Problems in Family Practice

Chronic Airway Obstruction

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The initial diagnosis and evaluation of patients with chronic airway obstruction should include an assessment of the degree of pathophysiologic abnormality by means of pulmonary function tests and arterial blood gas analysis; a chest roentgenogram and an electrocardiogram provide information on the extent of parenchymal disease and its cardiac effects. The management of these patients should focus on measures to reduce airway irritation, prevent and treat pulmonary infections, and decrease the functional effects of the airway obstruction. Abstinence from cigarettes and the avoidance of air pollution such as working in dusty atmospheres would reduce continued airway irritation. Exacerbations due to pulmonary infections, most commonly due to Hemophilus influenzae and Streptococcus pneumoniae, require treatment with an appropriate antibiotic: ampicillin, amoxicillin, or trimethoprim sulfamethoxazole. Bronchodilator drug therapy should be used in patients who demonstrate some reversibility of airway obstruction; useful bronchodilator drugs include theophylline, metaproterenol, isoetharine, and terbutaline. A trial of steroid therapy may be indicated in some patients. The treatment of cor pulmonale requires adequate oxygenation and diuretic therapy; digoxin is not indicated, except for the treatment of arrhythmias.

Chronic airway obstruction is a term given to a heterogeneous group of diseases[1] in which there is an increased resistance to pulmonary airflow. These diseases include emphysema, chronic bronchitis, and asthma. It is to be noted that whereas the diagnosis of emphysema (defined as a condition in which there is dilation and destruction of the distal units of the lung) is definitively made on histopathologic examination, the diagnosis of chronic bronchitis (defined as a productive cough during at least three months of the year in two consecutive years) is made from the clinical history, and the diagnosis of asthma (hyperirritability of the airways) is based on both the clinical history and examination. In practice, however, most patients will have a combination of chronic bron-

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Table 1. Clinical Features of Chronic Airway Obstruction

	Predominant Emphysema (Type A: pink puffer)	Predominant Chronic Bronchitis (Type B: blue bloater)
Dyspnea	+++	+
Sputum production	+	+++
Arterial PCO_2	Low (<40 mmHg)	High (>46 mmHg)
Cor pulmonale	Occurs late	Early onset
Chest roentgenogram	Hyperlucent areas Attenuation of vascular shadows Low, flat diaphragms	May be normal Evidence of right ventricular enlargement and pulmonary hypertension

Table 2. Initial Evaluation of Chronic Airway Obstruction

1. Peripheral blood count
2. Chest roentgenogram
3. Electrocardiogram
4. Spirometry
5. Arterial blood gases

chitis and emphysema, since in most instances the underlying cause—cigarette smoking—is the same. It is estimated that about 10 to 20 percent of the adult population of the United States suffers from chronic bronchitis and/or airway obstruction.

Diagnosis

The clinical presentation of these patients can be broadly divided into two types (Table 1), although many patients show features of both.

The family physician, when first presented with a patient with airway obstruction, needs to assess the degree of pulmonary pathophysiologic abnormality, and the presence or absence of chest infection and cor pulmonale. Thus, initial investigation (Table 2) would include a peripheral blood count, chest roentgenogram, pulmonary function tests, and an electrocardiogram.

Examination of the peripheral blood may reveal the presence of secondary polycythemia, suggesting chronic hypoxemia. A polymorphonuclear leukocytosis would suggest the presence of infection.

Chest roentgenography, posteroanterior and lateral views, provides valuable information. Hyperlucency and the degree of attenuation of the vascular shadows give some indication of the degree of emphysema. In addition, a recent study has shown that when the level of the right diaphragmatic dome is at or below the level of the anterior end of the seventh rib, airway obstruction is likely.[2] Evidence of cor pulmonale may be obtained from the roentgenographic heart size. In addition, the presence of infection may be indicated by parenchymal opacities in the chest roentgenogram.

Cor pulmonale (defined as right ventricular

Table 3. Management of Chronic Airway Obstruction

1. Reduce exposure to smoke and air pollutants
2. Vaccination
3. Antibiotics
4. a) Bronchodilator therapy
   b) Steroids
5. Treatment of cor pulmonale
6. Oxygen therapy
7. Long-term rehabilitation

hypertrophy due to pulmonary hypertension secondary to lung disease) may be present in the absence of florid clinical signs of right heart failure. Therefore, an electrocardiogram is very helpful: right axis deviation and tall (>2.5 mV) P waves in standard Lead II are indicative of cor pulmonale.

Pulmonary function tests consist as a minimum, of spirometry before and after bronchodilator administration, to assess the degree of airway obstruction and whether it is partly reversible. In addition, arterial blood gas analysis gives an excellent assessment of ventilatory function and pulmonary gas exchange.

In patients presenting at an earlier age (eg, 30 to 40 years) with advanced emphysema, measurement of the serum alpha-1 antitrypsin level is indicated.[3] If the level is very low (<60 mg/100 ml), suggestive of homozygous deficiency, siblings should be screened for a similar defect with a view to prevention.

Management

The management (Table 3) of chronic airway obstruction involves measures to reduce airway irritation, prevent and treat pulmonary infections, and treat the existing disease.[1] The patient must be advised to reduce exposure to airway irritants. Cigarette smoke is the most common irritant and the patient should be advised at each office visit to stop or reduce the number of cigarettes smoked. Although, this may not result in complete abstinence from cigarettes, many patients will reduce the number smoked. In addition, the patient should be made aware of the harmful effects on lung function of living or working in areas of heavy air pollution (eg, coal dust and rock quarry dust).

Frequent exacerbations of airway obstruction occur in these patients, and in about 40 to 50 percent of these episodes, a chest infection can be documented to be present. These infections give rise to a significant degree of morbidity even though there appear to be no long-term effects of these infections on the course of the disease. Vaccines are available for the prevention of influenza and pneumococcal pneumonia, and patients should be offered these vaccines—influenza vaccine every year in the fall, and pneumococcal vaccine at not less than three-year intervals, although experience with the latter may alter current practice.

The most common infecting organisms in these patients are Hemophilus influenzae and Streptococcus pneumoniae.[4] A chest infection may be presumed to have occurred when any two of the following are present: increased cough and sputum production, fever, polymorphonuclear leukocytosis, and parenchymal opacities on the chest roentgenogram. The drugs of choice are ampicillin, amoxicillin, or trimethoprim sul-

Table 4. Use of Bronchodilator Drugs

	Advantages	Disadvantages	Availability
Theophyllines	Potent bronchodilator Orally effective	Non selective beta adrenergic agonist No inhaled form available	Oral and parenteral forms
Isoproterenol	Potent bronchodilator Effective orally and by inhalation	Non selective beta adrenergic agonist Short duration of action	Oral and inhaler
Metaproterenol	Potent bronchodilator Effective orally and by inhalation Selective beta-2 adrenergic agonist	Not available in parenteral form	Oral and inhaler
Isoetharine	Potent bronchodilator Selective beta-2 adrenergic agonist	Only available in inhaled form	Inhaler
Terbutaline	Potent bronchodilator Selective beta-2 adrenergic agonist	Not available in inhaled form	Oral and parenteral forms

famethoxazole. Patients who cannot take these antibiotics for any reason can be treated with tetracyclines or erythromycin. Treatment in appropriate dosage should be continued for at least one week. An alternative technique is to give daily antibiotics throughout the winter months.

Bronchodilator therapy should be given to patients in whom a significant decrease in airway obstruction following inhaled or oral bronchodilator drugs can be demonstrated on spirometry. There is probably no role for bronchodilator drugs where no significant bronchodilator response can be demonstrated.

Aminophylline is probably the bronchodilator drug of choice in terms of cost and effectiveness. The usual adult oral dose is 200 mg four times daily, and the dose should be adjusted to achieve maximum effect while avoiding the toxic side effects of nausea, vomiting, and nervousness. It is rarely necessary to measure serum theophylline levels; however, when high doses (eg, 400 mg three times a day) are being given or when the patient develops symptoms of toxicity at relatively low doses, it does become necessary to measure serum theophylline levels. Serum theophylline levels of 10 to 20 mg/100 ml give maximum bronchodilation with minimum side effects.[5] Other bronchodilator drugs may be equally useful (Table 4). In particular, inhaled bronchodilator drugs, such as isoproterenol or metaproterenol, have the advantage of rapid action and can be used to supplement oral aminophylline therapy. In patients with heart disease, particularly patients with arrhythmias, the use of the more selective, beta-2 adrenergic agonist drugs such as metaproterenol, isoetharine, or terbutaline should be considered.

In patients who are clearly responsive to bronchodilator drugs and in whom symptoms of airway obstruction persist in spite of maximal bronchodilator therapy, a trial of oral steroids is warranted. In such cases, the administration of oral prednisone, 30 mg twice a day for one week, should be tried. If, at the end of this period, no symptomatic or objective spirometric improvement has occurred, the steroids can be rapidly tapered and stopped. If there is spirometric and

symptomatic improvement, the steroid dose can be slowly reduced to the point where symptoms again worsen or spirometric deterioration occurs. At this point, either the prednisone dose can be continued at this level or inhaled steroids (eg, beclomethasone dipropionate) should be added and a further attempt made to stop or reduce the oral steroid dosage.

Physical therapy in the form of percussion and postural drainage, breathing exercises, or IPPB (intermittent positive pressure breathing) therapy in the patient with chronic airway obstruction has not been shown to have any beneficial long-term value.[6] Indeed, the use of IPPB therapy increases the risk of developing a pneumothorax.

Cor pulmonale occurs in these patients as a result of pulmonary hypertension secondary to pulmonary vasoconstriction due to hypoxia and hypercapnia. Therefore, measures to prevent or improve hypoxia and hypercapnia will also reduce the degree of cor pulmonale. The prevention and treatment of infection and bronchodilator therapy are very helpful in this regard. In addition, where salt and water retention resulting in peripheral edema exists, diuretic therapy is indicated. On the other hand, there is little evidence of a beneficial effect of digoxin on cardiac function in cor pulmonale; conversely, digoxin may cause dangerous arrhythmias in the presence of hypoxemia and acidosis. However, digoxin administration may be necessary if atrial arrhythmias exist. The relief of hypoxia with oxygen therapy is one of the most effective treatments for cor pulmonale. Oxygen at low flow rates (1-2 liter/min), given for at least 12 hours every night, has been shown to result in both symptomatic and objective hemodynamic improvement.[7] These changes become apparent after three to four weeks of oxygen therapy. Patients selected for oxygen therapy should obviously be those in whom significant hypoxemia exists (eg, arterial PO_2 less than 55 mmHg) and in whom oxygen therapy at flow rates of 1-2 liter/min increases PaO_2 without causing an unacceptable rise in arterial PCO_2 (eg, to $PaCO_2$ levels greater than 60 mmHg). Recent studies have suggested that some patients with airway obstruction may have adequate PaO_2 levels during waking hours, but develop severe hypoxemia during sleep and these patients will need to be identified for oxygen therapy. Recently oxgen concentrating machines have become commercially available; these relieve the patient of the burden of replacing oxygen gas cylinders at regular intervals.

Currently, several studies are examining various modalities for the long-term rehabilitation[8] of patients with chronic airway obstruction. The following modalities are being examined either singly or in programs combining these modalities: exercise conditioning by means of graded exercise to attempt to improve effort tolerance, breathing training, coughing and postural drainage, and long-term home oxygen therapy combined with psychological support and indoctrination of the patient and his/her family. It is to be hoped that these studies will soon bear fruit.

Although previous data indicated that the long-term progress of chronic airway obstruction is little affected by the various therapeutic maneuvers discussed above, recent studies are more hopeful that prognosis in these patients may indeed improve.[9] In any case, there can be little doubt that these modalities of treatment improve the quality of life for the patient.

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