

see Robbins Basic Pathology 7th ed. Pp. 454 - 465

ATELECTASIS – Collapse of a lung

Resorption atelectasis (obstruction of airway, air resorbed)

Compression atelectasis (lung compressed)

Microatelectasis (loss of surfactant in ARDS or in babies with hyaline membrane dz.)

This term refers to collapse of the lung which may be partial, such as 25 or 50% collapse, or may be total, 100%. Air or fluid in the pleural space may compress and collapse the lung. Air may come from a hole in the chest wall or from the lung itself through a hole in the visceral pleura. Loss of surfactant may also lead to collapse. Obstruction of a large airway may lead to atelectasis. If blood is still flowing through the lung and no more air is getting in, the O₂ will be absorbed quickly, and eventually the nitrogen will too.

OBSTRUCTIVE LUNG DISEASE

The obstructive lung diseases listed in Robbins (top, left p. 455) are

- Asthma
- Emphysema
- Chronic bronchitis
- Bronchiectasis
- Cystic fibrosis
- Bronchiolitis

I don't object to this, but I'd like to offer a few comments.

Think of **asthma** as acute airway narrowing (constriction) leading to wheezing. Occasional. Reversible. (bronchodilator, it goes away) No chronic inflammatory changes.

However, sometimes it is chronic, occurring almost every day. In this case, chronic bronchial inflammatory changes occur, with overproduction of mucus, and this is then a chronic obstructive disease.

The term **chronic bronchitis** is not what it seems. It is a clinical term meaning cough and phlegm. It does not imply chronic inflammatory cells (lymphocytes, macrophages) in the bronchial wall.

Bronchiectasis is a distortion of airways, usually localized, and leads to recurrent infections in the part of the lung involved. Since it is not diffuse (like the other diseases in the list usually are), it may be locally severe, but not cause much functional obstruction to airflow. It fits well with the topics of aspiration and abscess, but also fits here.

Cystic fibrosis is a generalized disease that can lead to chronic bronchitis, bronchiolitis, bronchiectasis, pneumonia. In other words, it might fit better as a cause of some of the other things listed than as a type, itself, of COPD.

This is picky, but I have to say it: Getting back to asthma, on p. 457 under “morphology,” you find the sentence “Histologically, the mucous plugs contain whorls of shed epithelium (Curschmann’s spirals).” I think of Curschmann’s spirals as a condensation and alteration in chronically inspissated mucus, not as epithelium. The epithelium does shed (eosinophil major basic protein loosens epithelial attachment from the basement membrane). The shed clusters of epithelium found in mucus / sputum in asthma are called Creola bodies. By the way, a note on the words “mucus” and “mucous”. The former is a noun, latter an adjective. Mucus comes from mucous glands. (‘snot always what it seems.)

So - - - in the bronchus in asthma, you see
Inflammatory cells of all kinds, especially eosinophils
Edema
Bigger mucous glands
More mucous cells in the glands and on bronchial surfaces (goblet cells)
Plugs of mucus in the airway lumen
Thick basement membrane (scarring just under normal basement membrane)
Thick smooth muscle (been working overtime, so becomes hypertrophied)

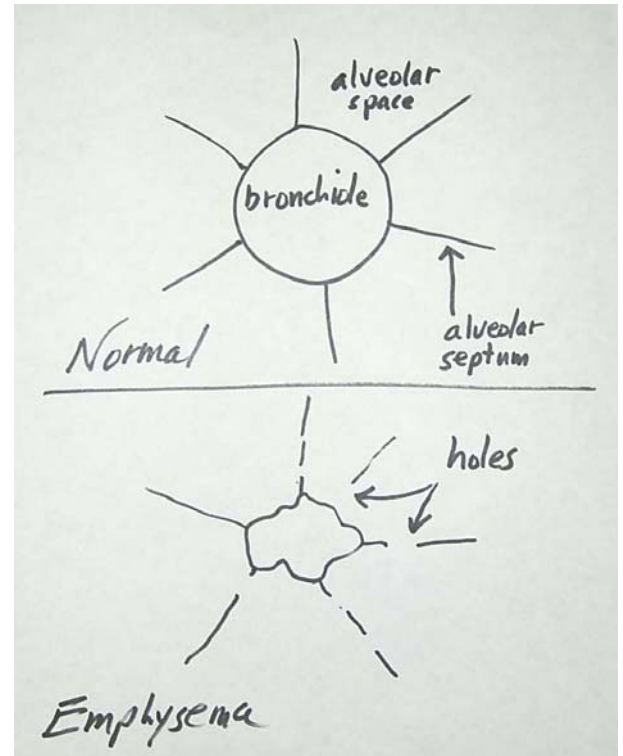
Byssinosis is an occupational asthma also known as “Monday asthma” and is characterized by bronchoconstriction resulting from inhalation of cotton mill dust and certain other vegetable dusts. This seems to be a chemical drug-like response rather than an allergic reaction. Person goes to work in a cotton mill (especially carding room) and gets short of breath on Mondays. The reaction is less on other days of the week, but after recovery, being away from dust over the weekend, it recurs the next Monday. Because of medical monitoring and dust control, this disorder is seldom seen in today’s modern American textile mills, although it still occurs elsewhere. Prolonged cases may result in COPD.

Emphysema: pp. 458-463. There’s a lot packed into these pages. Let me accentuate a few things. By a mechanism of proteolysis, the alveolar walls in emphysema dissolve resulting holes in the alveolar walls, eventually bigger holes in the lungs, and finally reduced surface area for gas exchange. The proteolytic enzymes come from neutrophils and macrophages. There is prominent obstruction to airflow, especially on exhalation, resulting in trapping of air in the lung. As a result, the lungs get big, so much so that the whole chest wall, including the ribs, is deformed (Barrel chest). Fluffy soft lungs bent the rigid bones. Most emphysema is caused by cigarettes. Common form is “centrilobular” (“centriacinar”). Prominent punched out holes. Mild case is illustrated in fig. 13 – 6 A. “Panacinar” emphysema may also be caused by cigarettes, but when it’s very bad, you might suspect congenital alpha-1 antitrypsin deficiency. Cigarettes increase the numbers of inflammatory cells which contain proteolytic enzymes, thus increasing the amount of enzyme to a point that the normal circulating anti-enzyme (alpha-1 antitrypsin) is overwhelmed and a hole appears in an alveolar wall. Enough cigarettes and enough holes, and you can lose a lot of lung. In congenital alpha-1 antitrypsin deficiency, there is a primary lack of the antiproteolytic controlling substance, so even a normal amount of inflammatory cell proteolytic enzyme can cause emphysema. Occasional cases in (nonsmoking) children have occurred. Obviously, if somebody has the deficiency, they, especially, should not smoke. The normal phenotype is “MM”. The usual really bad phenotype is “ZZ”. There are a lot of S’s around and a good many others. Heterozygotes (MS, MZ) are usually OK, no problems.

The physiologic obstruction in emphysema is multifactorial, but loss of support of small airways is especially important. This is mentioned in Robbins in the Morphology section on page 462 and a crude diagram follows at the end of this handout.

“Chronic bronchitis” is common but usually doesn’t cause much functional disability. It is, by definition, productive cough for 3 consecutive months, in 2 consecutive years. The corresponding pathology is enlargement of bronchial mucous glands, which is the result of an activated mucin gene mediated by EGF. Chronic bronchitis is usually caused by smoking, but can result from atmospheric pollution, noxious gases, viral infections, etc. It is one of the constituents of chronic asthma, as previously noted.

Bronchiectasis has been previously discussed. It is a permanent distortion of the airways which may be saccular or cylindrical, and is often caused by aspiration of foreign material, by certain infections such as whooping cough, staph, or klebsiella. Bronchiectasis is common in longstanding CF, also in longstanding severe sarcoidosis or TB. It is sometimes the result of one of the ciliary dysmotility syndromes, eg. Kartagener’s syndrome. Because of poorer drainage from the lower lobes (gravity), bronchiectasis is usually worse there. Generally, 2 things have to happen to cause bronchiectasis. First, the bronchus has to be dilated, often because of obstruction by a mucus plug. Secondly, the bronchial wall has to get infected resulting in scarring which then “locks” the airway in its dilated, distorted shape.



Some questions you might ask after reading this are:

“What is an acinus? What are the different types of emphysema and where in the acinus are they found? What does a person with emphysema look like? In general, what would PFT’s (pulmonary function tests) show? What changes would be found in arterial blood gases? Most emphysema is caused by cigarettes. What is another cause? How does it differ from the usual cigarette-smoke type? What changes are seen in the bronchial wall in chronic asthma? What causes bronchiectasis? Can I name 4 things? What are complications of bronchiectasis?”