongoing education, and emotional support. Attention to the pulmonary illness must not entirely supplant addressing other health promotion/disease prevention issues. Advance directives should also be reviewed periodically.

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