### *Other Forms of Emphysema*

The following are conditions to which the term emphysema is applied because they are associated with lung overinflation or focal emphysematous change

- ***Compensatory hyperinflation.*** This term is sometimes used to designate dilation of alveoli in response to loss of lung substance elsewhere. It is best exemplified by the hyperexpansion of residual lung parenchyma following surgical removal of a diseased lung or lobe.
- ***Obstructive overinflation.*** In this condition the lung expands because air is trapped within it. A common cause is subtotal obstruction of the airways by a tumor or foreign object. Another example is *congenital lobar overinflation* in infants, probably resulting from hypoplasia of bronchial cartilage and sometimes associated with other congenital cardiac and lung abnormalities. Overinflation in obstructive lesions occurs either (1) because the obstructive agent acts as ball valve, allowing air to enter on inspiration while preventing its exodus on expiration, or (2) because *collaterals* bring in air behind the obstruction. These collaterals consist of the *pores of Kohn* and other direct accessory bronchioloalveolar connections (the *canals of Lambert*). Obstructive overinflation can be a life-threatening emergency, because the affected portion distends sufficiently to compress the remaining lung.
- ***Bullous emphysema.*** This is a descriptive term for large subpleural blebs or bullae (spaces greater than 1 cm in diameter in the distended state) that can occur in any form of emphysema (Fig. 15-9). These localized accentuations of emphysema occur near the apex, sometimes near old tuberculous scarring. On occasion, rupture of the bullae may give rise to pneumothorax.
- ***Interstitial emphysema.*** Entrance of air into the connective tissue stroma of the lung, mediastinum, or subcutaneous tissue produces *interstitial emphysema.* In most instances, alveolar tears in pulmonary emphysema provide the avenue of entrance of air into the stroma of the lung, but rarely, chest wounds that allow entry of air or fractured ribs that puncture the lung substance underlie this disorder. Alveolar tears are usually caused by rapid increases in pressure within alveolar sacs, such as occurs when there is a combination of coughing and bronchiolar obstruction. Premature children on positive pressure ventilation adults who are being artificially ventilated are most at risk.

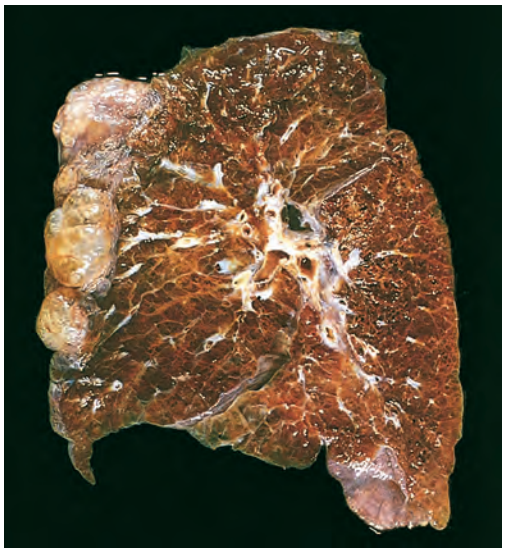

**Figure 15-9** Bullous emphysema. Note the large subpleural bullae *(upper left)*.

## Chronic Bronchitis

**Chronic bronchitis is defined clinically as persistent cough with sputum production for at least 3 months in at least 2 consecutive years, in the absence of any other identifiable cause.** Common in habitual smokers and inhabitants of smog-laden cities, chronic bronchitis is one end of the spectrum of COPD, with emphysema being the other. Most patients lie somewhere in between, having features of both. When chronic bronchitis persists for years, it may accelerate decline in lung function, lead to cor pulmonale and heart failure, or cause atypical metaplasia and dysplasia of the respiratory epithelium, providing a rich soil for cancerous transformation.

***Pathogenesis.*** The primary or initiating factor in the genesis of chronic bronchitis is exposure to noxious or irritating inhaled substances such as tobacco smoke (90% of patients are smokers) and dust from grain, cotton, and silica.

- *Mucus hypersecretion.* The earliest feature of chronic bronchitis is hypersecretion of mucus in the large airways, associated with hypertrophy of the submucosal glands in the trachea and bronchi. The basis for mucus hypersecretion is incompletely understood, but it appears to involve inflammatory mediators such as histamine and IL-13. With time, there is also a marked increase in goblet cells in small airways—small bronchi and bronchioles—leading to excessive mucus production that contributes to airway obstruction. It is thought that both the submucosal gland hypertrophy and the increase in goblet cells are protective reactions against tobacco smoke or other pollutants (e.g., sulfur dioxide and nitrogen dioxide).
- *Inflammation.* Inhalants that induce chronic bronchitis cause cellular damage, eliciting both acute and chronic inflammatory responses involving neutrophils, lymphocytes, and macrophages. Long-standing inflammation and accompanying fibrosis involving small airways (small bronchi and bronchioles, less than 2 to 3 mm in diameter) can also lead to chronic airway obstruction. This feature is similar to that described earlier in emphysema and is a common denominator in COPD.
- *Infection.* Infection does not initiate chronic bronchitis, but is probably significant in maintaining it and may be critical in producing acute exacerbations.

It should be recognized that cigarette smoke predisposes to chronic bronchitis in several ways. Not only does it damage airway lining cells, leading to chronic inflammation, but it also interferes with the ciliary action of the respiratory epithelium, preventing the clearance of mucus and increasing the risk of infection.