American Thoracic Society

MEDICAL SECTION OF THE AMERICAN LUNG ASSOCIATION

STANDARDS FOR THE DIAGNOSIS AND CARE OF PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) AND ASTHMA[1,2]

THIS OFFICIAL STATEMENT OF THE AMERICAN THORACIC SOCIETY WAS ADOPTED BY THE ATS BOARD OF DIRECTORS, NOVEMBER 1986.

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Chronic obstructive pulmonary disease (COPD) and asthma are the major causes of pulmonary disability in the United States, with at least 10 million Americans suffering from COPD and up to 5% of the population afflicted with asthma. Over the past 20 years, major strides have been made in our understanding of the pathophysiology of these two disorders, although there are still large gaps in our knowledge.

While a number of position papers and statements have been promulgated by the American Thoracic Society concerning various aspects of the diagnosis and treatment of COPD and asthma, it was felt that a review of the overall topic was timely. This statement represents the combined efforts of a Task Group appointed by the Scientific Assembly on Clinical Problems of the American Thoracic Society to accomplish this task.

Clearly, we could not cover every aspect of this broad topic nor even provide a detailed review of those areas addressed. We elected instead to concentrate on clinically relevant topics and to provide sufficient data to be useful as a guide as well as to include selected, but in no way exhaustive, references. The first two chapters define the entities and set forth recommendations for diagnosis, hospital admission, and discharge. The remaining four chapters critically review the various facets of therapy. We have noted controversial areas and those where conclusive experimental data are not yet available. In these situations, the committee often decided to take a position on one side or the other based upon the best available information.

CHAPTER 1

Chronic Obstructive Pulmonary Disease

I. Introduction

Thirty years ago, British and North American terminology for the same major lung condition differed: The clinical term "emphysema" in the United States was equivalent to "chronic bronchitis" in Great Britain. Generations of British physicians had recognized chronic bronchitis as a potentially disabling and even lethal affliction that was associated with cigarette smoking and dusty working environments (1). Attention in the United States somehow had been directed more to the profound structural changes of emphysema that were first described by Laennec (2). The morbidity and mortality due to these diseases increased during the 20th century on both sides of the Atlantic.

Improved physiological techniques were applied after World War II that rapidly advanced understanding of pulmonary pathophysiology (3-6). The significance of reduced expiratory airflow was widely appreciated. The first Aspen Conference in 1958 had international representation, and appointed a committee on terminology, which agreed unanimously that emphysema must be defined morphologically (7). British workers met in 1959 to discuss terminology, and published comprehensive suggestions (8) for an orderly transition to the newly recommended terminology. The term "chronic non-specific lung disease" was devised, but proved too awkward (9). It also included a category of "obstructive lung disease" that could occur either with or without reversibility, and with or without emphysema. The present recommendations continue along this line of reasoning.

Chronic obstructive pulmonary disease (COPD) is defined as a disorder characterized by abnormal tests of expiratory flow that do not change markedly over periods of several months observation. The qualification is intended to distinguish COPD from asthma. The airflow obstruction may be structural or functional. Specific causes of airflow obstruction such as localized disease of the upper airways, bronchiectasis, and cystic fibrosis are excluded. Bronchial hyperreactivity may be present in patients with COPD as measured by an improvement in airflow following the inhalation of beta-adrenergic agents or worsening after inhalation of methacholine or histamine (10).

Three disorders are incorporated in COPD: emphysema, peripheral airways disease, and chronic bronchitis. Of these, only emphysema is further classified. Any individual patient may have one or all of these conditions, but the dominant clinical feature in COPD is always impairment, or limitation of expiratory airflow.

II. Definitions

A. Emphysema

The American Thoracic Society previously defined emphysema as an anatomic alteration of the lung, characterized by an abnormal enlargement of the airspaces distal to the terminal non-respiratory bronchiole, accompanied by destructive changes of the alveolar walls (11). This definition was reconsidered and modified by a recent workshop of the National Heart, Lung and Blood Institute. Our committee recommends adoption of the concepts and definitions published by that group in 1985 (12).

Emphysema is defined as "a condition of the lung characterized by abnormal permanent enlargement of the airspaces distal to the terminal bronchiole, accompanied by destruction of their walls, and without obvious fibrosis." Destruction in emphysema is de-

[1] This project was supported by a grant from Boehringer Ingelheim Pharmaceuticals, Inc.

[2] Reprints may be requested from your state or local lung associations.

fined as nonuniformity in the pattern of respiratory airspace enlargement so that the orderly appearance of the acinus and its components is disturbed and may be lost.

It is recognized that emphysema, depending on its severity, may be diagnosed in a variety of ways, including naked eye examination, by examination of an inflation-fixed lung slice using a dissecting microscope (sub-gross examination), or by light microscopic examination of thick (200 to 400 μm) or thin (4 to 6 μm) stained and mounted sections.

Emphysema is recognized as a subcategory of respiratory airspace enlargement which includes:

Respiratory Airspace Enlargement

1. Simple Airspace Enlargement
   a) congenital
   b) acquired
2. Airspace Enlargement with Fibrosis
3. Emphysema
   a) centriacinar
   b) panacinar
   c) distal acinar

In simple airspace enlargement the pattern of the acinus is retained with no evidence of destruction. Congenital airspace enlargement occurs in Down's syndrome or congenital lobar overinflation, whereas acquired forms of respiratory airspace enlargement include compensatory overinflation and the uniform respiratory airspace enlargement in the aging lung. It is still possible that the airspace enlargement of age is not due to age alone, but due to the combination of age and environmental conditions. However, the changes occur in nearly all subjects and it has been suggested that these changes are therefore "normal." The term "aging lung" is preferable to the term "senile emphysema."

A spectrum of airspace enlargement is usually associated with fibrosis of the lung. There is generally no difficulty recognizing honeycombed interstitial pulmonary fibrosis or airspace enlargement associated with fibrosis in granulomatous lesions such as tuberculosis, sarcoidosis, or eosinophilic granuloma.

Three subtypes of emphysema are recognized:

a) Centricinar emphysema. This is also referred to as proximal acinar emphysema because the proximal part of the acinus (respiratory bronchiole) is dominantly involved. There are 2 subdivisions of this form of emphysema. The first is classically associated with cigarette smoking and airflow obstruction, and is also referred to as centrilobular emphysema. Inhalation of coal dust and other mineral dust also results in dilatation of respiratory bronchioles with accumulation of dust-laden macrophages in and around respiratory bronchioles, and has been referred to as focal emphysema. However, in those exposed to coal dust, the term coal pneumoconiosis is preferable.

b) Panacinar emphysema. In this subtype, all components of the acinus tend to be involved about equally. It is the form of emphysema commonly associated with alpha-1-antiprotease deficiency (13). It may also occur in the bases of the lung in patients with centrilobular emphysema, and as an incidental finding in older subjects.

c) Distal acinar emphysema. In this subtype the distal part of the acinus, alveolar ducts and sacs, are predominantly involved. Because of the association of this form with the secondary interlobular septa, it is also known as paraseptal emphysema; the distal acinus also abuts on pleuras, vessels, and airways, and the emphysema may be worse in these regions.

Additional types of emphysema have been suggested, but considerable overlap exists even with the types already described, and there seems to be little reason for further subdivisions. When emphysema becomes severe, it is difficult to classify, and expert pathologists often disagree on the classification of such emphysematous lungs.

Emphysema severity as assessed morphologically is the single best correlate with an index of airflow obstruction such as the forced expiratory volume in one second (FEV_1) (14–16). Patients who have significant physical impairment due to COPD usually exhibit emphysema of at least moderate severity when examined postmortem. Occasionally, such patients have only minimal emphysema (17), and, rarely, it is absent (18).

B. Peripheral Airways Disease

A variety of morphologic abnormalities have been identified in the peripheral airways of patients with COPD. These include inflammation of the terminal and respiratory bronchioles, fibrosis of airway walls with narrowing, and goblet cell metaplasia of the bronchiolar epithelium (19). The distribution and severity of these changes varies considerably among individuals. Structure-function correlations suggest that these lesions contribute to airflow obstruction in severe COPD, but that their importance is secondary to that of emphysema (14–16). In persons at risk for developing COPD (e.g., cigarette smokers), pathological changes in the peripheral airways appear to precede the development of emphysema (20). It has been suggested that inflammation and other changes in the peripheral airways may be responsible for subtle abnormalities in pulmonary function tests that are not associated with physical impairment and that these physiological and pathological abnormalities may represent "early, or preclinical" COPD (15, 21). It is emphasized that these relationships remain unconfirmed by long-term studies, and that their clinical relevance remains uncertain.

C. Chronic Bronchitis

Chronic bronchitis, as previously defined, refers to "the condition of subjects with chronic or recurrent excess mucus secretion into the bronchial tree." Chronic was defined as "occurring on most days for at least three months of the year for at least two successive years" (8). The excess secretion should not be brought about by other diseases such as bronchiectasis or tuberculosis. Excess mucus secretion was empirically recognized as the production of any sputum, whether expectorated or swallowed, and in most instances sputum production is accompanied by chronic cough. Although not explicitly stated in this definition, it was generally held that excess mucus production is an important cause of airflow obstruction (19), and "chronic bronchitis has been commonly used to mean expiratory airflow obstruction."

Many patients with COPD have excess sputum production as well as hyperplasia of the mucus glands of the trachea and large bronchi. Both abnormalities have been linked etiologically to cigarette smoking. However, available evidence indicates that these effects of cigarette smoke are independent of those which cause airflow obstruction. Numerous structure-function correlative studies have failed to identify a close relationship between airflow obstruction and mucus gland hyperplasia (6, 8, 22, 23). More importantly, longitudinal population studies have failed to identify an independent effect of cough and excess sputum production upon the development of airflow obstruction (1, 24, 25).

III. Diagnosis

A. Clinical Assessment

A complete history and physical examination should be performed during the initial assessment of each patient suspected of having COPD, and repeated on those occasions when the condition of the patient changes (e.g., hospitalization). Limited histories and physical examinations should be performed at intervals to evaluate the course of the disease and the response to therapy.

Characteristically, COPD affects middle-aged and older persons. The dyspnea due to COPD cannot be reliably distinguished from that due to other causes, and is frequently associated with cough, wheezing, sputum production, and recurrent respiratory infections. Occasionally, dyspnea is the only symptom of COPD. In this situation it is insidious in onset and progressive in severity. Long-term cigarette use is the principal identified cause of COPD, but these disorders do not occur exclusively in cigarette smokers and the majority of smokers do not develop clinically manifested lung disease (26). Inhaled toxins encountered in the workplace or in the environment pose additional risk factors for the development of COPD and a history of such exposures should be sought. The inherited deficiency of plasma alpha-1-antiprotease renders the patient more susceptible to the damaging effects of cigarette smoke and predisposes to the early development of COPD (13, 27, 28).

Physical examination of patients with COPD may reveal signs of lung overinflation, increased respiratory muscle effort, altered breathing patterns, and abnormal breath sounds. Wheezes, especially on forced expiratory and diminished breath sounds, may be detected by auscultation. One or more of these

physical signs are usually present in patients with advanced COPD, but the changes on physical examination may be sufficiently subtle as to be overlooked even in the presence of moderate airflow limitation. The need for consultative services may arise anytime the condition of the patient deteriorates.

B. Laboratory Tests

1. Roentgenographic examination. A plain chest roentgenograph in posterior-anterior and lateral projections is necessary for the evaluation of patients with suspected COPD since the presence of regional hyperlucency and vascular attentuation confirm the existence of emphysema. With severe emphysema, overinflation is present often, and bullous lesions are fairly common. It is pertinent, however, that roentgenographic studies have limited sensitivity for the detection of emphysema, and that the correlation of roentgenographic abnormalities with the severity of airflow obstruction or of anatomic emphysema is imperfect. Specialized studies such as computed tomography are not usually necessary in patients with uncomplicated COPD.

2. Pulmonary function testing. Spirometric evaluation establishes the diagnosis of COPD. Testing should be performed by methods, and with instrumentation, that conform to standards established by the American Thoracic Society (29). The spirometric abnormalities associated with COPD consist of a reduction in the forced expiratory volume in one second (FEV_1) and in the ratio of the FEV_1 to the forced vital capacity (FVC). Many other parameters may be calculated from the spirogram (30, 31), but there is no evidence that they provide useful diagnostic information beyond that contained in the FEV_1 and FVC. It is desirable to perform spirometry in all patients who have unexplained dyspnea and/or in whom COPD is suspected. It has been advocated that all individuals at risk for developing COPD (e.g., habitual cigarette smokers) be screened regularly by spirometry to detect mild abnormalities with the rationale that severe disease might be prevented by smoking cessation and early treatment measures. The efficacy of such programs has not been demonstrated.

Repeat spirometric testing following medications (e.g., bronchodilators, corticosteroids) should be performed to determine to what extent the disease is reversible and to provide guidelines for rational therapy. The failure of forced expiratory flows to improve acutely after bronchodilator inhalation does not preclude a long-term beneficial response to either bronchodilators or corticosteroids (10, 30, 32). The response of spirometric tests to the inhalation of bronchoconstrictor substances (e.g., methacholine) have been useful in asthma but their use in the diagnosis and management of COPD has not been defined, and a role seems unlikely.

COPD is frequently associated with an increase in total lung capacity and residual volume, and a reduction in the diffusing capacity for carbon monoxide (D_{LCO}) (33). The measurement of lung volumes and of the D_{LCO} may be helpful in the initial evaluation of patients suspected of having COPD. In subsequent follow-ups, forced expiratory spirometry alone usually suffices to demonstrate the response to therapy, or to explain symptomatic deterioration.

COPD is also associated with abnormalities in lung mechanics (e.g., compliance, airflow resistance), and abnormalities of various tests of ventilation distribution (e.g., the single breath nitrogen washout test) have been described. The clinical relevance of these tests in the diagnosis and evaluation of COPD is unsettled, and their routine use is not recommended.

A reduction in exercise tolerance is commonly found in COPD, and routine evaluation of exercise capacity is unnecessary. Such an evaluation may be indicated, however, when considering the need for supplemental oxygen therapy or when looking for additional causes of disability in patients whose exercise tolerance seems out of proportion to the limitation of airflow.

Any impairment in the efficiency of oxygen uptake or carbon dioxide elimination by the lung can be detected by analysis of the arterial blood. COPD is characteristically associated with hypoxemia of varying severity and, in advanced stages, with hypercarbia. The efficacy of supplemental oxygen for patients with a defined degree of hypoxemia has been established. Arterial blood oxygenation should be assessed directly by measurement of Po_2 or indirectly by oximetry in all patients with moderately severe airflow limitation (e.g., FEV_1 below 1.5 L) at the time of initial evaluation, and subsequently, at appropriate intervals. The adequacy of therapy in those patients receiving oxygen should be documented by repeated analyses. In patients hospitalized for respiratory insufficiency, frequent measurements of arterial blood gases may be necessary to assess the adequacy of ventilation and oxygenation, and to monitor acid-base balance. The methodology for measuring arterial blood gases and for performing ear oximetry should conform to accepted laboratory standards.

3. Additional laboratory tests. The need for other laboratory investigations in patients with COPD is defined largely by special circumstances and by complicating clinical conditions. The detection of secondary erythrocytosis from periodic measurements of hemoglobin or hematocrit levels suggests chronic hypoxemia and represents an indication to assess the need for oxygen therapy. Additional details about nocturnal hypoxemia are provided in the section on oxygen therapy. The presence or evolution of changes on the electrocardiogram consistent with right ventricular enlargement suggests the need for arterial blood gas analyses and supplemental oxygen.

Many patients with severe COPD experience recurrent illnesses characterized by increased cough and expectoration of purulent sputum. Although suspected of being infectious in origin, the precise etiology of these episodes remains speculative. In the absence of clinical or radiographic signs of pneumonia, bacterial or viral cultures of sputum usually provide little useful information.

In a very small percentage of cases, COPD is associated with and is thought to result from a severe deficiency in the plasma level of alpha-1-antiprotease. Deficiency of this inhibitor permits the early development of panacinar emphysema (13, 27, 28, 34). This genetic disorder should be suspected in patients who develop severe COPD at a relatively young age, especially if they have affected siblings or parents, and have smoked sparingly or not at all. The diagnosis can be made by measurement of the serum alpha-1-antiprotease levels specifically, or by determining that the tiny sharp peak in the alpha 1-globulin region of the plasma electrophoretogram is absent. Quantitation of the alpha 1-globulin fraction as usually reported from the automated densitometer readout of the serum protein electrophoretogram is worthless for diagnosing the deficiency since it is virtually always normal. Specific phenotype determinations are desirable but not essential. At the present time, replacement therapy has not been completely assessed, and is not generally available. Thus, the practical implication of establishing this diagnosis, aside from admonishing against smoking, relates to its possible use in genetic counseling. Modestly reduced levels of alpha-1-antiprotease, associated with the heterozygous deficient phenotype, do not pose a clear-cut risk of premature COPD (35).

IV. Indications for Hospital Admission

The principal indications for hospitalization of the patient with COPD include: (*1*) acute exacerbation of symptoms such as markedly increased dyspnea, cough, and sputum production that have not responded to adjustments in ambulatory care, (*2*) acute respiratory failure characterized by respiratory distress, hypercarbia, or worsening hypoxemia, (*3*) acute cor pulmonale with dependent edema, further impairment of exercise capacity, and hypoxemia, (*4*) complications of COPD such as acute bronchitis or pneumonia, (*5*) the performance of invasive diagnostic procedures on the lung such as bronchoscopy, transbronchial biopsy, or needle aspiration of nodules, (*6*) the need for surgery or other procedures that require significant amounts of analgesics, or anesthesia, and (*7*) diseases that might not require hospitalization by themselves, but that in the presence of severe COPD represent a significant risk to the patient.

A continuing program of education is an important hospital function. Whenever possible the patient must know the schedule of each medication and understand its purpose. Guidelines for clinical response in the hospital include improvements in symptoms and signs, as well as in the results of spirometry and arterial blood gas analyses.

V. Discharge Criteria

Criteria for hospital discharge rest with improvement to the point that the patient is able to care for his personal needs and manage his medication, or that these requirements can be arranged for outside the hospital.

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CHAPTER 2
Asthma

I. Definition

Asthma is a clinical syndrome characterized by increased responsiveness of the tracheobronchial tree to a variety of stimuli. The major symptoms of asthma are paroxysms of dyspnea, wheezing, and cough, which may vary from mild and almost undetectable to severe and unremitting (status asthmaticus). The primary physiological manifestation of this hyperresponsiveness is variable airways obstruction. This can take the form of spontaneous fluctuations in the severity of obstruction, substantial improvements in the severity of obstruction following bronchodilators or corticosteroids, or increased obstruction caused by drugs or other stimuli. Histologically, patients with fatal asthma have evidence of mucosal edema of the bronchi; infiltration of the bronchial mucosa or submucosa with inflammatory cells, especially eosinophils; and shedding of epithelium and obstruction of peripheral airways with mucus.

II. Diagnosis

The diagnosis of asthma can occasionally be confusing because of its overlap with COPD. In addition, the diagnosis of asthma is occasionally confused with other causes of airway obstruction such as tumors, foreign bodies, laryngospasm, or even cardiogenic pulmonary edema. Patients with COPD may have significant reversibility after treatment and patients with asthma may develop airflow obstruction with little to no reversibility. The separation of these overlap patients is often arbitrary and difficult, and from a clinical standpoint probably not important unless the diagnosis has therapeutic implications, i.e., the bronchospasm results from a specific and avoidable etiologic agent.

A. Clinical Assessment

The symptoms of episodic cough, wheezing, and dyspnea suggest a diagnosis of asthma. The history should explore these symptoms in detail including: (*1*) family and personal history of allergic disease, (*2*) age at onset of symptoms, and frequency and severity of attacks, (*3*) known provocative stimuli (table 1), and (*4*) prior pharmacologic and immunologic therapy. Initial symptoms may be a vague heavy feeling or tightness in the chest accompanied in the allergic patient by rhinitis and conjunctivitis. The patient may complain of a nonproductive cough followed by wheezing and dyspnea. Although initially nonproductive, the cough frequently does become productive of a viscous, mucoid sputum that may contain casts of the distal airways or may appear purulent. A subset of patients with asthma are characterized by recurrent or chronic nonproductive cough without any overt wheezing (1).

In the asymptomatic patient, the examination of the chest may be normal, although examination of the eyes, ears, nose, and throat may reveal concomitant serous otitis media, conjunctivitis, rhinitis, nasal polyps, paranasal sinus tenderness, and signs of postnasal drip, including pharyngeal mucosal lymphoid hyperplasia. In mild asthma, wheezing may only be detected on forced expiration. With increasing degrees of severity, wheezing may

TABLE 1

STIMULATORS OF BRONCHOCONSTRICTION

A. Nonspecific
   1. Exercise
   2. Cold air
   3. Environmental pollutants and irritants
   4. Pharmacologic agents, i.e., histamine, cholinergic agonists
   5. Reflux esophagitis

B. Specific
   1. Aspirin and all nonsteroidal anti-inflammatory drugs (NSAID's)
   2. Occupational antigens
   4. Ingested antigens
   5. Beta-adrenergic antagonists

be heard on quiet expiration and on inspiration. The findings of severe airways obstruction include restlessness, agitation, orthopnea, tachypnea, breathing through pursed lips with a prolonged expiratory phase, using accessory muscles of respiration, diaphoresis, coughing, audible wheezing, and difficulty speaking. In the acutely ill asthmatic, the abatement of wheezing may occur with increasing severity of airways obstruction and must not be taken as a clinical sign of improvement. The evaluation of the blood pressure may reveal that the patient has a widened pulse pressure, and a pulsus paradoxus (10 mm Hg or greater) may be present. The latter sign is a relatively reliable indicator of the severity of the asthma, the FEV_1 almost always being less than 40% predicted in this situation (2). The presence of fever is indicative of an infectious complication such as pulmonary infection. The thorax is often hyperinflated.

Patients with asthma and persistent sinusitis and nasal polyposis with or without middle ear disease frequently benefit from an evaluation by an otorhinolaryngologist. Aggressive management of sinusitis and correction of upper airway obstruction may improve the asthmatic condition. Patients with poorly controlled asthma requiring hospitalization should be evaluated by a specialist in pulmonary disease or allergy. The accurate recognition of the importance of allergic disease in the asthmatic syndrome may require consultation with a physician skilled in testing for allergic diseases.

B. Laboratory Tests

1. Pulmonary function tests. Spirometric studies of pulmonary function are valuable both in the diagnosis of asthma and in assessing the severity of the disease and the response of therapy (3). Spirometry and peak flow measurements are also useful on a regular basis during outpatient management. All measurable parameters of pulmonary function may be within normal limits when the patient with asthma is in remission. If the diagnosis is suspected, bronchial provocation testing utilizing a cholinergic agonist, histamine, cold air, or specific antigens or industrial agents may demonstrate significant airways obstruction with a quantifiable stimulus known to be tolerated by a normal individual (4). Induced airways obstruction may be severe and delayed for up to 24 h. Hospitalization may be warranted when this approach is taken (see INDICATIONS FOR HOSPITAL ADMISSION). When preexisting airways obstruction is noted, bronchial provocation is contraindicated.

The typical abnormalities noted with spirometry in the asthmatic patient include a reduction in FEV_1, peak expiratory flow rate, FEV_1/FVC ratio, and an increase of 15% or greater in the FEV_1 in response to a bronchodilator. (Patients with asthma may not improve their FEV_1 in response to bronchodilators during episodes of severe airways obstruction.) Abnormalities in lung volumes include a decreased vital capacity, an increase in functional residual capacity, total lung capacity, and especially in the residual volume; however, it is not necessary to measure lung volumes in order to make the diagnosis of asthma.

2. Arterial blood gases. The measurement of arterial blood gases may be helpful in determining the severity of disease in the hospitalized patient. Hypercapnia and respiratory acidosis implies severe disease with FEV_1 of less than 15% of predicted (5). Under these circumstances, frequent monitoring of arterial blood gases is essential in patient management. Signs and symptoms of hypoxemia, such as cyanosis, are unreliable and should not be substituted for actual blood gas determinations. With milder bronchospasm, the arterial blood gases usually reflect a respiratory alkalosis, with a near normal P_{O_2} and a widened alveolar-arterial oxygen gradient. In these milder cases the P_{O_2} and P_{CO_2} are relatively insensitive indicators of airways obstruction (6). Normalization of the pH and P_{CO_2} in the face of a falling P_{O_2} usually indicates worsening obstruction with an FEV_1 less than 25% of predicted.

3. Blood leukocytes. Peripheral eosinophilia is common in both allergic and nonallergic forms of asthma; consequently, this parameter cannot be used as a differentiating point between the two. Values of 5 to 15% of the total white blood cell count are common. Total eosinophil counts provide a more accurate measure of peripheral eosinophilia.

Although leukocytosis may suggest the presence of infection, leukocytosis with marked eosinophilia (greater than 3,000 per cubic mm) should raise the possibility of another diagnosis, such as Loffler's syndrome, the hypereosinophilic syndrome, allergic bronchopulmonary aspergillosis, or Churg-Strauss allergic granulomatous angiitis.

4. Sputum. Grossly purulent sputum may reflect eosinophilia rather than PMNs associated with infection. In this situation, microscopy may document eosinophilia, Curschmann's spirals, and Charcot-Leydon crystals, all consistent with the diagnosis of asthma.

5. Electrocardiography. The electrocardiogram is of little value in the diagnosis or management of asthma. ECG changes are usually noted only in severe acute attacks of asthma, are nonspecific in nature, and include sinus tachycardia and rarely ventricular strain pattern and right axis deviation.

6. Radiology. The chest radiograph is not helpful for the diagnosis of asthma or for determining the severity of the acute attack (7). It may be helpful in evaluating potential complications of asthma, such as rib fractures, pneumothorax, pneumomediastinum, atelectasis, and pneumonia. Paranasal sinus films may be of use in evaluation of the patient thought to have concomitant allergic rhinitis and sinusitis.

7. Measurement of serum IgE. A large proportion of the allergic asthmatic population has normal IgE levels, and many conditions other than asthma are associated with elevated IgE levels. Therefore, the usefulness of obtaining a serum IgE measurement in pulmonary conditions other than allergic bronchopulmonary aspergillosis has not been established.

8. Detection of IgE antibody. Tests to detect antigen-specific IgE are indicated when the asthma is thought to be due to an identifiable and avoidable substance.

Bronchial provocation testing with specific antigens (8) can also be used to demonstrate bronchial reactivity and is useful in the evaluation of (*1*) the asthmatic patient with intermittent episodes of asthma who presents for evaluation in the asymptomatic stage, and (*2*) a patient with suspected occupational asthma, since the use of skin testing in occupational asthma is complicated by a lack of specific antigens and the nonspecific irritating effect of the available antigens on the skin.

Immediate-type hypersensitivity skin testing remains the most important tool for the detection of IgE antibody and confirms the clinical suspicion of an allergic component to the patient's asthma as elicited by the history. Skin tests, which are done by prick, scratch, or intradermal methods, must be interpreted in the light of a well-taken history, as false positive results may occur. A diagnosis of specific allergy should rarely be made in the absence of a correlating positive history. Skin testing is safe, sensitive, and useful for the evaluation of allergy to inhalant aeroallergens that may be a trigger in airways obstruction.

The RAST (Radioallergosorbent test) assay or ELISA (enzyme linked immunosorbent assay) permit the *in vitro* evaluation and semiquantitation of antigen-specific IgE antibodies in serum (9). These tests correlate well with clinical provocation testing and are free of

the risk of systemic reactions. They may be useful when the patient is currently receiving forms of symptomatic therapy that alter the interpretation of skin tests. Disadvantages include expense, a lag in the time from testing to the availability of results (at least 48 h), a lesser availability of potential antigens that may be utilized compared with skin testing, a decreased sensitivity, and the potential misuse of these assays by physicians not engaged in the evaluation of patients with allergic disease.

III. Indications for Hospital Admission

A. Diagnosis

In general, the diagnosis of asthma or asthmatic syndromes can be firmly established in outpatients. However, when inhalational challenge testing with environmental allergens or occupational substances are to be administered and there is reason to believe a late response (one which occurs 4 h or later after inhalation) may occur, hospitalization may be required to firmly and safely establish a diagnosis (7, 10). The hospital facilities are used in this circumstance to confirm and quantitate obstructive airways dysfunction at a time of day that may preclude testing as an outpatient. Another indication for hospitalization is to establish a diagnosis of asthma when diagnostic challenge testing is planned in patients with possible or probable complicating medical illnesses, such as cardiac disease, where close monitoring of cardiopulmonary status is advisable.

B. Treatment

1. Treatment of the primary disorder. Hospitalization for the treatment of asthma is indicated for the acute onset of symptoms or physiological changes that are so severe as to preclude successful initial management as an outpatient or in an emergency room (1-5, 7). This indication is more likely to occur in patients in whom the diagnosis of asthma had not been previously established, in patients who have not previously required treatment, or in patients whose treatment requirements have recently changed. Another indication is exacerbation of symptoms in individuals who are undergoing therapy as outpatients but in whom the control of symptoms or physiological changes is such that outpatient management is no longer feasible. Rather than defining specific physiological or clinical criteria for admission, the criteria should be the failure or probable failure of outpatient management as judged by the physician (11-16).

Hospitalization is recommended for initiation of therapy in asthmatic subjects with serious complicating medical conditions such as cardiac disease or pregnancy (17), depending on the severity of the asthmatic condition or the underlying medical complication. Hospitalization may be indicated for the purpose of removing the patient in exacerbation from an unfavorable environment when there is substantial reason to believe that the environment is contributing to the patient's condition.

2. Treatment of complications of the primary disorder. In addition to treatment of the asthmatic condition itself, a number of complications may arise that require hospitalization. These include serious infectious complications such as acute bronchitis, pneumonia, or sinusitis. Hospitalization is also indicated for treatment of pneumothorax and pneumomediastinum. Hospitalization may be indicated for the treatment of iatrogenic complications of the primary disorder, including medication overdose or severe adverse reaction to medication, and complications of standard treatment, including severe side effects from steroid therapy, both acute, such as hyperglycemia or fluid retention, and chronic, including opportunistic infections, ocular, and skeletal complications. In the case of patients with status asthmaticus requiring treatment with mechanical ventilation, there may be complications of the mechanical ventilation, including disorders of the trachea or persistent bronchopleural fistula, which may require prolonged hospitalization or readmission (18).

3. Treatment of asthma in association with other disorders. Hospitalization may be indicated prior to elective surgery or invasive diagnostic tests for the asthmatic subject who has required chronic treatment (19). The duration of the in-hospital treatment would be proportional to the severity of the patient's disease and the likelihood of the surgery to result in respiratory insult, but usually would be 2 to 5 days. Asthma may be a factor that contributes to prolonged hospitalization in patients with non-pulmonary medical or surgical disease. The combination of asthma with other forms of pulmonary disease may also extend the duration of required hospitalization beyond that of the simple disorder; the duration of additional hospitalization will vary with the severity of the patient's asthmatic condition.

IV. Discharge Criteria

In patients admitted to the hospital for the purpose of establishing a diagnosis of asthma, discharge is indicated when the diagnosis has been established, the diagnostic procedure has been completed, appropriate treatment begun, and the patient's condition is stable. In those cases in which a diagnosis cannot be established but in which asthma has been reasonably excluded as a diagnostic possibility, discharge would be indicated depending upon the patient's overall condition.

In patients admitted for the treatment of asthma as a primary diagnosis, discharge is indicated when a stable treatment regimen using oral or inhaled medications has been both established and demonstrated effective for 24 to 48 h after withdrawal of intravenous medications. Further, these patients should have documented clinical and physiological improvement (comparing the time of their admission to discharge) consistent with them pursuing activities of daily life. Subsequent monitoring of spirometry or peak flows after discharge is important in the patient's routine management. When complications of the disease are the indication for admission, then appropriate diagnosis and treatment of these complications should be instituted prior to discharge. This does not necessarily mean that the treatment must be completed, but rather it must be demonstrated that an outpatient regimen is sufficient for the condition diagnosed. In patients with pneumothorax and pneumomediastinum, the associated radiographic changes must be demonstrated to have improved or stabilized with implementation of a stable medical regimen. In circumstances in which complications arising from medications were the indication for admission, these complications must be shown to be resolving or to have resolved prior to discharge. In circumstances where the patient was admitted for other disorders but in which asthma occurs in association with those disorders, the recovery from the primary disorder, or elective surgery, must have progressed to the point where discharge is usually considered; at that time the asthmatic symptoms must be under control using oral or inhaled medications that can be administered by the patients or their families. In all hospitalized patients with asthma, definite plans for follow-up care are an essential part of the management.

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CHAPTER 3
Pharmacologic Therapy

I. Introduction

The use of pharmacologic agents is an important part of the management of patients with obstructive airways diseases, both during exacerbations and during interim periods of stability. This section will discuss the principal pharmacologic agents and their use in the therapy of asthma and COPD. The information in this chapter is based upon current medical knowledge, and, where controversy exists, upon accepted medical practice. The authors recognize that the following recommendations may need to be revised as new information appears.

II. Therapeutic Agents

1. Beta-adrenergic agonists. The sympathomimetic bronchodilators are the keystone of therapy in patients with obstructive airways disease. Because they can improve mucociliary clearance and serve prophylactically to protect against bronchospasm produced by various stimuli, they may be of value even if they do not result in improvement of spirometric responses on pulmonary function testing. Aerosol formulations provide the optimal therapy for chronic outpatient use (1). In most patients, the metered-dose inhaler (MDI) is preferred, whereas for inpatient therapy powered nebulizers are often used (see RESPIRATORY CARE MODALITIES, Chapter 5).

Oral preparations are falling into disfavor, since they are no more effective than aerosols in most patients and they cause more side effects (2). Appropriate oral dosages are difficult to establish in individual patients, since variables in bowel absorption and first-pass metabolism may markedly reduce the fraction of the drug that enters the circulation in an active form. Aerosol dosages are easier to titrate, result in a more rapid onset of bronchodilation than oral preparations, and achieve a comparable peak response and persistence of effect with a decreased incidence of tremor, nervousness, and palpitations. Furthermore, aerosol bronchodilator delivery is more effective than oral dosing in the prophylaxis of exercise-induced bronchospasm.

Although many patients are unable to use the MDI optimally, repeated instruction results in a satisfactory outcome in the majority of cases (3). There is a small group of patients who fail to learn to use their MDI effectively, and for them the addition of a large volume reservoir or spacer can be advantageous (4).

Optimal use of an MDI results in not more than 10% of the dose being deposited in the lung, while as much as 85% is deposited in the oropharynx. The use of a large volume reservoir may increase the amount deposited in the lungs to 15%, while reducing the oropharyngeal deposition to 5%. In contrast, a typical powered nebulizer unit will result in about 10% of the initial dose being deposited in the lung, whereas only about 10% will be deposited in the mouth and pharynx; about 80% of the dose remains in the apparatus or is lost in the atmosphere (5). The actual amounts deposited will be determined by the apparatus used, the breathing technique, and the length of the treatment session, and therefore considerable variability in the effective dosage can result.

The manufacturers' recommended dosages for use in powered nebulizers are comparable to the oral dosages, and both are many times greater than the dosage delivered by the typical 2 or 3 actuations of an MDI (table 1). Since patients using powered nebulizers or oral tablets usually tolerate these much larger dosages, it is reasonable to evaluate the effect of increasing the number of puffs from an MDI beyond the customary 8 to 12 per day up to 16 to 24, and to add a large volume reservoir, before deciding to add an oral beta-adrenergic drug to the regimen or to change to a powered nebulizer.

The older sympathomimetic agents ephedrine, epinephrine, and isoproterenol have been generally replaced by the newer, longer acting, more beta-2 specific bronchodilators (table 1). Metaproterenol, albuterol, terbutaline, and bitolterol are mainly used as aerosols, but oral preparations of the first 3 are available and terbutaline can be given subcutaneously. In Europe, additional agents that are in use include fenoterol, pirbuterol, clenbuterol, reproterol, and rimiterol. Metaproterenol, albuterol, terbutaline, and bitolerol, when given as aerosols, appear to be comparable enough in potency, length of action and side effects such that when used in recommended doses, they can be used serially or interchangeably. Individual patient preference and cost may be the best determinants for selecting one for chronic use.

Intravenous sympathomimetic bronchodilators have been recommended by physicians in Europe for treating severe bronchospasm, but experience in their use is limited. Only isoproterenol is available for intravenous use in the U.S. and it is not approved by the FDA for treatment of bronchospasm.

2. Theophylline. Theophylline is usually given orally as sustained-release formulations for chronic maintenance therapy. Although the benefits of theophylline are difficult to prove in patients with COPD, its use is favored by most clinicians when appropriately used sympathomimetic agents fail to produce adequate bronchodilation. Twice-a-day administration is generally adequate, although some patients are better controlled if the daily dose is given in 3 equal portions. Recently, some formulations have been demonstrated to provide effective airway dilation when given once a day to patients with less severe asthma. The reliability of longer-acting preparations can be of concern; established products should be favored, and their optimal bioavailability ensured by giving them before meals.

Although some patients respond adequately when their theophylline serum levels are as low as 5 mcg/ml, most require 8 to 20 mcg/ml. A major problem with theophylline is that some patients experience toxic symptoms while blood levels are in the therapeutic range (6). The main side effects are nervousness and tremor resulting from the endogenous release of cathecholamines that theophylline causes, and gastrointestinal symptoms. When a patient begins to use theophylline, a relatively low dosage schedule should be selected; the theophylline serum level can be checked after a few days, and the dosage adjusted appropriately to maintain adequate bronchodilation without associated side effects. Therapeutic serum levels of theophylline on stable doses of a sustained-release oral

TABLE 1

SYMPATHOMIMETIC AGENTS

Drug	Recommended Dosage per Treatment: Subcutaneous (*ml*)	MDI (*mg*)	Nebulizer* (*mg*)	Oral (*mg*)	Duration Of Action (*h*)
Epinephrine (1:1000 solution)	0.1–0.5	0.32–0.9	2.5–22	–	1–2
Isoproterenol	–	0.16–0.39	0.63–3.8	–	1–2
Isoetharine	–	0.68–1.02	1.25–5	–	2–3
Metaproterenol	–	1.3–1.95	10–15	5–20	3–4
Albuterol	–	0.18–0.27	–	1–4	4–6
Terbutaline	0.25–0.5	0.4–0.6	–	1.25–5	4–6
Bitolterol	–	0.37–1.11	–	–	4–6

* Dosages vary widely. These are typical treatment doses, usually given at intervals of 3–6 h.

theophylline preparation occur for about 8 to 12 h in most adults (7).

The dosage of oral theophylline for the average nonsmoking, reasonably healthy adult is 10 to 12 mg/kg/day (e.g., 400 mg twice a day). Smokers may require up to 50% larger dosages, whereas hypoxemic patients or those with liver insufficiency may require a 25 to 50% reduction in dosage. If cimetidine therapy is given, the serum level of theophylline may be rapidly increased by as much as 30 to 50%; the related agent ranitidine has little effect on serum theophylline levels (8). Many drugs (e.g., erythromycin) and other environmental conditions (e.g., diet, hydrocarbon exposure, illness) affect theophylline clearance, and a serum level determination is indicated when a serious environmental or health change alters the control of the bronchospasm.

Aminophylline contains about 80% theophylline solubilized by the addition of ethylenediamine. The latter can rarely cause hypersensitivity reactions in susceptible patients. Aminophylline or theophylline can be given intravenously in critically ill patients, as is discussed later.

3. Anticholinergics. Atropine was used for many years for the management of asthma, but with the availability of potent beta-adrenergic agonists its use declined in the U.S. In recent years there has been an increased interest in inhaled atropine sulfate, especially for patients with chronic bronchitis associated with bronchospasm, although its use as a bronchodilator is not approved by the FDA (9). Atropine is usually given by powered nebulizer, often in combination with a beta-adrenergic agent. Its side effects include tachycardia, dryness of the oral mucosa, blurred vision, urinary obstruction, and constipation. Ipratropium bromide, a quaternary ammonium derivative of atropine, is bronchoselective when delivered by inhalation (10). It is relatively free of systemic side effects because it is minimally absorbed into the systemic circulation and does not cross the blood-brain barrier. It has been shown to be an effective bronchodilator in patients with COPD and in selected patients with asthma both alone and when used concomitantly with beta-2 agonists and theophylline (10). When administered via MDI aerosol, the recommended dose is 2 puffs (40 mcg) 4 times daily.

4. Cromolyn. Cromolyn is neither a bronchodilator nor an antagonist of anaphylactic mediators. However, it has been shown to inhibit histamine release from mast cells (11, 12). Cromolyn is poorly absorbed when given orally and must be given by inhalation, either as a powder or as an aqueous solution available for nebulization and more recently as an MDI. Its advantages are its lack of toxicity and its effectiveness in preventing asthma when used properly, especially in younger patients. A trial period of 4 to 6 wk may be required to determine its usefulness. Cromolyn has been found to be effective in preventing exercise-induced bronchospasm (EIB) and for this condition its preventive effects are immediate (13). Because of its irritating effects on the airways, the powder form of cromolyn should not be used in acute asthma attacks.

5. Corticosteroids. Corticosteroids are useful in the management of acute exacerbations of most cases of asthma and for a minority of cases of chronic airways obstruction (14, 15). They may be given orally or intravenously during acute attacks along with bronchodilator agents. Both oral and inhaled corticosteroids may prove beneficial in preventing acute asthma attacks, and oral therapy can help improve airflow in some patients with COPD. Response should be monitored with objective tests such as FEV_1 or peak flows, and therapy should be continued only if significant improvement occurs (13). Oral corticosteroids are associated with significant toxicity when administered chronically, but alternate-day dosing (in which the entire two-day dose is given once in the morning on alternate days) can be effective in asthma with fewer side effects (16). It is important to use bronchodilators concomitantly in an effort to reduce or discontinue steroid administration.

6. Mucolytics and expectorants. The most troublesome area in pulmonary pharmacology is the treatment of abnormal mucus (17). Sympathomimetic bronchodilators and theophylline offer the advantage of stimulating mucociliary clearance, and these drugs are indicated for any obstructive disease syndrome that is accompanied by impaired mucokinesis. There is also evidence that corticosteroid therapy can improve mucokinesis in bronchitis and asthma. Inhaled atropine does not adversely affect clearance, although systemic anticholinergic and antihistamine therapy can impair mucociliary clearance.

Oral expectorants are popularly used in over-the-counter preparations, e.g., guaifenesin, terpin hydrate, ammonium and other salts, iodide and ipecac. The only topical mucolytic available is n-acetylcysteine, which can be given by aerosol or instillation. The value of inhaled expectorants and mucolytic agents has not been demonstrated in objective studies (18).

7. Antibiotics. Although antibiotics have been used extensively for years to treat acute exacerbations of chronic bronchitis, as well as for prophylaxis in stable chronic bronchitis, their value for either purpose has not yet been established (19–22). Bacterial infection or colonization of the trachea, bronchi, and small airways has been shown not to influence the natural history of COPD. It has been demonstrated repeatedly that the large airways of most of these patients are colonized by the same aerobic bacteria that are found in the oropharynx (23, 24). Two organisms, *Hemophilus influenzae* and *Streptococcus pneumoniae*, have been cultured from sputum and transtracheal aspirates more frequently and in greater numbers from patients with acute exacerbations of chronic bronchitis. The *H. influenzae* strains are almost always nonencapsulated and therefore cannot be typed with specific antiserum, although they can be studied biochemically and placed into "biotypes." Clinicians have prescribed short-term antimicrobial therapy directed specifically against these 2 organisms. The value of such short-term treatment is difficult to assess, although the few carefully controlled and properly designed studies reported thus far have failed to show any clear-cut benefit. A few patients undoubtedly have repeated exacerbations due to bacterial infection and do benefit clearly from antimicrobial therapy; the clinician has little trouble identifying these relatively uncommon individuals. Prophylactic therapy has not been shown to arrest deterioration of pulmonary function over time or to decrease symptoms.

If antimicrobial therapy is to be used in the patient with COPD, the microbial agents of most concern are *H. influenzae* and *S. pneumoniae*. The role of other bacteria that colonize the bronchial tree is unknown, and investigators give them little or no place of importance as this condition is understood at present. Since the value of antimicrobial therapy for most patients is doubtful, any drug chosen for this purpose must be economical and nontoxic. The most suitable agents are ampicillin, amoxicillin, tetracycline, erythromycin, and trimethoprim-sulfamethoxazole. If antimicrobial therapy is to be given, an empirical choice is usually made without knowledge of results from Gram stain of sputum, sputum culture, or studies for antimicrobial resistance.

8. Vaccines. Influenza vaccine has been established to be of great value in reduction of mortality and morbidity during epidemics of influenza. Most deaths from influenza result from bacterial pneumonia which leads to respiratory failure, although influenza viral pneumonia is well documented. The administration of influenza vaccine is associated with a protection rate of 60 to 80%.

Complications from the vaccine directed against Influenza A and B are relatively minor: 2% develop febrile reactions and muscle aching that may last for a few hours, while hypersensitivity reactions can be seen in persons allergic to egg protein. The severe reactions observed following the mass vaccination for swine influenza in 1976 have not been noted with the vaccine containing types A and B.

Since the mortality rate for pneumococcal pneumonia among persons over 60 yr of age has remained unchanged over the past 30 yr, attempts have been made to control this infection with a vaccine containing purified capsular polysaccharide from 23 pneumococcal serotypes. The vaccine is immunogenic in healthy, ambulatory, elderly persons, although its efficacy in preventing pneumococcal pneumonia in debilitated patients with COPD has not been established. The U.S. Public Health Service recommends use of the vaccine in all persons over 50 years of age and in patients with chronic disease including cardiopulmo-

nary disorders. A single dose of the vaccine is judged to be sufficient, but experience is limited and the duration of protection after primary vaccination has not been determined.

9. Amantadine. Amantadine hydrochloride is a tricyclic amine that inhibits an early state of replication of the Influenza A virus. A number of controlled trials have demonstrated the prophylactic effectiveness of amantadine against the development of clinical illness in naturally-occurring and experimentally-induced Influenza A infection. Estimates of efficacy range from 50 to 90%. Side effects include mental changes, ataxia, tremors, and convulsions, especially in the elderly. The recommended adult dose is 100 mg twice daily. In patients over age 65, renal excretion is decreased and daily dosage should be decreased to 100 mg after an initial loading dose of 200 mg.

10. Immunotherapy. Controversy persists concerning the use of immunotherapy in patients with allergic asthma and hay fever (25, 26). The repeated injection of extracts from substances that cause positive immediate skin-test reactions results in the production of "blocking" antibodies, which may decrease the late, but not the early, IgE mediated allergic response (27). Major problems with immunotherapy today include the lack of standardization of allergen extracts and dose, the lack of criteria for selection of those patients who might benefit, and the lack of objective studies to document its possible benefit in asthma (26). Pending the results of these investigations, it seems reasonable to employ immunotherapy for selected patients with episodic wheezing associated with rhinorrhea and conjunctivitis following exposure to known allergens (e.g., animal danders, ragweed pollen), provided contact with these allergens is unavoidable.

III. Drug Therapy of Asthma

1. Therapy of the acute attack. Management of the acute asthma attack is dependent upon the severity of the airway obstruction and the response to initial therapeutic maneuvers. Severity of the attack is determined by objective measurements, such as the FEV_1 or peak flow (28). The initial therapy of the acute attack includes the administration of oxygen and bronchodilator agents (29). Beta-2 specific agents administered via a metered-dose inhaler or powered nebulizer are advised, particularly in older patients and those with cardiovascular problems. Larger doses than those used in stable asthmatics may be required. In young, otherwise healthy asthmatics, subcutaneous epinephrine or terbutaline give results that are comparable to inhaled beta-adrenergic agents.

In patients who have severe obstruction and have not responded to inhaled beta-adrenergic agents, theophylline should be added to the regimen. If intravenous aminophylline or theophylline is selected, an initial loading dose followed by a continuous drip is preferred (table 2). If the patient has been on oral methylxanthines at home, a serum theophylline level should be obtained and the initial loading dose decreased or eliminated. Serum levels should be maintained within the therapeutic range of 8 to 20 mcg/ml and an immediate reduction made if nausea, vomiting, severe nervousness, or cardiac arrhythmia develop. Peak serum theophylline levels should be determined on 2 or 3 occasions when initiating therapy to obtain an estimate of daily requirements. Thereafter, they need not be repeated unless there is a change in the patient's status or new therapeutic agents are added. Severely ill patients may require frequent determinations due to rapid changes in their clinical state.

TABLE 2

MAINTENANCE DOSAGES OF IV AMINOPHYLLINE AND THEOPHYLLINE*

	Aminophyline		Theophylline	
	Calculated (mg/kg/h)	Typical Dose (mg/day)	Calculated (mg/kg/day)	Typical Dose (mg/day)
Nonsmokers	0.5–0.7	900	0.4–0.6	800
Smokers	0.9	1,300	0.75	1,100
Cimetidine use	0.3–0.4	600	0.25–0.3	500
Cor pulmonale	0.25–0.3	500	0.2–0.25	400
Hepatic insufficiency	0.2–0.25	450	0.18–0.2	350

* Recommended loading doses of patients who have not been on maintenance oral therapy are: aminophylline, 5–7 mg/kg, or theophylline, 4–6 mg/kg.

Corticosteroids should be administered promptly if the patient has had frequent recent attacks, if steroids have been required in the recent past, or if the attack is severe and does not respond rapidly (within 30 to 60 min) to sympathomimetic and theophylline therapy. In such cases, corticosteroids are given intravenously along with the bronchodilator agents; their onset of effect is not seen for 3 to 6 h, even with parenteral therapy (30). The dosage of corticosteroids for the acute asthma attack is still controversial; however, a loading dose of intravenous hydrocortisone of 4 mg/kg followed by 0.5 mg/kg/h or an equivalent dose of methylprednisolone (0.8 mg/kg initially followed by 0.1 mg/kg/h) is probably adequate for the initial therapy (30). As soon as flow rates improve, the patient may be switched to oral prednisone or methylprednisolone. The initial maintenance doses should be about 40 mg of prednisone per day or its equivalent; tapering should occur as rapidly as possible while the patient is monitored to avoid relapse. Weaning should be complete within about 2 wk and if this is not possible, a long-term maintenance regimen may be necessary. If attempts at weaning from corticosteroids fail, the lowest effective maintenance dose should be given, with repeated attempts at lowering the dose. Inhaled corticosteroids should be substituted if possible; if not, alternate-day therapy should be tried.

Inhaled atropine, given by powered nebulizer in doses of 0.025 to 0.035 mg/kg, may be given at intervals of 4 to 6 h if the acute attack does not respond to the above measures. The atropine may be combined with a beta-adrenergic agent. Inhaled corticosteroids, cromolyn, and n-acetylcysteine are ineffective and may increase bronchospasm during the acute asthmatic attack. Sedatives are also hazardous and should not be given during an acute attack unless mechanical ventilation is required.

2. Preventive and long-term therapy. Effective management of patients with asthma is designed to prevent acute attacks. In the patient with mild, exercise-induced asthma, most agents have been shown to be effective, including the inhaled beta-adrenergic agonists, oral sustained-release theophylline and cromolyn. The easiest and most convenient way of preventing exercise-induced asthma is by administration of a beta-2 adrenergic agonist from a metered-dose inhaler shortly before engaging in exercise. Inhaled cromolyn is also effective in preventing exercise-induced asthma (18). For more severe asthma (exercise-induced or otherwise troublesome), oral sustained-release theophylline twice daily may be combined with an inhaled beta-adrenergic agent taken at regular intervals of 4 to 6 h.

If long-term corticosteroid therapy is required, the dosage should be tapered to the lowest possible maintenance dose. At this time, patients should be tried on alternate-day therapy, although this may be less effective. Inhaled corticosteroids (beclomethasone, triamcinolone, or flunisolide) may be administered with the goal of eliminating oral steroids. The inhaled agents do not have significant systemic effects, and their major side effects are sore throat and oral candidiasis. This is avoided by using an aerosol spacer and rinsing the mouth and throat with water after each inhalation. When switching from oral to inhaled corticosteroids, it is important to observe the patient for the development of adrenal insufficiency, especially during periods of stress; an overlap period is advised. Non-asthmatic symptoms such as rhinorrhea and arthralgias may appear if they were suppressed by systemic steroid therapy.

In patients with asthma inadequately controlled by other therapy, a trial of inhaled cromolyn is indicated using either a spinhaler, MDI, or powered nebulizer. Cromolyn powder by inhalation often causes bronchospasm

and preceding the cromolyn with a beta-adrenergic agent is generally advisable; when cromolyn is given by MDI, bronchospasm is uncommon, and this is the preferred method of administration.

IV. Drug Therapy of Chronic Obstructive Pulmonary Disease (COPD)

Many patients with COPD have a bronchospastic component, and these patients usually respond to appropriate bronchodilator therapy (14, 31, 32). While inhaled beta-adrenergic agents are often effective, the addition of oral sustained-release theophyllines and/or an inhaled anticholinergic agent may be beneficial in some patients.

For many years, corticosteroids were considered to be contraindicated for patients with COPD; however, investigators have recently shown that there is a subgroup of these patients who may benefit from oral corticosteroid therapy. These are usually patients who respond to inhaled beta-adrenergic agonists, but some patients with less reversible disease occasionally derive significant benefit from corticosteroid therapy (11). A therapeutic trial consists of determining baseline flow rates on optimum bronchodilator therapy, then administering a dose of 32 mg of oral methylprednisolone or its equivalent once daily for 2 to 3 wk. Following this, flow rates are again determined and if no objective benefit is demonstrated by spirometry or peak flow, the corticosteroids should be discontinued. If there is significant improvement, the steroid dose is tapered as rapidly as possible until a maintenance dose is determined. At this time the patient may be changed to alternate-day therapy or an attempt may be made to substitute an inhaled corticosteroid; however, these methods of steroid administration are not effective in all patients with COPD who respond to oral steroids (33). In acute exacerbations of COPD, the addition of intravenous corticosteroids has been shown to be of benefit (34). Benefits of long-term corticosteroids must be weighed against the multiple and varied side effects of these agents.

The prevention of acute attacks of bronchitis includes influenza immunization in the fall of each year utilizing that antigenic combination recommended by the U.S. Public Health Service. This action will reduce death and morbidity from pneumonia and from influenza-induced exacerbations of bronchitis.

Amantadine is recommended for short-term prophylaxis during presumed Influenza A outbreaks for high-risk patients who have not been immunized, and in situations where the vaccine may be ineffective, as in patients who may show a poor antibody response to vaccination. It should be given throughout the epidemic period for patients who cannot be immunized, but should not be a substitute for vaccination for most patients. In an outbreak, nonimmunized patients should be vaccinated and treated with amantadine for 2 wk. The usual dose is 200 mg/day given in 2 divided doses.

For the patient who is hospitalized with an acute exacerbation of bronchitis, antimicrobial therapy is almost always given even though many of these episodes are induced by viral infection. When the patient is to receive intravenous drugs, ampicillin and/or amoxicillin are the drugs of choice. If allergy to penicillin is a concern, alternative agents include erythromycin, cephalosporin, trimethoprim-sulfamethoxazole, chloramphenicol, and tetracycline. The duration of treatment must be individualized since these patients usually show a prolonged recovery period. For the patient with less severe disease who develops a sudden worsening of the bronchitis with increased cough and sputum production with or without fever, leukocytosis, change in sputum volume and sputum purulence, symptomatic care, and rest are indicated. Some physicians treat these patients with oral antibiotics for 1 to 2 wk, but there is little evidence to prove that this approach produces a more favorable outcome than symptomatic care and rest.

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CHAPTER 4
O_2 Therapy

I. Introduction

Supplemental oxygen is one of the most common modalities used in treating patients with obstructive lung disease. The ultimate goal of such therapy is to prevent hypoxic tissue damage. In patients with obstructive lung disease, hypoxic tissue damage results primarily from arterial hypoxemia, which is efficiently treated with O_2 therapy. When other diseases complicate obstructive lung disease, tissue hypoxia may result from other causes of inadequate O_2 delivery to tissues, e.g., reductions in cardiac output or hematocrit. Inadequate O_2 delivery in these situations obviously should be treated by maneuvers aimed at the basic abnormality and is not efficiently treated by O_2 therapy, and will not be considered further in this report. Arterial hypoxemia is one of the most ominous manifestations of obstructive lung disease and is at present the only acceptable indication for O_2 therapy. The immediate goal of O_2 therapy, therefore, must be to increase arterial oxygenation to acceptable levels.

In general, O_2 therapy in obstructive lung disease is used in 2 situations: in acutely ill hospitalized patients, and in chronically ill patients who are not in the hospital.

II. O_2 Therapy for Acutely Ill Patients

A. General Guidelines

As noted above, the indication for O_2 therapy is significant arterial hypoxemia. Arterial oxygenation is usually assessed by measuring the partial pressure of O_2 in arterial blood (Pa_{O_2}), and when $Pa_{O_2} < 60$ Torr, hypoxemia sufficient to treat with oxygen is present. The goal of O_2 therapy should be to increase Pa_{O_2} to at least 60 Torr, equivalent to an arterial O_2 saturation of approximately 90%. On the other hand, increasing Pa_{O_2} beyond 65 Torr is associated with relatively minor further increases in arterial O_2 content (1, 2), therefore, little purpose is usually served by increasing Pa_{O_2} to values greater than 80 Torr, and O_2 doses that do so should generally be avoided. The O_2 dose that will increase Pa_{O_2} to 65 to 80 Torr in a given patient with obstructive lung disease can vary greatly, depending on the severity of the initial hypoxemia and the precise nature of the physiological disturbance. Attaining the correct dose is best done by trial and error, i.e., starting at a given dose (see below), measuring the Pa_{O_2} and adjusting the dose accordingly. It should be noted that in patients with severe COPD, it can take 20 to 30 min for a steady state to be achieved after a change in the inspired gas mixture, so that arterial blood should usually not be sampled at shorter intervals after changes in O_2 dose.

B. O_2 Therapy and CO_2 Retention

In some patients with COPD, O_2 therapy and the associated increase in Pa_{O_2} produce CO_2 retention, or an increase in arterial CO_2 tension (Pa_{CO_2}) (3-5). This is a potentially serious problem in that CO_2 retention may produce coma. There is at present no good way to predict whether or not a given patient will develop a rising Pa_{CO_2} with O_2 therapy, though it generally occurs in very sick patients with Pa_{O_2} of < 40 Torr and with an elevated Pa_{CO_2} while breathing room air, and very rarely occurs in asthmatics (5-7). It follows that the only accurate way to assess the effect of O_2 therapy on Pa_{CO_2}, as well as its effect on Pa_{O_2}, is to measure Pa_{O_2} and Pa_{CO_2} repetitively. In patients who demonstrate increases in Pa_{CO_2} with O_2 therapy, the latter should be used with caution. CO_2 produces central nervous system disturbances by changing brain pH, so that the level of Pa_{CO_2} that produces clinically significant complications depends, in turn, on brain bicarbonate levels. CO_2 narcosis, therefore, cannot be predicted on the basis of the Pa_{CO_2}, since no fixed level of Pa_{CO_2} may be defined as "too high," and CO_2 narcosis can only be diagnosed by careful serial clinical observations. In hypoxemic patients who develop CO_2 retention with O_2 therapy, the physician should attempt to increase the Pa_{O_2} without causing an increase of Pa_{CO_2} sufficient to cause drowsiness or stupor. Often it is possible to produce clinically significant increases in Pa_{O_2} that do not reach the ideal goal of 65 Torr, but are not associated with disturbances of consciousness. It must be recalled that severe hypoxemia causes death, whereas the disturbances associated with severe CO_2 retention are not usually lethal. In severe hypoxemia the first priority must be to increase Pa_{O_2}. If excess O_2 is given, and the patient develops signs and symptoms thought to represent CO_2 narcosis, the inspired O_2 concentration should be reduced, but not to room air since abrupt cessation of all O_2 therapy can produce fatal hypoxemia. If adequate oxygenation cannot be achieved without progressive hypercapnia, mechanical ventilation may be required (3, 5, 6).

C. O_2 Therapy in Emergency Situations

The above discussion has assumed that the diagnosis of obstructive lung disease is clearly established and that measurements of Pa_{O_2} and Pa_{CO_2} are readily available. These assumptions do not always apply; patients are frequently encountered who are in respiratory distress for reasons that are not entirely clear, and, irrespective of diagnosis, O_2 therapy is frequently undertaken without prior knowledge of arterial blood gases. It is reasonable to treat all patients in respiratory distress with O_2 before the results of blood gas analyses are known. If such patients clearly do *not* have COPD, short-term O_2 therapy is essentially without risk, and any O_2 dose – up to 100% – may be safely employed. If, however, COPD is a diagnostic possibility, high-dose O_2 therapy carries the risk of CO_2 narcosis and should be avoided. The lower the O_2 dose, the lower the risk of CO_2 retention. Inspired concentrations of less than 40% O_2 are uncommonly associated with rapidly rising Pa_{CO_2}, and it is rare with inspired concentrations of less than 30%. Thus, when blood gas results are not available, patients suspected of having COPD should be treated with O_2 concentrations of 24 to 40%. In general, it is best to start at the lower end of the dose range, and to increase the dose only when there is clinical or laboratory evidence that this should be done. These inspired oxygen concentrations are usually achieved by nasal flows on the order of 1 to 5 L/min (see below).

Oxygen therapy without arterial blood gas measurement is acceptable only under emergency conditions. Ideally, arterial blood should be sampled in the emergency room as O_2 therapy is started, and the procedure repeated some 20 to 30 min later. In acutely ill patients seen for the first time, prolonged (1 to 2 h) O_2 therapy without measurements of Pa_{O_2} and Pa_{CO_2} are acceptable only under exceptional circumstances. Though noninvasive measurements of Pa_{O_2} and Pa_{CO_2} are available, they should not be relied upon in acute situations, or when rapid changes might be expected.

D. Methods of O_2 Administration

In acute, inpatient situations, O_2 sources are readily available, as are a variety of techniques of transferring O_2 from the source to the patient (8). In general, there are 2 methods of delivering $\leq$ 40% O_2. Nasal "prongs" are popular because they do not interfere with eating and conversation. The O_2 dose can be varied by varying O_2 flow, but the precise inspired concentration achieved depends on the patient's ventilation and breathing pattern, and the dose delivered at a given O_2 flow will show both interindividual and intraindividual variations. The inspired O_2 concentration in % (F_{IO_2}) can theoretically be calculated as: $F_{IO_2} = 20 + 4 \times O_2$ flow (L/min). This is, however, only an approximation. At flows of $\geq$ 4 L/min, the O_2 should be humidified, although at lower flow rates this is not necessary. Alternative methods of administration of $\leq$ 40% O_2 are Venturi masks, which, though they interfere with activities such as conversation and eating, supply fixed, known inspired O_2 concentrations ranging from 24 to 50%.

Oxygen at concentrations exceeding 40% can only be administered by mask. Masks vary in design, but those employing a high flow of O_2 into a reservoir bag are most efficient in that they can deliver inspirates of up to 90% O_2. As noted above, these systems are potentially dangerous in patients with COPD.

III. O_2 Therapy in Chronic Lung Disease

A. O_2 Therapy in Patients with Continuous Hypoxemia

It has been conclusively shown that the survival of patients with hypoxemia COPD is improved by long-term O_2 therapy, and that this benefit is greatest if the treatment is applied at least 18 h/day (9-13). Thus, chronically hypoxemia COPD patients should, in general, be treated in this way. There is no

evidence of benefit for long-term O_2 therapy used less than 12 to 15 h/day.

Benefits from home O_2 therapy have been demonstrated in stable patients with Pa_{O_2} < 55 Torr (arterial O_2 saturation, < 90%), and in patients with Pa_{O_2} 55 to 59 Torr with evidence of polycythemia or right heart failure when stable; it is, therefore, to this group that the treatment should be applied. Stability is best defined in terms of arterial blood gas measurements. Patients are defined as chronically hypoxemic if, when clinically stable, they meet the above criteria during an observation period of 2 wk. Patients with Pa_{O_2} of 45 to 60 Torr can usually undergo the observation period as outpatients, but sicker patients may have to be stabilized in the hospital.

Patients who qualify as outlined above should receive continuous O_2 therapy, i.e., as close to 24 h/day as possible, and the dose should be sufficient to raise resting Pa_{O_2} to 65 to 80 Torr (saturation, 91 to 95%). The dose should be increased by 1 L/min while the patient is sleeping or exercising to eliminate hypoxemic episodes during these activities. With such a regimen, specific studies of oxygenation during sleep and exercise are seldom necessary. The appropriateness of the daytime resting O_2 dose should be assessed periodically.

B. Patients with Intermittent Hypoxemia

Some patients with COPD—not asthma—who have Pa_{O_2} of at least 60 Torr while awake and at rest develop more severe hypoxemia while sleeping or exercising (14). At present there are few data to indicate whether, in such patients, O_2 therapy during sleep or exercise are of benefit, so any standards suggested must be regarded as provisional.

C. Nocturnal Hypoxemia

There appears to be little question that some COPD patients who do not qualify for continuous home O_2 therapy have episodes of severe hypoxemia (arterial saturation, < 85%) while sleeping. Though it is not established that such episodes are harmful, it is probably unwise to assume that they are harmless, and we believe nocturnal O_2 therapy can be justified in such patients. Detection of nocturnal hypoxemic episodes in patients who are not hypoxemic during wakefulness requires study during sleep; the criteria for patient selection for study has not yet been determined (9). COPD patients with $Pa_{O_2} \geq 60$ Torr who are obese, or who have CO_2 retention, polycythemia, or evidence of right heart failure, probably merit sleep studies. The O_2 dose required to eliminate severe nocturnal hypoxemia in these patients can also only be determined with accuracy by sleep study. A minimum nocturnal oxygen saturation of approximately 90% is a reasonable therapeutic goal.

It should be noted that O_2 therapy may not be appropriate for obstructive sleep apnea, which may coexist with COPD. In patients with nocturnal hypoxemia due to obstructive sleep apnea, therapy should be aimed at relieving nocturnal upper airway obstruction.

D. Exercise Hypoxemia

Some COPD patients—usually those with very severe airway obstruction—develop hypoxemia during exercise while maintaining $Pa_{O_2} \geq 60$ Torr at rest. Home oxygen has been prescribed for use during exercise in such patients, though there is no solid evidence for long-term benefit. Since most of these patients spend relatively little time exercising, it is difficult to believe that the hypoxemia of exercise affects survival or function at rest. Thus, the best rationale for supplemental O_2 during exercise is that it will increase exercise tolerance and useful daily activity. However, it is not clear that arterial hypoxemia limits exercise tolerance in all of these patients. Supplemental O_2 during exercise should probably be prescribed only when it has been shown by appropriate testing to increase exercise tolerance significantly. The simplest way to measure the benefits of O_2 during exercise is to conduct exercise tests with the patients breathing both room air and supplemental O_2. These tests are best conducted in such a way that the patient is not aware of whether O_2 or room air is being supplied (15).

E. Methods of O_2 Delivery

The only practical way of delivering home O_2 to patients is via nasal prongs (8). Recently, prongs that supply O_2 only during inspiration have been developed in an attempt to conserve gas; oxygen has also been delivered directly to the sublaryngeal trachea via a chronic transtracheal cannula. These systems have not yet been fully evaluated, and cannot be recommended at present.

Sources of O_2 suitable for use in the home vary, and each has advantages and disadvantages. Liquid O_2 systems were used early in home O_2 therapy, and are the most versatile because of their easy portability: patients can easily carry an O_2 supply for shopping excursions, etc. Liquid systems are also more expensive than any other, and can only be used in urban areas near a source of liquid O_2.

Steel cylinders containing compressed gas can be used as sources for home O_2. These are available in towns large enough to have welding suppliers. They are nearly as expensive as liquid systems, and afford much less portability. Small steel O_2 cylinders are too heavy to carry and must be moved in wheeled carts, which is frequently difficult for sick patients. Recently, aluminum compressed gas cylinders have become available. These are light enough to carry while containing several hours of O_2 supply and are, in theory, an improvement over steel cylinders. However, the most efficient use of aluminum cylinders involves filling them from large steel cylinders in the home, which is regarded as dangerous by many municipal safety authorities.

The cheapest source of home O_2 is the so-called concentrator, which separates atmospheric O_2 from N_2 and supplies the former. Though there is a substantial initial cost for these machines, they require little subsequent service and maintainance. They operate using electrical power and can be used in any home with electricity. Their major drawback is that they are non-portable: If the patient leaves the home, it is either without O_2 or with another portable system. Most programs supply a large steel cylinder of compressed O_2 along with the oxygenator, to provide against electrical failure.

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CHAPTER 5

Respiratory Care Modalities

I. Introduction

Respiratory care modalities are valuable as adjunctive therapy in the care of patients with obstructive airways diseases. Patients, fam-

ily members or other caregivers should be trained in the use of these modalities by qualified practitioners, usually respiratory therapists and respiratory therapy technicians under the aegis of a medical director who has been trained in the care of acute and chronic pulmonary diseases (1, 2). This section will cover the indications and guidelines for the use of incentive spirometry, intermittent positive pressure breathing (IPPB), intermittent continuous positive airway pressure (CPAP), bland aerosol and humidity therapy, medicated aerosol therapy, chest physiotherapy (including postural drainage), chest percussion and vibration, and breathing exercises. Oxygen therapy is discussed in Chapter 4. These modalities may be administered separately or concomitantly.

II. Measures for Lung Expansion

Incentive spirometry is a technique to encourage a patient to take a sustained, deep breath, utilizing a measuring device for direct visual feedback. It is indicated as an aid to facilitate lung expansion in hospitalized patients both to prevent and to treat pulmonary atelectasis. It may be particularly useful before and after major surgical procedures as part of a regimen to help prevent postoperative atelectasis and other respiratory complications (3, 4). The deep breathing maneuvers may also stimulate patients to cough and thereby aid in the removal of abnormal bronchial secretions.

The frequency of use usually depends upon the clinical condition of the patient. In the initial treatment of pulmonary atelectasis, incentive spirometry may be used hourly, at least during the waking hours. With improvement of atelectasis or with prophylactic use of incentive spirometry, the frequency of therapy may be less. It is critical that patients receive instruction in the proper use of incentive spirometry from individuals trained in respiratory care, with the emphasis on sustained maximum inhalation for 5 to 6 s, and 5 to 10 sequential deep breaths (6). Subsequent use of the incentive spirometer may be supervised by nursing personnel, but it is reasonable to provide periodic follow-up assessment by respiratory care personnel to assure that incentive spirometry is being properly administered and utilized.

Although chronic use of lung expansion maneuvers in the home or at other community living sites may be indicated in persons with severe restrictive pulmonary dysfunction, there is no evidence that this form of therapy is useful in patients with obstructive airways diseases. Breathing exercises with a simple inspiratory resistance device or with an incentive spirometer may be of value as a means to increase inspiratory muscle strength and endurance (6), but studies of long-term benefit are still lacking.

The use of the IPPB in patients with COPD is controversial. There is no evidence that it is helpful or desirable for home use (7). Possible selected indications for IPPB in the hospital may include the following (8):

(*1*) The management of atelectasis that has not improved with voluntary deep breathing or incentive spirometry, when it can be documented that the inspiratory capacity is increased by at least 25% using IPPB (9). In these patients, IPPB should be administered by a volume-oriented technique. IPPB cannot be recommended as a routine prophylactic technique to prevent atelectasis.

(*2*) As a measure to provide frequent periodic deep breathing for a patient with acute ventilatory failure in an attempt to avoid intubation or reintubation. In such circumstances, IPPB is a temporizing procedure and not meant to normalize arterial blood gases, allowing the patient time to improve by virtue of correction of reversible factors that have precipitated the acute ventilatory failure.

(*3*) For the delivery of aerosol medications, primarily bronchodilators, in pateints who are unable to breathe slowly and deeply because of acute respiratory distress. It is doubtful, however, that IPPB is more effective in the delivery of nebulized bronchodilators or in causing bronchodilatation than is a nebulizer operated by a constant pressure compressed gas source.

Continuous positive airway pressure (CPAP) by mask has been used in the past for treatment of pulmonary edema. Recently, the use of intermittent CPAP by mask has been investigated in the management of pulmonary atelectasis (10, 11). This modality requires further study in patients with COPD before specific recommendations for its use can be made. In particular, the possible detrimental effects of further hyperinflation on cardiac function and respiratory muscle efficiency must be evaluated. The use of intermittent mask CPAP would appear to have no place in the management of respiratory failure associated with obstructive airways diseases.

III. Bland Aerosols and Humidity Therapy

The use of bland aerosols and humidity therapy in patients with obstructive airways disease centers primarily on humidification of the inspired gas being delivered through an artificial airway (endotracheal tube or tracheostomy). Any patient with obstructive airways diseases and respiratory failure who requires hospitalization and who has an artificial airway, either with or without mechanical ventilation, should be provided with a heated and humidified gas source during the period of tracheal intubation. In patients with chronic obstructive airways diseases who have permanent tracheostomies, the need and method for continued humidification of the inspired gas after discharge must be individually assessed. Further studies are needed to determine if adequate long-term humidification using a moisture-exchange device ("artificial nose") connected directly to the airway opening can be efficacious.

Bland aerosols, utilizing either water or saline, have not been shown to aid in the clearance of abnormal secretions. They neither thin secretions nor enhance bronchial clearing and may precipitate bronchospasm (12, 13). Room humidifiers should be discouraged because of their ineffectiveness in providing increased humidity for the patient and because of their high rate of bacterial and fungal contamination. Hypertonic saline delivered by ultrasonic nebulization may be used for short periods of time to induce sputum for diagnostic studies, although there is no evidence it is superior to coached coughing.

IV. Medicated Aerosol Therapy

It is well established and generally agreed that bronchodilator drugs, administered to patients who demonstrate symptomatic or objectively measured improvement, are useful both in the hospital and in the home. Advantages of aerosol versus oral bronchodilator delivery include: more rapid, predictable onset of therapeutic effect, smaller quantity of active agent required for given degree of objective response, generally greater attainable response at tolerated doses, and fewer systemic side effects.

Bronchodilators may be given as aerosols via a metered-dose inhaler (MDI), with or without a variety of spacer devices (tube, collapsible bag, cone or pear-shaped) introduced to improve the efficiency of delivery of the bronchodilator agents (14, 15) in patients who are unable to use a MDI properly (16, 17). Alternatively, they may be administered with a nebulizer powered by compressed gas or by a small electrical air compressor. Patient instruction in the proper technique of using a nebulizer or an MDI with or without a spacer is essential. This may be done by respiratory care personnel, a specially trained nurse, or by a knowledgeable physician.

In the hospital, aerosolized bronchodilators are usually delivered by nebulizers, although recent studies have demonstrated that, in patients not severely ill, the effects of metaproterenol administered by MDI plus spacer were the same as metaproterenol administered by a nebulizer (18, 19). Administration of the aerosol bronchodilators by either MDI or nebulizer should be performed by respiratory care personnel or other trained hospital personnel for patients who are acutely ill, confused, or feeble. In the stable patient who has demonstrated proficiency in using an MDI, with or without a spacer, little supervision may be required.

The frequency of administration of aerosolized bronchodilators will depend upon the severity of the illness but may be required as often as every hour in those patients with acute severe asthma (20). As the patient improves, the frequency of administration should be dictated by the duration of action of the drug administered (see PHARMACOLOGIC THERAPY, Chapter 3). In the patient with obstructive airways disease who requires surgery, particularly of the thorax or upper abdomen, aerosolized bronchodilator agents should be started preoperatively and continued in the postoperative period, in order to reduce postoperative pulmonary complications (21, 22).

Outside the hospital, aerosolized bron-

chodilators are usually delivered with a metered-dose inhaler with or without a spacer. When inhaled corticosteroids are required, the use of spacer devices with a MDI results in a substantially lower incidence of thrush, and fewer problems with dysphonia, than when the MDI alone is used (14).

Some outpatients, particularly those who are unable to use an MDI, derive benefit from aerosolized bronchodilator agents delivered by a nebulizer. If the nebulizer is used on a daily basis, it may need to be powered by an air compressor since hand-bulb nebulizers may be difficult to coordinate with inhalation. These devices are portable. Patients using a nebulizer in the home should be instructed by trained personnel in the proper use and cleaning of the equipment. Periodic servicing and inspection of home nebulizer equipment may be necessary for some patients.

V. Chest Physical Therapy

Chest physical therapy (or chest physiotherapy) encompasses the use of postural drainage, chest percussion and vibration administered by hand or by mechanical percussion, as well as cough and deep breathing. The rationale for this therapy in the treatment of patients with obstructive airways diseases is the belief that gravity and applied external force to the chest wall will facilitate mobilization and clearance of secretions from the airways, leading to an improvement in pulmonary function. In order for chest physiotherapy to be effective in the home or hospital, the patients must have excessive secretions (30 cc/day or greater) that are difficult to expectorate (23–27).

In the hospital setting, chest physiotherapy is indicated in those patients who have great difficulty raising secretions and in those patients who develop atelectasis either postoperatively (28) or under other circumstances (29, 30). Fiberoptic bronchoscopy can be effective in acute lobar atelectasis, but is no more effective than vigorous chest physiotherapy given by experienced personnel (27). Chest physiotherapy may be effective in acutely ill patients with obstructive lung diseases who expectorate large sputum volumes (30, 31) even if they require mechanical ventilation (30, 32). Use of chest physiotherapy has not been shown to be effective in acute exacerbations of chronic bronchitis (33–35), in patients with scant secretions receiving mechanical ventilation (31), in patients with status asthmaticus (26), or in patients who have pneumonia (36).

Chest physiotherapy is indicated either in the hospital or at home in stable patients with bronchiectasis, cystic fibrosis, and chronic bronchitis who chronically produce large sputum volumes (37–41). It is not effective in patients with COPD who produce less than 30 cc/day (42). To facilitate bronchodilation and mucociliary clearance, chest physiotherapy should be delivered after the administration of an aerosolized bronchodilator (35).

The frequency of administration of chest physiotherapy in patients who might benefit from it has not been established. In patients with atelectasis and in others who have difficulty expectorating sputum, probably no more than 4 treatments per day are practical or tolerable. Patients with more stable conditions, especially in the home setting, usually will require fewer treatments.

In those patients who will benefit from the continuation of chest physiotherapy outside the hospital, the patient and family members should undergo a complete educational program on the technique and goals of chest physiotherapy in the home prior to discharge. Trained respiratory care personnel, nurses, or physical therapists usually provide this instruction. Family members can be taught to administer percussion and vibration. The number of teaching sessions required before the patient and family members are competent will depend upon their ability to grasp the concepts and apply them during therapy. Usually several sessions will be required.

VI. Breathing Exercises

Breathing exercises encourage patients to inspire slowly and to expire through pursed lips (25, 26). Simultaneous relaxation of the neck and upper thoracic musculature should be encouraged (26). Breathing exercises may be effective in increasing the patient's tidal volume, decreasing the respiratory rate, and lowering the FRC (43, 44), thereby improving the efficiency of gas exchange and reducing the work of breathing.

In the acutely ill patient, the use of breathing exercises may be helpful in aborting hyperventilation episodes precipitated by panic or anxiety, provided the patient is familiar with the technique. Even if the patient is untrained, coaching by respiratory care personnel or other trained professionals to inhale slowly and exhale through pursed lips is helpful.

Patients with stable obstructive airways diseases may benefit from breathing exercises, both physiologically and symptomatically (43–46). They are most effective over short-term use, since long-term studies show no improvement in pulmonary flows (45, 47). Breathing exercises are effective in helping the patients overcome attacks of hyperventilation precipitated by fear and anxiety and may be useful to combat the urge to hyperventilate after mild exercise.

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CHAPTER 6

Physical Rehabilitation and Home Care

I. Introduction

The most common and distressing symptom in patients with COPD is dyspnea resulting in limitation of activity. The objectives of pulmonary rehabilitation are to control and alleviate symptoms and pathophysiologic complications and to achieve optimal ability to carry out activities of daily living. The broad concepts and elements of pulmonary rehabilitation have been reviewed in a previous ATS statement (1). Most pulmonary rehabilitation programs include either encouragement for patient activity or a regular exercise component. Although there is general agreement that patients benefit from pulmonary rehabilitation programs, the specific contribution of exercise to the improvement is not well defined.

Limitation of exercise in patients with COPD is related to multiple factors including: abnormal pulmonary mechanics, impairment in pulmonary gas exchange, an abnormal perception of breathlessness and ventilatory control, the presence of impaired cardiac performance due to cor pulmonale, poor nutritional status, and the development of respiratory muscle fatigue (2).

Determination of the exact number of factors involved and their relative importance in an individual patient with COPD is difficult and often impossible. The inability to accurately characterize the exercise-limiting factors in individual patients has led to confusion and controversy in assessing the effectiveness of various forms of therapy designed to improve exercise performance.

Several physical rehabilitation techniques are utilized to increase the dyspnea-limited level of activity or to decrease the degree of dyspnea associated with the same level of activity. The methods utilized include exercise reconditioning, inspiratory muscle training, breathing retraining, and energy conservation techniques.

II. Patient Selection

Physical rehabilitation should not be considered in the COPD patient until optimal medical control of the disease has been achieved. Motivation is the most important factor in the selection of patients for physical rehabilitation. In younger patients with less than advanced disease, preservation of body weight and muscle mass and minimal disease in other organ systems are more likely to persist with and benefit from physical rehabilitation. It is important that patients be realistically apprised of the effort and time involved and the limited benefits to be expected from physical rehabilitation before embarking on an extensive program.

Screening should include spirometry, arterial blood gases, and exercise evaluation. A standard 6- or 12-min walk, cycle or treadmill testing can be used; the ventilatory limit to exercise can be estimated from the FEV_1. The presence of exercise arterial desaturation should be evaluated by exercise oximetry or exercise blood gases since patients with exercise desaturation should receive oxygen during exercise if it increases exercise capacity (see O_2 therapy). On the basis of these data, a prescription for the tolerable level of exercise can be formulated. The type(s) of physical rehabilitation program and adjunctive therapy must be individualized to each patient. For some patients, there may be some psychosocial and motivational advantages to group programs. The program should be initiated under medical supervision. Periodic assessment of the benefits or side effects of the program is required. Improved general fitness frequently results in improved exercise tolerance. In general, physical activity should be encouraged for patients with COPD. Whether additional benefit is derived from formal exercise programs is uncertain and unproved.

III. Comments of Rehabilitation

A. Exercise Reconditioning

Exercise training to improve performance in COPD patients, using methods similar to those used to improve athletic performance, was suggested in 1951 (3) and demonstrated to be effective in 1964 (4). A recent review of the numerous studies of exercise reconditioning in COPD summarizes the potential benefits (table 1) (5).

The type of exercise (stair climbing, walking, treadmill or bicycle ergometer) appears to be unimportant and is best determined by patient preference and cost. Although the intensity of exercise is usually determined by patient tolerance, the minimal duration and frequency required to improve performance appears to be 20 to 30 min, 3 to 5 times per wk. Leg exercise is usually better tolerated than arm exercise. Due to ventilatory limitation, the level of exercise tolerated by most patients will not increase cardiovascular fitness. Many patients are not suitable candidates for exercise training because of far-advanced lung disease, advanced age, lack of motivation, and associated diseases. The effects of exercise conditioning disappear rapidly with cessation of exercise.

B. Inspiratory Muscle Training

Measures to specifically increase the strength and endurance of respiratory muscles have received recent attention as a potential part of a physical rehabilitation program for patients with COPD (6-9). The efficacy of this therapy is unproved and its role is unknown at present.

The physiologic basis for the improved performance associated with either exercise reconditioning or inspiratory muscle training is not fully understood. Major factors involved appear to be improved aerobic capacity, increased motivation, desensitization to the sensation of dyspnea, improved muscle

TABLE 1

BENEFITS OF EXERCISE RECONDITIONING

ACCEPTED BENEFITS
Increased endurance and exercise tolerance.
Increased maximal oxygen consumption (generally small).
Increased skill in performance of a task with decreased ventilation oxygen consumption, heart rate.
UNLIKELY OR UNKNOWN BENEFITS
Improved survival.
Improved pulmonary function tests.
Lowered pulmonary artery pressure.
Improved arterial blood gases.
Improved blood lipids.
Change in muscle O_2 extraction.
Change in sleep desaturation or apnea.

function, and improved technique of performance (6, 8, 9, 12).

In addition to physical conditioning, several forms of adjunctive therapy may improve exercise performance in some patients. Those generally accepted to be beneficial include oxygen, beta-2 bronchodilators, and theophylline. As yet unproved modalities include pulmonary vasodilators, nutritional manipulation, and improved psychosocial health.

C. Breathing Retraining

Breathing retraining consists of teaching patients to utilize pursed-lip breathing, expiratory abdominal augmentation, synchronization of movement of abdomen and thorax, and relaxation techniques for the accessory respiratory muscles, as well as psychological assurance and education about COPD. Breathing retraining appears to allow patients to recover more rapidly from dyspnea induced by exercise. Such training is a useful component of a comprehensive care program for patients with COPD.

D. Energy Conservation

A standard aspect of physical medicine and rehabilitation is instruction in work simplification and the use of energy conservation devices to allow disabled patients more independence and greater participation in activities of daily living (13). Such instruction, particularly in patients with advanced COPD, may result in similar benefits (14).

E. Nutrition

Advanced COPD is frequently associated with loss of weight and muscle mass. This may produce respiratory muscle weakness and further limit ventilatory capacity. Patients need to maintain sufficient protein/calorie intake to prevent malnutrition. Frequent small feedings or use of liquid formula diets may help. Enteral or parenteral hyperalimentation has been attempted to restore muscle mass in malnourished COPD patients but the efficacy of such therapy is unknown at present. There is a recognized hazard in administering high carbohydrate loads, which may result in increased CO_2 production, requiring increased ventilation (15), although the clinical importance of this is uncertain.

F. Smoking Cessation

Cigarette smoking affects lung structure and function in the following ways: increased mucous secretion due to mucous gland hypertrophy and hyperplasia leads to increased cough and sputum production; small airways demonstrate a spectrum of abnormalities from mild inflammation to airway closure; lung parenchymal changes vary from a simple increase in inflammatory cells to destruction of alveolar walls which results in centrilobular emphysema (16–18).

A significant functional impairment is identified in 10 to 15% of smokers, and is best identified by changes in FEV_1. In nonsmoking adults, FEV_1 declines at the rate of approximately 20 to 30 ml per year. In those smokers who develop significant impairment, the rate of decline is 50 to 100 ml per year (19–21).

The profoundly adverse pulmonary effects of cigarette smoking demand that smoking cessation efforts be implemented by all who care for patients with chronic obstructive pulmonary disease. Physicians should assume major responsibility in this regard.

Motivational factors governing smoking behavior vary. There is evidence that strongly suggests that smoking is an addictive behavior due, in large part, to nicotine.

Successful smoking cessation programs are those that address the biologic, behavioral, and psychological forces responsible for smoking as specifically as possible for each individual patient. Cessation programs include education and counseling by the physician or other trained personnel, and a variety of other techniques, including provision of self-help materials, group or individual behavior modification programs, pharmacologic management using nicotine polacrilex to minimize nicotine withdrawal, adversive conditioning using rapid cigarette smoking, hypnosis, and acupuncture.

Current data indicate that 20 to 30% of those enrolling in a "successful" smoking cessation program will not have resumed smoking at the end of 1 yr. Using this criterion, the efficacy of counseling and nicotine polacrilex, and of adversive conditioning, have been proved. Self-help programs result in somewhat lower quit rates, but can be provided at lower cost to greater numbers of smokers (22–24). The efficacy of hypnosis has neither been proved nor disproved. Controlled clinical trials have not demonstrated acupuncture to be an effective strategy (25).

The prompt benefits of smoking cessation for patients with chronic obstructive pulmonary disease are reduction of cough and sputum production. Ultimately, a decrease in the rate of decline of the FEV_1 may be seen (21). Additional major clinical benefits include a reduction in the risk of cardiovascular mortality and in the risk of cancers of the lung, larynx, mouth, esophagus, and bladder (26).

Smoking cessation should be of highest priority in the comprehensive care of patients with COPD.

G. Psychosocial Management

Patients with chronic obstructive pulmonary disease frequently suffer from anxiety, depression, and problems related to cognitive, perceptual, and motor activity. Limitations of financial and social resources are commonplace. Comprehensive care requires that attention be given to the psychosocial as well as the physiologic problems of patients with COPD. The medical history, specific psychosocial interviews, questionnaires, and formal testing may be used to develop a thorough psychosocial assessment. Problems should be identified by evaluation of the patient's social, cultural, ethnic, and educational background. Employment history, current financial resources, and family and community support should be identified. In addition, the patient's personality, psychosexual concerns, significant life events, previous and current lifestyle, and level of disability should be assessed (27, 28).

Individuals responsible for the psychosocial evaluation may include the primary physician, pulmonary consultant, nurses, therapists, and chaplains. Formal rehabilitation programs frequently use psychologists, social workers, and psychiatrists in performing the psychosocial evaluation.

Effective psychosocial interventions include education, counseling, and supervised exercise and supportive therapy provided by the medical staff, family, and groups of similarly afflicted patients. Motivation and the development of realistic goals are emphasized. Other aspects of a psychosocial, intervention program include community referral, vocational counseling, psychiatric consultation, and the use of anti-anxiety and/or antidepressive medications (29, 30).

Psychosocial interventions provide the patient with improved understanding of the physiologic factors responsible for symptoms, and aid in coping with dyspnea, stress, anxiety, and depression. The patient and family are helped in identifying and using available community, social, financial, and health care resources. Successful psychosocial management helps the patient accept physiologic limitations, optimize strengths, and clarify reasonable goals and priorities. Such intervention allows the patient to participate more

actively and effectively in a therapeutic program. When psychosocial approaches are used effectively, the patient is provided with a sense of control and mastery of his disease, and his quality of life is enhanced.

To summarize, physical rehabilitation is an important component of a comprehensive care program for patients with COPD. In many patients, activity level can be improved or maintained, resulting in both physical and psychologic benefits. Programs must be individualized to the needs and capabilities of each patient. Investigation is needed in identifying and assessing specific exercise-limiting factors in patients with COPD and determining the optimal therapy for each factor. This would allow more appropriate prescription of physical rehabilitation techniques and other adjunctive therapy for the individual patient.

IV. Home Care

Home care refers to health services that are provided to individuals and families in their place of residence for the purpose of promoting, maintaining, or restoring health, or minimizing the effects of illness and disability. Services appropriate to the needs of the individual patient and family are planned, coordinated, and made available by an agency or institution, and organized for the delivery of health care through the use of employed staff, contractual arrangements, or a combination of administrative patterns. Home care encompasses components including but not limited to, medical care, dental care, nursing, respiratory care, physical therapy, speech therapy, occupational therapy, social work, nutrition, homemaker, home health aide, transportation, laboratory services, medical equipment and supplies (31).

The goals of home care are to: (*1*) improve the quality of life by allowing those patients with advanced disease to remain in their own environment and be with family and friends; (*2*) minimize or prevent complications that would require hospitalization; (*3*) detect changes in physical and psychosocial status that indicate the need for changes in management; (*4*) provide treatment for the patient's primary diagnosis and foster adherence to the therapeutic program; and (*5*) foster a positive and independent attitude.

A small number of studies document the benefits of home health care (32–34), but more work needs to be done to confirm that continuing care at home will, in fact, decrease hospital admissions and length of hospital stay, and reduce overall cost of care.

A. Qualifications of the Home Care Provider

The skills required of the home health care team are outlined in a position paper by the ATS (35). In addition, if durable medical equipment is required, the vendor should provide 24-h coverage by a respiratory care practitioner, rapid response to correct problems of equipment malfunction, and adequate back-up equipment.

The home care programs may be: (*1*) hospital-based, or (*2*) community-based, under the direction of: (a) visiting nurse association, community nursing or public health agency, (b) proprietary nursing agency, or (c) durable medical equipment companies.

B. Patient Selection

Selection of patients and authorization for home health care is the responsibility of the treating physician based on medical evaluation and information obtained by nurses, social workers, and other members of the health care team. Referral to an organized home health program is necessary for patients when there is doubt that the medical care program can be carried out in the home because of lack of knowledge, motivation, or adequate family caregivers, or because of the severity of illness. Any patient requiring additional teaching or support should be considered as a candidate for home care. Examples of the spectrum of home health needs include: (*1*) patients who require only periodic outpatient medical supervision; (*2*) patients who require the assistance of home health aides and/or homemakers and infrequent or no professional visits; (*3*) patients newly diagnosed or newly educated in a comprehensive care program who require visits for 2 to 4 wk to reinforce details and to help with adapting family caregivers and the home environment to the patient's needs. Generally, 1 or 2 visits per wk are sufficient; (*4*) patients with repeated hospitalizations who need regular supervision for an indefinite period to prevent clinical deterioration and repeated hospitalizations. The number of required visits depends on the complexity of the treatment program – from 1 to 2 times per month to several times per wk; and (*5*) patients with complex treatment programs, such as home ventilator care. The number of visits required depends on the skill of the patient and family caregivers. The need may last for the rest of the patient's life.

C. Types of Services Provided in the Home

A position paper developed by the American Lung Association describes the essential components of a home care program (36). These include evaluation, education, observation, sexual counseling, consultation, psychosocial support, monitoring of respiratory equipment, direct patient care, and household help. Not all services are required by all patients and the types of services required by patients with COPD must be individualized. Duplication of services is both unnecessary and expensive. Although home care is most commonly instituted upon hospital discharge, such care may be initiated in the absence of a hospital admission. In hospitalized patients, the plan of care should be determined and communicated to the home care team prior to discharge to assure a smooth transition from hospital to home. The physician, nursing staff, and other allied health professionals need to work collaboratively to develop a feasible plan of care. The discharge planner, generally a nurse or social worker, needs to be aware of the patient early in the hospital course. Equipment requirements need to be assessed before discharge and, preferably, the hospitalized patient should be able to see and use the equipment that will be available in the home. The process of discharge planning should begin at the time of admission (37).

A care plan needs to be developed for each patient. The standards for nursing care outlined by the ATS Section on Nursing (38) and guidelines developed by the California Thoracic Society Nursing Section (39) may be used along with standards of home care outlined by the American Association for Respiratory Care (40) as a basis for the development of this plan.

V. Home Mechanical Ventilation

Because the care of ventilator-assisted patients in the hospital setting is extremely expensive, there is great interest in identifying those ventilator-assisted patients who can be managed safely in the home. At present, the principal role for mechanical ventilation in the home is in the management of patients with ventilatory failure due to neuromuscular disease. Patients with severe chronic obstructive pulmonary disease are rarely suitable candidates for mechanical ventilation at home because of complicating and frequently unstable medical problems that create such stringent demands upon caregivers' therapy, thus making management in the home unsafe and impractical. However, a select and undoubtedly very small group of patients with severe, *stable* chronic obstructive pulmonary disease who are unable to maintain adequate pulmonary gas exchange on their own may be candidates for mechanical ventilation at home. They consist of an unknown number of patients with severe chronic obstructive pulmonary disease who are hospitalized for acute respiratory failure, placed on mechanical ventilation, and, who despite stabilization of their condition, cannot be weaned from total ventilator support. It has been noted that intangible factors such as familiar surroundings and the attention of friends and loved ones may, when coupled with respiratory muscle rest, result in increasing independence from the ventilator in some patients who could not be weaned in the hospital setting despite exhaustive attempts by skilled physicians, nurses, and respiratory therapists. The potential for ventilator dependency should be recognized and, when appropriate, patients with severe obstructive lung disease should be informed of the possibility of permanent ventilator dependency prior to initiating mechanical ventilation in the hospital setting.

In addition, an as yet poorly defined subset of these ventilator-assisted patients with chronic obstructive pulmonary disease may suffer from respiratory muscle fatigue due to abnormal resistive loads, muscle weakness and, in part, to mechanical disadvantage and shortening of the diaphragm due to pulmonary overinflation (41). When total intermittent ventilator support is discontinued, these patients may experience increasing dyspnea, hypercapnia, and hypoxemia. The quality of life and physical well-being of such patients

may be improved with intermittent mechanical ventilatory support (42, 43). In this group, ventilation is usually provided at night with a negative pressure ventilator. Insufficient data exist with regard to the benefit of this form of partial ventilator support, and, at present, it should be considered investigational.

Several reports have formulated criteria for patient selection, established guidelines for home ventilator management, and demonstrated that ventilator-assisted patients can be managed safely in the home (44–51); however, there have been no large-scale, controlled clinical trials to evaluate the health and/or economic benefits and risks of such management.

A. Patient Selection

Consideration of mechanical ventilation in the home setting is indicated when a competent respiratory care team is unable to wean a patient with COPD from total ventilator support after several attempts over a period of weeks. The patient's health status must be stable such that no major therapeutic or diagnostic interventions are contemplated within a 30-day period of discharge from the hospital. The patient should maintain an arterial oxygen tension of greater than 60 mm Hg with an inspired oxygen concentration of less than 40%. The patient should not demonstrate wide fluctuations in arterial oxygen or carbon dioxide tensions. The patient should have an active cough and gag reflex, and should not require frequent endotracheal suctioning. A secure tracheostomy tube should be present, except in those patients managed with a negative pressure ventilator. The patient should be free of active infection, and should not be subject to frequent or recurrent infections. Any comorbid medical conditions should be stable, and should not require frequent therapeutic interventions. The candidate for mechanical ventilation in the home should express a desire to be discharged to home on a ventilator, and a willingness to cooperate with the respiratory care team in order to acquire the information and skills required of a ventilator-dependent patient at home. Care of a ventilator-assisted patient in the home often creates significant physical and emotional stress for the patient, family, and caregivers. Patients and their caregivers must be fully aware of this potential prior to deciding to participate in a home ventilator program. A psychosocial evaluation should confirm that the patient and caregivers are aware of and understand the demands and stresses associated with maintaining a ventilator-dependent patient in the home, and that coping resources are adequate to meet these demands (52).

B. Caregivers

Within the circle of family and friends, caregivers must be identified who are available and express the willingness and physical, emotional, and cognitive ability to provide care. A support network of family, friends, and neighbors should be available to provide additional assistance when needed. Individuals identified as caregivers must demonstrate a commitment to participate in an educational program, and devote sufficient time and energy to develop, utilize, and demonstrate the skills necessary to care for a ventilator-assisted patient at home.

Caregivers must learn, master, and *demonstrate* those skills that will enable them to provide total patient care. The skills required include the ability to: (*1*) administer medications in a correct and timely manner and familiarity with the actions of and side effects of such medications; (*2*) assemble and disassemble ventilator circuits; (*3*) adjust ventilator settings and alarms; (*4*) clean, maintain, and troubleshoot equipment; (*5*) use a hand resuscitator; (*6*) administer breathing treatments; (*7*) continue weaning efforts; (*8*) administer supplemental oxygen when necessary; (*9*) set up, use, and clean suction apparatus; (*10*) perform tracheostomy care and suctioning; (*11*) clean, change, and plug tracheostomy tubes when indicated; (*12*) maintain proper tracheostomy cuff inflation when applicable; (*13*) provide clapping, vibration, and postural drainage as well as cough assistance; (*14*) position patients correctly and assist with transfers, strengthening and range of motion exercises; (*15*) understand and instruct in energy conservation techniques; (*16*) measure vital signs and recognize changes in vital signs and other signs and symptoms of respiratory distress; (*17*) recognize signs and symptoms of respiratory infection; (*18*) perform proper skin care; (*19*) feed the patient or administer enteral feedings if necessary; (*20*) perform required bladder and bowel care; (*21*) communicate effectively with the patient; (*22*) contact the local emergency systems; and (*23*) perform cardiopulmonary resuscitation.

C. Resources

A suitable and safe home environment with sufficient space and appropriate hygienic and electrical requirements is necessary. Doorways and halls must permit access and mobility of the patient and equipment.

Required resources include a primary physician, medical and pharmaceutical suppliers, home health agencies, an emergency transport system, and a reasonably proximate hospital and emergency room. Sufficient financial resources to cover the total costs of environmental modification, equipment, supplies, and paid caregivers must be available.

Prior to beginning the process of patient and caregiver education, administrative approval for home management of a ventilator-assisted patient must be obtained from third-party providers. The physical home environment and availability of required resources must be evaluated by members of the respiratory care team, and plans for modifications in doorways, electrical outlets, hygiene facilities, and access must be made as necessary.

D. Planning for Discharge

The process of transferring a ventilator-assisted patient with chronic obstructive pulmonary disease from the hospital to home requires the involvement and cooperation of an experienced respiratory care team, working with community health agencies, durable medical equipment suppliers, state and local agencies including the phone company, utility companies, fire department, and emergency medical services. Although the program for discharging a ventilator-assisted patient to home should be structured and the team experienced, the specific educational routine and plans for each patient must be individualized and tailored to the patient's unique needs.

During the process of preparing the patient for discharge, the full resources of the respiratory care team must be devoted to an ongoing process of rehabilitation in which the patient's potential for independence in activities of daily living are maximized. Ideally, patients will be able to breathe independently for periods throughout the day. In practice, the degree of ventilator dependence ranges from ventilatory support 24 h a day to a requirement for nighttime ventilation only.

The respiratory care team leader may be a pulmonary nurse specialist, discharge coordinator, or qualified respiratory therapist. This individual should coordinate the activities of the team and meet with the patient, members of the team, and home caregivers to establish specific goals, develop training schedules, and insure that supply and equipment needs are met. Team conferences to evaluate and accept potential candidates, to plan the treatment program, and to measure progress are required. Documentation of training activities and patient progress is essential. A physician must be willing to accept responsibility for overall supervision and periodic follow-up care of the patient, and should approve all plans for medications, ventilator care, and nursing care. The physician should periodically assess the need for continued ventilator assistance. The physician should be willing to make home visits if necessary. If office visits occur, appropriate transportation must be prearranged.

The members of the respiratory care team possess a variety of skills. Where resources vary, there may be overlapping functions of health care personnel. The skills of the following personnel may be required depending on individual patient needs: (*1*) primary physician, (*2*) nurse, (*3*) respiratory therapist, (*4*) physical therapist, (*5*) occupational therapist, (*6*) social service worker, (*7*) psychologist/psychiatrist, (*8*) durable medical equipment vendor, and (*9*) home health agency.

Prior to discharge, the patient and caregivers may make several brief excursions outside the hospital, perhaps spending some time at home. At the completion of a training program, the caregivers must have demonstrated competencies in all required care of the patient to the satisfaction of the respiratory care team. A checklist should document the demonstration of required abilities and the availability of necessary equipment and resources. Prior to discharge of the patient, required

equipment and supplies must be delivered to the home.

Respiratory care and nursing personnel will accompany the patient home, and make frequent home visits during the first 1 to 2 wk following discharge. Thereafter, the attending physician and respiratory care team will periodically evaluate the patient to determine if modifications of the medical, nursing, or ventilator program are necessary.

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This statement was developed by a Task Group appointed by the ATS Scientific Assembly on Clinical Problems. Members of the Task Group were:

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NOTICES

ADVANCES IN SECTIONAL IMAGING

The Department of Radiology, University of California, San Diego School of Medicine is presenting the following postgraduate course: "Advances in Sectional Imaging," September 10–12, 1987, at the U.S. Grant Hotel, San Diego, California. The Program Director is Robert F. Mattrey, M.D. The guest faculty includes Matthew D. Rifkin, M.D.; Thomas Jefferson University Hospital, Philadelphia, Pennsylvania; and Elias A. Zerhouni, M.D.; Johns Hopkins, Baltimore, Maryland. The faculty from the University of California, San Diego School of Medicine is Giovanna Casola, M.D.; Robert Edelman, M.D.; John Forsythe, R.D.M.S.; Paul J. Friedman, M.D.; Barbara B. Gosink, M.D.; John R. Hesselink, M.D.; George R. Leopold, M.D.; Robert F. Mattrey, M.D.; Thomas R. Nelson, Ph.D.; Dolores H. Pretorius, M.D.; David J. Sartoris, M.D.; and Eric van Sonnenberg, M.D. The registration fee for the course is $375.00 for physicians and $275.00 for residents, fellows, or technologists. The course is accredited for 14 hours in Category I. To receive more information, please contact: Dawne Ryals, Ryals & Associates, P.O. Box 920113, Norcross, GA 30092-0113; (404) 641-9773.

BOARD REVIEW IN CRITICAL CARE MEDICINE

Board Review in Critical Care Medicine (ACLS Option). October 7–11, 1987 – Portland, Oregon. School of Medicine, Oregon Health Sciences University, CME-GH., OHSU, Portland, OR 97201; (503) 225-8700.

CARDIOPULMONARY UPDATE '87

The Heart & Lung Institute at St. Vincent's Medical Center in Jacksonville, Florida will hold a seminar at the Marriott at Sawgrass, Ponte Vedra Beach, Florida on current topics of interest to the pulmonologist, cardiologist, oncologist, and internal medical physician. The registration fee is $225.00, payable by Sept. 10, 1987. For further information, please contact: Alberta Hipps, Adm. Director, Heart & Lung Institute, P.O. Box 2982, Jacksonville, FL 32203; phone: (904) 387-7563.

12th INTERNATIONAL CONFERENCE ON LUNG SOUNDS

The 12th International Conference on Lung Sounds will be held in Paris, France at the Institut D'Electronique Fondamentale, Wednesday through Friday, September 16–18, 1987.

Call for abstracts: Papers for presentation during the Conference will be selected by the Program Committee. Abstracts should not exceed 200 words in length and should be submitted by July 1, 1987. Notifications of acceptance will be mailed out by July 15, 1987. Abstracts may relate to any aspect of lung sounds; examples are studies of mechanisms of production, clinical implications, physiological correlations, methods for recording, analysis or representation.

Registration: Registration fee is $100 per person. Checks should be made payable to: International Lung Sounds Association.

Correspondence: All abstracts and questions regarding arrangements should be addressed to:

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PRACTICAL SPIROMETRY COURSE

The course "Practical Spirometry" will be held August 19–20, 1987, in Atlanta, Georgia and October 8–9, 1987 in Chicago, Illinois. Sponsored by: Mayo Pulmonary Services. For further information, please contact: Ginnie Allie, Mayo Pulmonary Services, 432 Plummer, Mayo Clinic, Rochester, MN 55905; or call toll free 1-800-533-1653 (Minnesota residents, 1-800-562-1767).