CHAPTER 2

CLINICAL MANIFESTATIONS AND DIAGNOSIS OF OBSTRUCTIVE AIRWAY DISEASE

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The term "obstructive airway disease" includes several different disease entities of which the most important are asthma, chronic bronchitis, and emphysema. Often included in the group is bronchiectasis. Although obstruction of the airways occurs in this disease, obstruction is certainly not the preeminent feature; bronchiectasis has therefore been omitted from this section. Physiologically these diseases fall into two groups: those with reversible obstruction, viz, asthma, and those with irreversible obstruction, viz, chronic bronchitis and emphysema. Reversible obstruction implies a 20 percent or more improvement in one or more of the several tests used to measure ventilatory capacity, eg, the forced expiratory volume in one second (FEV_1) or the maximal voluntary ventilation (MVV). Despite the relative precision which the physiologist brings to the study of respiratory impairment, the differentiation of the various types of obstructive airway disease still depends mainly on the history and, to a somewhat lesser extent, the physical examination (Table 1).

Although relatively pure forms of chronic bronchitis and emphysema exist, more often there are features of both diseases in the same person. While the ensuing description deals with each disease in its pure form, only occasionally does one find one disease without some of the features of the other. Despite this admixture, there is a tendency for the clinical features of either emphysema or bronchitis to predominate. Dornhorst has coined the term "blue bloater" for the person whose symptoms and signs are predominantly those of chronic bronchitis and "pink puffer" for the person whose clinical features are mainly those of emphysema[2] (Table 2).

Until recently the major impediment to an understanding of obstructive lung disease lay in the fact that not only did different countries use different terms for what was often the same disease, but also a language barrier existed between different disciplines in the same country. The term "emphysema," for example, had, and unfortunately sometimes still has, a different connotation in the minds of the radiologist, pathologist, internist, and pulmonary physiologist. Thus, in the past the physiologist maintained that any condition associated with a significant increase in the residual volume denoted emphysema. The internist, on the other hand, felt that the physiologist's definition was too far removed from clinical medicine, since it ignored the patient's airway obstruction and dyspnea. The radiologist diagnosed (and unfortunately many still diagnose) emphysema on the basis of hypertranslucency of the lung fields. None of these restricted viewpoints is satisfactory. Because of the obvious limitations, various organizations, including the American Thoracic Society and World Health Organization, have seen fit to take upon themselves the task of defining the various types of obstructive airway disease. Fortunately, and perhaps surprisingly, the definitions proposed by the various organizations are essentially similar. For the sake of convenience, the writer employs those adopted by the American Thoracic Society.[1]

TABLE 1

DIFFERENCES BETWEEN ASTHMA, BRONCHITIS, AND EMPHYSEMA

Points for Comparison	Asthma	Bronchitis	Emphysema
Nature of bronchospasm	Reversible	May occasionally be slightly reversible	Nonreversible
Sex distribution	Slightly commoner in females (3:2)	Much commoner in males (10:1)	Much commoner in males (10:1)
Usual age of onset	5 to 30 years	30 and upward	Over 40
Development of symptoms	Abrupt	Insidious	Insidious
Loss of alveolar-capillary surface	No	No	Invariable
Presence of right ventricular hypertrophy	Very rare	Frequent	Occasional
Increased residual volume	Present during exacerbations	Usually present but need not be marked	Always present
Radiologic findings	Overdistention	Either none or increased bronchovascular markings	Overdistention
Increased translucency	Frequent	Absent	Frequent
Attenuation of peripheral pulmonary vasculature	Absent	Absent	Frequent
Effect of steroids	Marked benefit	Either none or very minimal effects	None

ASTHMA

Asthma is a disease characterized by an increased responsiveness of the trachea and bronchi to various stimuli, and made manifest by difficulty in breathing due to generalized narrowing of the airways. This narrowing is dynamic and changes in degree either spontaneously or in response to therapy.

It is customary to subdivide asthma into the extrinsic or allergic and the intrinsic or infective varieties. This classification is useful in the management of the disease; however, it is probably an oversimplification as far as the intrinsic type is concerned, since many factors besides infection play a role in its etiology.

Extrinsic Asthma. This disease usually starts in childhood or early adult life. There is frequently a family history of allergy, migraine, or urticaria. The person who is prone to develop this condition often has other manifestations of allergy; he may have suffered from eczema as an infant, he may be sensitive to certain foods, or he may be afflicted with hay fever. His attacks are often precipitated by exposure to certain substances or pollens. In some subjects the attacks occur only in the ragweed season, while in others they occur earlier when grass pollens are in the air. Dogs, cats, house dust, lobster, and a multitude of other substances may bring on an attack.

TABLE 2

CHARACTERISTICS OF THE TWO TYPES OF IRREVERSIBLE OBSTRUCTIVE AIRWAY DISEASE

	"Pink Puffer"	"Blue Bloater"
Sputum	Small volume and mucoid	Copious and mucopurulent or purulent
Shortness of breath	Severe or extreme	Moderate
P_{CO_2}	Normal	Elevated
P_{O_2}	Normal or slightly down	Desaturated
X-ray	Large proximal pulmonary vessels and attenuated peripheral vessels	Normal or increased bronchovascular markings
Heart	Seldom gets heart failure	Repeated bouts of cor pulmonale
Polycythemia	Hardly ever	Frequent

The above facts go a long way to explain the popularity of hyposensitization in asthma. Nonetheless, it must be emphasized that objective studies to show that desensitization is beneficial are few, and it is only in persons with definite pollen allergy that this mode of therapy has anything to offer. Recent advances in immunochemistry have provided a better understanding of the allergic mechanisms taking place in extrinsic asthma. It has been shown that there are two types of allergic reaction in extrinsic asthma[3]: type I allergy is reagin mediated and immediate; type III is delayed in onset and is related to the Arthus phenomenon. In type I allergy the antigen reacts with reaginic antibody and causes the mast and other cells to liberate histamine and slow-reacting substance (SRS-A). The reaginic antibody has been identified as IgE, and methods now exist for its quantitative measurement. IgE is present in the body in minute amounts as compared to IgG and IgA; nonetheless, it has been shown that in 65 percent of subjects with extrinsic asthma, IgE is elevated, while in the intrinsic variety, only 5 percent of the patients have raised levels. Many subjects with atopic eczema also have elevated IgE levels. A new drug, disodium cromoglycate, inhibits the relase of histimine and SRS-A from the mast cells by blocking the attachment of allergic specific reagin to the cells.

The delayed allergic reaction is related to a precipitating antibody. Patients with allergic bronchopulmonary aspergillosis often present with episodic asthma. Aside from the immediate urticarial type reaction when skin tested, these patients develop an extensive edematous type III response several hours later. This may manifest itself in the lungs as recurrent pulmonary infiltrates. Type III reactions respond to steroids, while type I reactions are uninfluenced by them. Lysosomes are liberated in type III reactions and probably are responsible for the fever and other systemic reactions seen in "delayed" allergy.

Intrinsic Asthma. Intrinsic asthma tends to start later in life than does the allergic type, developing in the early adult period or between 40 and 50 years of age. There is no seasonal occurrence; moreover, intrinsic asthma is less episodic than the extrinsic type and is far more intractable. Although definite exacerbations occur, it seldom clears up completely and the patient remains wheezy and short of breath most of the time. When the patient develops an upper respiratory tract infection, his condition deteriorates rapidly; hence the use of the term "infective asthma." This condition is not associated with an increase in the level of IgE, and hyposensitization is completely ineffective.

There is little doubt that in some instances infection aggravates the disease. During acute exacerbations the patient often produces yellow sputum; this finding is considered by many to indicate the presence of infection. On the other hand, if the sputum is smeared and stained with Wright's stain for eosinophils, in many instances it becomes apparent that the "purulence" is a consequence of the presence of numerous eosinophils in the sputum rather than of true pus. When a patient is seen who is virtually always wheezy and short of breath and who has a fair amount of sputum but responds fairly well to bronchodilators, he is sometimes labeled as having "asthmatic bronchitis." In reality, most of these patients have infective asthma.

Apart from infection, various other factors play a part in intrinsic asthma. In certain subjects, psychologic factors are of great importance. Exacerbations may be precipitated by stress, usually of the domestic variety. The prospect of being greeted after a hard day's work by a disagreeable wife is sufficient to make most husbands wheeze. Fre-

quently the type of asthmatic attack that is precipitated by stress occurs in psychoneurotic individuals and often responds to hypnosis. In a few persons the development of the asthmatic attack is related to endocrine factors, eg, menstruation. The disease also tends to be more common in persons in the higher socioeconomic level; it is uncommon in primitive peoples.

Symptoms and Signs of Asthma. As already indicated, the disease is best diagnosed from the patient's history. A series of attacks of episodic wheezing lasting from 30 minutes to several days and associated with shortness of breath provides the clue. The paroxyms are often nocturnal; in this respect they resemble a common manifestation of left ventricular failure, viz, paroxysmal nocturnal dyspnea. The differentiation between bronchial and cardiac asthma is of paramount importance; while morphine is standard therapy for the cardiac variety, it may be fatal in bronchial asthma. The medical history and the absence of physical findings to indicate cardiac disease or hypertension nearly always serve to distinguish the two conditions.

During the acute attack the patient is usually sitting upright in bed. His wheeze may be clearly audible; he hangs his head forward with his mouth open and his alae nasi flaring. The accessory muscles of respiration are in use and he may be cyanosed. He converses in grunts and is barely able to drink or eat. If there have been repeated attacks, his chest is overdistended and barrel-shaped. Cardiac and hepatic dullness are reduced, and scattered rhonchi are present. The breath sounds are clearly audible, expiration is prolonged, and there are wheezes over the entire chest. Occasional scattered sonorous rhonchi are heard over the larger airways. If during an acute attack the breath sounds become distant, the pulse rate goes up, and the patient appears quieter and more difficult to arouse, this usually means the patient is exhausted and is no longer making much of an effort to maintain his blood gases at a normal level. This "quiet state" is commonly brought on by overenthusiastic sedation and often portends a fatal outcome. During an acute attack of asthma, most subjects are mildly desaturated, with a low or normal arterial carbon dioxide pressure (P_{CO_2}) of 32 to 38 mm. When the obstruction becomes very severe, viz, the FEV_1 falls below 20 percent, carbon dioxide retention occurs and profound hypoxia is a frequent result.

Episodic bronchospasm may be the first symptom of pulmonary aspergillosis, periarteritis nodosa, and sundry other conditions. Most of these are accompanied by fleeting pulmonary infiltrates and eosinophilia of sputum and blood.

Radiologic Features of Asthma. During the acute attack the chest film may be entirely normal, but if there have been repeated attacks, marked overdistention is common. The diaphragms are depressed and in the lateral film the anteroposterior (AP) diameter is markedly increased. In contrast to the situation in emphysema, the pulmonary vasculature is normal.

Right ventricular hypertrophy and cardiomegaly are absent. Occasionally small perihilar linear infiltrates are seen and, rarely, a conspicuous area of atelectasis involving a segment or subsegment may be apparent. Formerly these opacities were attributed to pneumonia, but in reality they are commonly areas of collapsed lung secondary to mucoid impaction. They may persist for two to three months before the patient manages to cough up the offending bronchial plug (Curschmann's spiral). However, pneumonia can occur. Fleeting pulmonary infiltrates also are seen in certain asthmatic subjects and usually indicate a parasitic or collagen vascular disease.

Chronic Bronchitis

Chronic bronchitis is best defined as a disorder characterized by excess mucous secretion by the bronchi. It manifests itself clinically as a chronic productive cough that occurs in the absence of localized destructive disease such as tuberculosis, bronchiectasis, or neoplasm. The British have recognized this entity for many years, but outside of Great Britain there has been an inexplicable reluctance to accept its existence. Although the entity is often referred to as "the English disease," it is also commonly found elsewhere in Europe and in urban North America. The normal person secretes 100 ml of mucus a day; it is continuously swept up the bronchial tree by ciliary action and is then subconsciously swallowed. In the patient with chronic bronchitis the volume of mucus produced greatly exceeds 100 ml/day and to get rid of the excess the patient develops a productive cough. This is often referred to as a "smoker's cough," an appellation which accurately reflects its origin but tends to underestimate its gravity.

Mucus is secreted by the bronchial mucous glands and also by the goblet cells found in the bronchial wall. The latter are innervated by the vagus; however, the glands respond only to direct stimuli, eg, irritant gases such as cigarette smoke, sulfur dioxide, or mechanical stimulation with a bronchoscope or tracheostomy tube.

Reid has shown that in chronic bronchitis there is hypertrophy of the mucous gland layer of the bronchial wall.[4] Normally the mean depth of the mucous gland layer is less than 25 percent of the distance from the epithelial surface of the bronchial mucosa to the cartilage. In persons with chronic bronchitis this depth ratio may be 50 percent or more (Fig 1). With the cessation of smoking, the mucous gland tends to revert toward normal.

Symptoms and Signs. The disease characteristically affects male cigarette smokers living in urban communities. As indicated above, in the early stages it is usually described as smoker's cough and the subject looks on the symptoms as if they were a normal part of living. After 10 to 15 years the patient with chronic bronchitis begins to notice that every time he has a cold it goes to his chest and his sputum changes from mucoid to purulent. If appropriate sputum cultures are done, it will be found that *Diplococcus pneumoniae* and *Haemophilus influenzae* can be isolated with increased frequency. The subject notices that during acute exacerbations he becomes short of breath, and even when his sputum has reverted to its original mucoid character he finds himself more short of breath than other men of his own age. Spirometric studies show irreversible obstructive airway disease, usually indistinguishable from emphysema. In a small minority of persons with chronic bronchitis there seems to be a partly reversible element to their bronchial obstruction; nonetheless, this is never as clear-cut as it is in asthma. Over the ensuing 5 to 10 years, the chronic bronchitic starts to become obviously cyanotic. He eventually develops cor pulmonale; this is invariably associated with an acute infection and usually occurs during the winter months. The

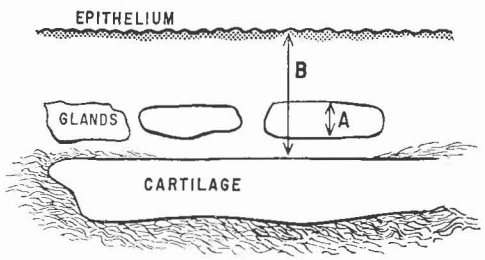

Fig 1. The mucous gland layer (A) is normally 25 percent of the distance from the epithelial surface of the bronchial mucosa to the cartilage (B). In chronic bronchitis there is hypertrophy of the mucous gland layer. (From Morgan, W. K. C.: *Postgrad Med* 41:191, 1967.)

type of cor pulmonale which commonly supervenes in longstanding chronic bronchitis is the high output type. The subject shows central cyanosis but his hands are warm and his pulse is full. Atrial fibrillation and other arrhythmias are uncommon. There may be mental confusion; in the past this has been attributed to carbon dioxide retention, but in the untreated patient it is almost always an hypoxic effect. After oxygen administration, hypercarbia and confusion are common. Edema of the extremities is likewise frequent and papilledema may be present. The latter is a consequence of raised cerebrospinal fluid pressure secondary to increased cerebral flow and carbon dioxide retention. There is often a right-sided summation gallop in the fourth interspace to the left of the sternum. The chest may be overdistended; however, in pure chronic bronchitis, signs of emphysema may be absent or minimal. Scattered rhonchi may be heard all over the chest. The breath sounds are often reduced, but when the subject pants they become more audible. This is a useful physical sign in differentiating chronic bronchitis from emphysema.

After two or three bouts of heart failure, there is a tendency for the pulmonary vascular resistance to climb; when this occurs, the pulse becomes small and thready, the hands cold, and the veins of the arm and face constricted. A giant "a" wave may be seen in the jugular pulse and a presystolic right ventricular gallop develops. The electrocardiogram is not very helpful but often shows a P pulmonale with right axis deviation and, less commonly, clear-cut right ventricular hypertrophy.

Radiologic Features. The plain chest plate of a patient with chronic bronchitis is often within normal limits; rarely is the AP diameter increased. More commonly the film resembles that of any obese and somewhat plethoric middle-aged man. In a few instances there is some peribronchial linear fibrosis, especially in the lower zones. When heart failure has occurred, there may be cardiac enlargement and evidence of right ventricular hypertrophy on the lateral and oblique films. The pulmonary vessels are normal or sometimes prominent, unlike those seen in emphysema.

There are several characteristic findings on bronchography. (1) Enlarged mucous glands arising from the undersurface of the larger bronchi may be demonstrable. They appear as dye-filled pits along the major and segmental bronchi and are often best seen in the left main bronchus (Fig 2). (2) The bronchial tree appears beaded and dilated, an appearance suggestive of mild cylindric bronchiectasis. This appearance entirely disappears if the films are taken in expiration. In addition, numerous "beads" of mucus appear in the bronchi, often abruptly occluding smaller bronchi. (3) If centrilobular emphysema is present, a mimosa or flower pattern may be seen peripherally. These appearances are produced in the centrilobular form of emphysema by the dye entering the sac.

Emphysema

Emphysema is best defined as an absolute increase in the amount of air distal to the terminal bronchioles associated with disruption of the alveolar-capillary surface. It is the latter part of the definition that is of paramount importance, for an asthmatic subject with an acute exacerbation will have an increased residual volume owing to dilatation of the alveoli and respiratory bronchioles. On the other hand, his alveolar-capillary surface will be intact. Thus, because emphysema is best defined in pathologic terms, the diagnosis is often difficult and must depend largely upon circumstantial evidence.

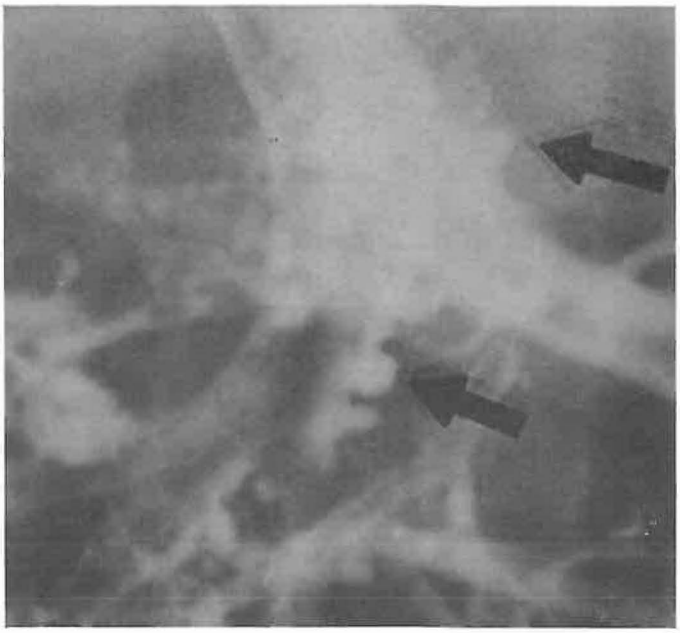

FIG 2. Bronchogram of subject with chronic bronchitis showing major bronchi with dilated mucous gland ducts filled with dye. These are most commonly seen on the undersurface of the main and segmental bronchi.

There are two common forms of emphysema, the centrilobular and the panlobular varieties. The former tends to be associated with chronic bronchitis, while the latter usually presents clinically as so-called dry emphysema. Nonetheless, combinations of the two occur. Approximately 0.5 to 2 percent of patients with irreversible obstructive airway disease have a deficiency of α_1 antitrypsin. This is an hereditary defect associated with a distinctive type of emphysema which often presents in early adult life. The lung bases are predominantly affected by the destructive changes; women seem to be affected as commonly as men. Homozygous and heterozygous forms of enzymatic defect occur but it appears that it is only the former that precipitates the full-fledged syndrome.

Most men and quite a few women who die after the age of 60 can be shown to have some emphysema at postmortem. Most of these may have had no appreciable respiratory symptoms during life. However, the fact that the disease may be asymptomatic in some instances does not imply that this is always true; there is little doubt that emphysema can and does produce shortness of breath in the absence of chronic bronchitis.

Symptoms and Signs. The person with relatively pure emphysema, ie, the pink puffer, is virtually always a male cigarette smoker around 45 to 50 years old when first seen. Unlike the chronic bronchitic, he usually produces either no mucoid sputum or only a small amount first thing in the morning. His main symptom is shortness of breath, especially on exertion, and he may be severely disabled by it. Unlike the dyspnea found in asthma, it is not episodic, although there are some occasions when it may be more troublesome than usual. A cough is seldom prominent until the patient develops a viral infection of the respiratory tract, at which time he may start to produce purulent sputum and become even more short of breath.

Physical examination nearly always reveals a thin and somewhat emaciated person who often is short of breath at rest. Slight exertion markedly exaggerates his dyspnea. He is seldom obviously cyanotic and clubbing is rare. His chest may show an increased AP diameter; however, this finding is no more common in emphysematous patients than it is in normal elderly persons. In fact, it is one of the least reliable signs of the condition. Hepatic and cardiac dullness may be diminished, but this again is a common finding in elderly subjects whose functional residual capacity has increased because of loss of lung elasticity. The patient's use of the accessory muscles of respiration is a more helpful sign, and undue prominence of sternomastoids may be present. The jugular venous pressure may rise markedly in expiration. The length of the trachea palpable above the suprasternal notch is reduced; this is best expressed in fingerbreadths and is measured from the suprasternal notch to the lower margin of the cricoid cartilage. This is also a common finding in asthma. Prominent epigastric pulsation is often seen, since the heart tends to be elongated. The abdominal muscles are rigid because they are being used in an attempt to squeeze out air during expiration. The liver is usually palpable 2 to 3 fingerbreadths below the costal margin. This finding is not the result of hepatomegaly, but is caused by lowering and flattening of the diaphragm, displacing the liver downward. The percussion note tends to be tympanitic, and diaphragmatic excursion is often reduced. The breath sounds are usually faint and expiration is prolonged, especially when the patient is panting. A few sibilant rhonchi may be heard; the sonorous variety usually indicates concomitant chronic bronchitis. In contrast to the findings in chronic bronchitis, hyperventilation does not render the breath sounds more audible. With the possible exception of this sign, all the above signs are nonspecific and may be found in the other types of obstructive airway disease. It is the patient's history which is of the most help in making the diagnosis.

The patient with emphysema notices that his shortness of breath slowly increases over the years. As in chronic bronchitis, upper respiratory tract infections aggravate the condition but seldom cause cor pulmonale until the patient is terminal. Death is often precipitated by a viral infection which leads to pneumonia. Under normal circumstances the patient's blood gases are relatively normal; while there may be slight hypoxia, carbon dioxide retention is rare. The relative normality of the blood gases is achieved at the expense of tremendous respiratory effort and gross dyspnea.

Radiologic Features. The radiologic interpretation of emphysema is notoriously inaccurate. The usual report which reads "there is some emphysema at the bases," is inaccurate and meaningless. As often as not the dark apearance at the bases and elsewhere in the lungs is a consequence of an overpenetrative exposure. The AP diameter of elderly persons tends to be increased as a result of normal senile kyphosis. When the radiology technician measures the AP diameter he notes the increase and adjusts the penetration accordingly. He fails to make allowance for the fact that in general the soft tissues and muscles of older persons have partially atrophied.

Even when the film shows low flattened diaphragms, an increase in the retrosternal space, and an attenuated heart and generalized hypertranslucency, emphysema is still not a justifiable diagnosis. The report should read that the chest film shows overdistention. Although overdistention is common in emphysema, it also occurs in asthma and in elderly persons who have lost the elasticity in their lungs. The latter condition used to be referred to as senile emphysema, but since airway obstruction is absent and there is no disruption of the alveolar-capillary surface,

the term "emphysema" is a misnomer. Some overdistention is a normal part of the aging process in which the elastic tissue of the lung atrophies and hence allows the intrapleural pressure to distend the lung to a larger functional residual capacity.

The only radiologic finding that correlates well with the postmortem demonstration of emphysema is the presence of large proximal pulmonary arteries coupled with attenuation and paucity of the peripheral radicles of the pulmonary artery.

The presence of obstruction in emphysema can often be demonstrated by taking inspiration and expiration films. In these films the grossly limited diaphragmatic excursion can be seen. An even more graphic demonstration of this phenomenon is evident when the patient is fluoroscoped while panting. Instead of the diaphragms moving freely, they tend to flutter and take an unduly long time to return to their normal expiratory position.

Other Forms of Emphysema. Focal emphysema occurs in coal miners. When the clearing mechanisms of the lung have been overwhelmed, coal dust aggregates around the first division of the respiratory bronchiole. This eventually produces some atrophy of the smooth muscle in the bronchiolar wall and mild dilatation takes place. This dilatation goes by the name of focal emphysema and is the cardinal lesion of simple coal workers' pneumoconiosis. Although it may produce an increased alveolar-arterial gradient, it is not associated with disability.

Bullous emphysema is characterized by the presence of thin-walled cysts in the lung. The apices are most commonly affected; however, the bases are not immune. While the blebs are often bilateral, both sides are seldom affected to the same extent. Bullae may be entirely asymptomatic and discovered only on a routine chest radiograph; under these circumstances they tend to be more common in young and middle-aged males. Much more frequently they occur in conjunction with generalized obstructive airway disease; they affect the pink puffer rather than the blue bloater. There are no specific symptoms and indeed the patient is usually asymptomatic unless he has coincident emphysema. Bullae may be suspected when the physical examination reveals an area of the lung where the percussion note is tympanitic and the breath sounds are absent or grossly diminished. The physical findings may be mistaken for a localized pneumothorax. There is little doubt that when the bleb is large, it can act as a space-occupying lesion and compress adjacent normal lung tissue. Under these circumstances resection may be helpful. Yet it must be emphasized that when dyspnea is a severe symptom, the bulla is always associated with generalized obstructive airway disease and the difficulty lies in deciding how much of the patient's impairment is a consequence of the bleb and how much is a consequence of the chronic bronchitis and emphysema.

Compensatory emphysema is seen after lobectomy or segmental resection; this is not emphysema in the true sense, since there is no disruption of the remaining alveolar-capillary surface. It does not produce symptoms, and if shortness of breath occurs, it is inevitably an indication that the remaining unresected lung tissue had compromised function prior to the resection.

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