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A.C. Hinshaw, J.F. Murray and H.C. Hinshaw, Jr., Diseases of the Chest, 4th edition, Philadelphia: WB Saunders 1980

28 PNEUMOCONIOSES AND RELATED DISEASES

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INTRODUCTION

An industrial society uses and produces a vast number of substances that are capable of injuring the lungs. Because there is also a large number of substances with little or no pulmonary toxicity, the mere existence of some type of exposure at the place of employment of a person with lung disease does not necessarily mean that the disease is related to the occupation, although the physician must consider this possibility in the differential diagnosis. If the occupation is the cause of the lung disease, the diagnosis as well as the relationship of the disease to the exact work exposure must be as precise as possible, both to ensure that the patient receives appropriate treatment and

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compensation and to define the hazard for other workers. Unfortunately it is not always possible to make a definite diagnosis or to obtain the exact details of the industrial exposure, especially if the contact occurred many years previously. In most cases, reasonably accurate decisions can be made, but these require knowledge of how the lungs react to various substances and what types of exposure are likely to occur in various occupations. It is just as wrong to make an incorrect diagnosis of an occupational illness, and hence possibly change a productive member of society into a nonproductive one, as it is to fail to make such a diagnosis when it is justified and deny the victim the benefits of specific treatment, when available, and compensation, when warranted.

The list of known agents capable of injuring the lungs is now very long and is continuing to grow. It is not possible to provide an encyclopedic compendium of all possible injurious substances in this book. It is also not possible to list all the many variants, or alleged variants, of occupational lung disease. In several instances, the same basic disease has a host of different names with new names being added as the same disease is discovered in previously unreported occupations. The objective of this chapter is to review the epidemiology, pathogenesis, diagnosis, and treatment of the most common types of pneumoconiosis and related diseases. Other lung diseases of occupational origin are described in the next two chapters.

The definition of pneumoconiosis has two components: (1) the permanent retention of inhaled dust in the lungs and (2) the tissue reaction to its presence. The pathologic response to dusts varies greatly depending on the kind of dust and on the amount retained. Dust that is not retained can also produce disease, but this is not included as a pneumoconiosis. It must be emphasized that the term pneumoconiosis refers to a heterogeneous group of different specific diseases. A simple diagnosis of pneumoconiosis is not clinically or epidemiologically useful; it is necessary to make a diagnosis of a specific disease, such as silicosis, asbestosis, or coal-workers' pneumoconiosis.

GENERAL CONSIDERATIONS

The respiratory tract is designed to protect the lungs from the effects of inhaled foreign substances; thus most dusts are filtered out in the nasal passages or trapped in the mucous blanket of the tracheobronchial tree. For dust to be retained permanently, it must reach the smallest air passages or the alveoli. The principal physical characteristic that determines penetration into the lungs is the size of the inspired particle. Particles greater than 10 μm in size are removed from the airstream before they reach the small airways or their terminal respiratory units. Hence, the potential of developing pneumoconiosis exists only when particles less than 10 μm are inhaled. Very small particles (less than 1 μm) do not settle out in the lung easily but tend to remain suspended in the air and, because they act more like a gas than a solid, to be exhaled. Thus the most dangerous dusts (i.e., those most likely to be retained) are those 1 to 5 μm; these are often called respirable particles. The importance of the size of the particles cannot be overemphasized. Most occupationally generated dusts, and virtually all naturally occurring dusts, are much too large to cause pneumoconiosis. To produce dust particles small enough to cause disease generally requires disruptive

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alteration of the natural mineral; in other words, some operation such as crushing, grinding, milling, drilling, cutting, or smoothing is usually involved.

Only a relatively few specific substances, compared to the large number of substances to which workers are exposed, seem to be capable of eliciting sufficient biologic reaction in the lung to produce detectable disease. Many kinds of dust, even if inhaled in small enough particles to be deposited, do not produce pneumoconiosis.

From these considerations, it is clear that for clinical pneumoconiosis to develop, three essential factors are required: (1) exposure to a specific substance or substances capable of causing disease; (2) exposure to particles that are of the appropriate size to be retained in the lungs; and (3) exposure for a sufficient length of time (to a concentration of appropriately sized particles) to retain a dust burden capable of causing detectable disease. If any of these three factors is absent, then pneumoconiosis is not present and some other cause for a particular patient's illness or roentgenographic findings must be found. Conversely, if all three are present and the clinical and roentgenographic abnormalities are consistent with the type and degree of exposure, then a diagnosis of pneumoconiosis can be made confidently, without the need for more specific procedures such as lung biopsy.

Most cases of pneumoconiosis represent significant exposure to only one type of injurious substance, but there will be some cases of mixed exposure, with a possibly confusing clinical or roentgenographic appearance. Of particular concern is the contributing role of nonoccupational injuries, particularly smoking and infection, in the pathogenesis of a patient's lung disease. Tuberculosis, formerly a scourge of patients with silicosis, is no longer a major problem; but the damage related to cigarette smoking—emphysema, chronic bronchitis, and pulmonary malignancies—compounds and may outweigh the effect of inhaled dusts.

Pathogenesis

It is useful to divide occupational lung damage into four basic pathologic mechanisms: (1) retention of particles, (2) direct injury, (3) immunologic damage, and (4) carcinogenesis. It should be kept in mind that substances may act by one mechanism in some persons and by another in others; at times, two or more mechanisms are involved. Physicians should understand the four processes if they are to evaluate either individual patients suspected of having occupational lung disease or reports of new diseases or new agents claimed to be responsible for producing lung injury.

RETENTION OF PARTICLES

Depending on the chemical and physical properties of the particular substance, actual deposition and permanent retention of particles in the lung and/or the draining lymphatics may or may not produce damage to the lung. When damage is produced, it is in the form of an inflammatory reaction with fibrosis. The classic pneumoconioses were originally believed to fall mainly into this group; however, immunologic mechanisms are also important and may account for some or possibly all of the damage. Because the material is retained perma-

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nently, it is capable of exerting a continuing biologic effect for the lifetime of the person. Thus progression of disease may continue long after exposure has ceased.

DIRECT INJURY

Another mechanism of occupational lung disease involves actual chemical or physical injury to the mucosal lining of the tracheobronchial tree or lung parenchyma. The site of reaction and its severity depend on the physical and chemical nature of the offending substance and on its concentration in the inspired air. Additional discussion concerning the importance of these factors is provided in Chapter 30 (Chemical, Radiation, Thermal, and Aspiration Injuries). Frequently, complete recovery takes place after a single exposure, but permanent injury can occur in severe cases. Because the material is not retained in the lung, when exposure ceases, no further damage will occur.

IMMUNOLOGIC DAMAGE

Injury to the lung parenchyma and/or airways may be produced by some types of immunologic responses (see Chapter 21 for a complete description of the four types of immunologic responses). Various terms are used to refer to immunologically mediated injury (e.g., allergy and hypersensitivity). The meanings of these terms differ and it is best to classify all reactions of this sort under the general heading of immunologic responses, as long as it is realized that in some situations the exact basis of the reaction may not be defined. Immunologically mediated reactions are generally self-limited and cease soon after the exposure ceases. Permanent injury can result from severe or repeated exposure, but with one important exception, the original reactions subside when the offending substance is no longer present in the inspired air. The exception occurs with agents that are permanently retained in the lung and that incite an immunologic reaction; in this instance, the reaction may continue long after actual exposure has ceased.

CARCINOGENESIS

The number of substances definitely known at the present time to be carcinogenic to the lungs is small, but more are likely to be added to the list in the future. One general principle of carcinogenesis needs to be emphasized: it takes prolonged contact with tissue (generally many years) for a carcinogen to produce a malignancy. Hence, in the case of substances not retained in the lungs, years of repeated exposure are needed to produce a neoplasm. When carcinogenic substances are permanently retained in the lungs, the tissue is continuously exposed to the substance for many years after occupational contact, and malignancy can develop many years later.

Roentgenographic Classification

It is important to have some standard format for evaluating the relative severity of pneumoconiosis by the roentgenographic appearance. This is needed both for evaluation of individual patients and for comparison purposes to enable

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TABLE 28–1. ILO U/C INTERNATIONAL CLASSIFICATION OF RADIOGRAPHS OF PNEUMOCONIOSES 1971 OUTLINE OF CLASSIFICATION

Feature	Short Classification	Extended Classification
No pneumoconiosis	0	Rounded 0/–, 0/0, 0/1
Pneumoconiosis:		
Small Rounded Opacities		
Profusion*	1, 2, 3	1/0, 1/1, 1/2; 2/1, 2/2, 2/3; 3/2, 3/3, 3/4
Type	p, q(m), r(n)	p, q(m), r(n)
Extent	–	zones: Right, Left; Upper, Middle, Lower
Small Irregular Opacities		
Profusion*	1, 2, 3	1/0, 1/1, 1/2; 2/1, 2/2, 2/3; 3/2, 3/3, 3/4
Type	s, t, u	s, t, u
Extent	–	zones: Right, Left; Upper, Middle, Lower
Large Opacities		
Size	A, B, C	A, B, C
Type	–	wd (well defined), id (ill defined)
Pleural Thickening		
Costophrenic angle	–	Right, Left
Walls and diaphragm		
Site		Right, Left
Width	pl	a, b, c,
Extent		1, 2
Diaphragm Outline	–	Ill defined: Right, Left
Cardiac Outline	–	Ill defined: 1, 2, 3
Pleural Calcification		
Site	plc	Walls, Diaphragm, Other; Right, Left
Extent		Length: 1, 2, 3

Symbols for Additional Abnormalities

ax Coalescence of small rounded pneumoconiotic opacities
bu Bullae
ca Cancer of lung or pleura
cn Calcification in small pneumoconiotic opacities
co Abnormality of cardiac size or shape
cp Cor pulmonale
cv Cavity
di Marked distortion of the intrathoracic organs
ef Effusion
em Marked emphysema
es Eggshell calcification of hilar or mediastinal lymph nodes
hi Enlargement of hilar or mediastinal lymph nodes
ho Honeycomb lung
k Septal (Kerley) lines
od Other significant disease. This includes disease not related to dust exposure, e.g., surgical or traumatic damage to chest walls, bronchiectasis, etc.
pq Pleural plaque (uncalcified)
px Pneumothorax
rl Rheumatoid pneumoconiosis (Caplan's syndrome)
tab Tuberculosis, probably active
tub Tuberculosis, activity uncertain

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meaningful epidemiologic studies. In the past, the systems used for roentgenographic classification have been of limited usefulness. They have not been applicable to all pneumoconioses; they have been too simple in view of the wide range of diseases present; and the various levels of classification had inadequate relationships to the actual clinical status of the patient.

In 1971, a combined International Labor Organization-University of Cincinnati (ILO U/C) classification was adopted that corrects as much as possible the deficiencies of previous systems. This is now the standard, internationally recognized classification (Table 28–1). Of necessity, it is relatively complicated, and the use of the full system requires considerably more experience than the average physician has accumulated. An understanding of the basic principles of the classification is important because it is frequently referred to in reports of individual patients as well as in articles in medical journals. Numbers from 0 to 3 are used to describe the profusion of densities present, and various letters are used to specify the type of density or densities present on a particular roentgenogram.

CATEGORIES OF SEVERITY

There are four main categories used to designate the extent of small opacities: 0 is normal, 1 is minimal disease, and 2 and 3 refer to increasingly more advanced disease with increasing extent of densities on the roentgenogram. Because there are no sharp dividing lines between these four and because when reading a roentgenogram one often seriously considers a definition in either a higher or a lower category than the one eventually chosen, the four basic categories are further divided into a total of 12 categories using two numbers with a "/" between them. The first number is the category the film reader eventually decides to place the film in; the second number is the other category into which the reader considered classifying the film. Thus category 0/1 refers to a roentgenogram read as normal, but serious consideration was given to designating it in category 1. Category 1/0

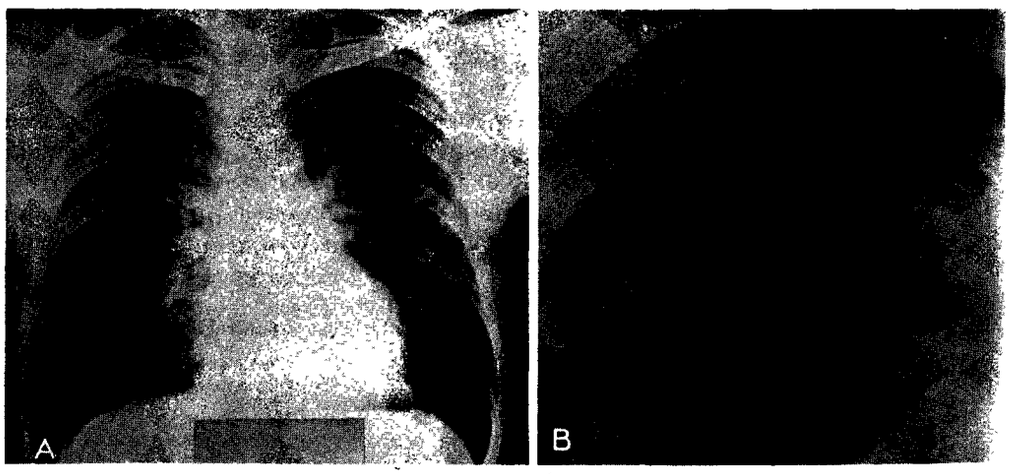


Figure 28–1. Pneumoconiosis (Category 1/1 type p)

Chest roentgenogram (*A*) of a 42-year-old coal miner showing category 1/1-p simple pneumoconiosis. Detailed view (*B*) showing 0.5 to 1.5 mm opacities.

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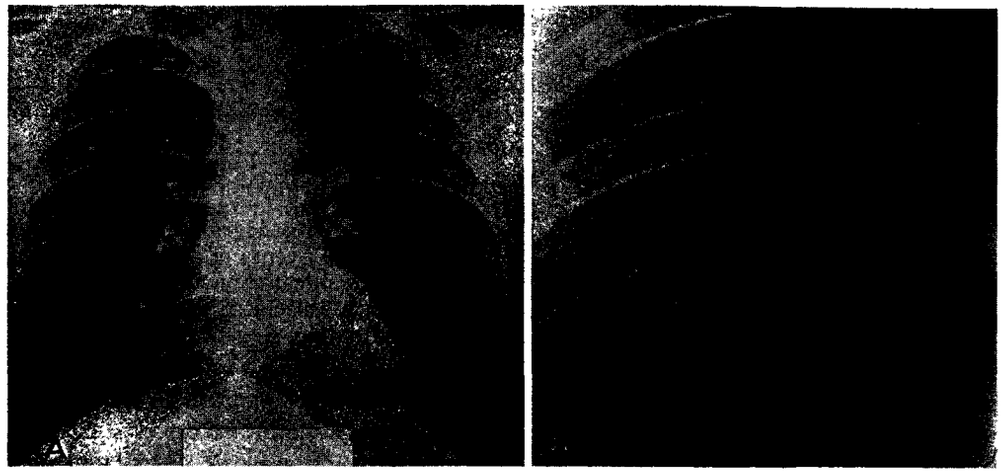


Figure 28–2. Pneumoconiosis (Category 2/2 type q)

Chest roentgenogram (*A*) of a 31-year-old coal miner showing category 2/2-q simple pneumoconiosis. Detailed view (*B*) showing 1.5 to 3 mm opacities in greater profusion than in Figure 28–1.

is read as abnormal, category 1, but consideration was given to calling it normal. Category 1/1 is unquestionably category 1, whereas 1/2 defines category 1, but consideration was given to classifying it in the higher category 2. The same principle is used on up the scale. Examples of the use of this classification are presented in Figures 28–1 to 28–3.

TYPES OF OPACITY

The sizes and shapes of individual opacities are designated by various letters. Because roentgenograms may contain several different types or sizes of opacities,



Figure 28–3. Pneumoconiosis (Category 3/2 type q)

Chest roentgenogram (*B*) of a 45-year-old coal miner showing category 3/2-q simple pneumoconiosis. Detailed view (*B*) showing 1.5 to 3 mm opacities in slightly greater profusion than in Figure 28–2.

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Figure 28–4. Complicated Pneumoconiosis (C Large Opacities)

Chest roentgenogram of a 68-year-old miner, with 31 years of exposure to silica, showing complicated pneumoconiosis, category C with associated paraseptal emphysema and bullae.

classification is by threes: three kinds of opacity, each divided into three subtypes.

"Small rounded opacities," those less than 1 cm, are classified according to the greatest diameter of the predominant lesions and are denoted by the following letters: "p" refers to opacities up to and including 1.5 mm, "q" to those from 1.5 up to and including 3 mm, and "r" to those from 3 up to and including 10 mm.

"Small irregular opacities" are those likely to be seen in such diseases as asbestosis. They are divided according to thickness and texture: "s" refers to fine irregular (linear) opacities, "t" to medium irregular opacities, and "u" to coarse irregular opacities.

"Large opacities" are defined as any opacity greater than 1 cm. Films with such opacities are further designated as representing complicated pneumoconiosis—as opposed to simple pneumoconiosis, in which no opacities over 1 cm are present. Large opacities are also divided into three subdivisions according to size: "A" is one or more opacities between 1 and 5 cm, with the sum of the diameters of all such opacities on the film being less than 5 cm; "B" is one or more opacities larger or more numerous than those in category A but with a combined area that is less than one third of the right lung; and "C" is one or more such opacities with a combined area that is greater than one third of the right lung (Figure 28–4).

Additional roentgenographic abnormalities, which should be identified, include involvement of the pleura (effusion or thickening with or without calcification), hilar lymph nodes (enlargement with or without calcification), distortion of airways, and presence of cancer, cavitation, pneumothorax, or tuberculosis (probably active or activity uncertain).

Control Measures

Almost all occupational pulmonary disorders result from workers breathing substances present in the ambient air. Prevention of exposure and subsequent disease is, of course, preferable to attempting to treat it after it has occurred. Prevention is especially important in the pneumoconioses because most of the

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damage is permanent and treatment is almost useless. Furthermore, symptoms often are not significant until advanced and irreversible disease is present. In the United States, recent legislation at both the federal and state levels has been enacted that is designed to protect workers from occupational hazards. Industry also now realizes that protecting the health of its workers is not only desirable on humanitarian grounds but is good business in the long run. There are three main ways to control exposure, which are listed in order of decreasing desirability: engineering or legislative controls, ventilatory controls, and individual worker controls.

ENGINEERING OR LEGISLATIVE CONTROLS

The most desirable control is complete avoidance of the hazardous substance. This can be accomplished by prohibiting use of the material or altering the manufacturing process so that there is no possibility of potentially damaging exposure of an unprotected worker. In some countries, silicosis has been prevented by legislation that prohibits the use of silica particles in sand blasting; this ensures the substitution of an innocuous, although often more expensive, material.

VENTILATORY CONTROLS

If engineering controls are not possible, or if they are only partially successful in eliminating exposure and the substance must be used, the next step is to design some kind of ventilation or exhaust system so that the hazardous materials are removed from the air before they reach the worker.

INDIVIDUAL WORKER CONTROLS

If neither engineering nor ventilatory controls are feasible, then the individual worker must wear some kind of protective device that ensures that inspired air is not hazardous. There are two principal ways to accomplish this. (1) Use of some type of mask and filter that removes the hazardous substance from the air. These are self-contained and allow free mobility of the worker, but they often impede the flow of inspired air so that, if a worker is doing heavy work, there may be difficulty in obtaining a sufficient minute volume. The particular device must be specifically designed to protect against the known hazard or hazards present. For example, a simple filter may protect against many dusts, but may not be fine enough to remove the small particles that cause pneumoconiosis. Moreover, a filter designed to eliminate small particles may be worthless as protection against gases or vapors. (2) Provision of a separate fresh air supply with a hood and a pump. This can be quite effective in eliminating a hazard, but cumbersome equipment and hoses are needed. If the worker is stationary, this type of apparatus may work well; but if the worker has to move about, such devices may be impractical.

Assessment of Hazards

Accurate measurements of the concentration of potentially hazardous substances in the air are of considerable importance. Specific measurement is the

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only way to determine definitely that a hazard is or is not present and is the only way to ensure that control measures are, in fact, effective. It is important that the actual air that the worker is breathing be analyzed rather than relying on measurements taken from fixed locations, which may not necessarily reflect what the worker is exposed to. Measuring devices are now readily available but which one is used varies with the nature of the material being studied. One of the most useful instruments is a small, battery-operated pump that is attached to the worker's clothing. A small collecting tube is placed near the worker's nose and mouth within the "breathing zone." Air from this site is pumped at a known rate through some kind of collecting device that is designed to remove the substance(s) being studied. The worker performs his usual duties while the air he breathes is being sampled for a measured period of time, at least several hours, but preferably for the whole work shift. The amount of material collected is then measured, and because the rate of flow generated by the pump and the total time are known, the average concentration of the substance that the worker breathed can be calculated. Several other devices are available to sample air in occupational environments. The collection principles vary, but all provide the same information: the amount of material collected during a known amount of time.

From these data a value called a time-weighted average (TWA) can be calculated; this reflects the average amount of the substance the worker is exposed to during a normal 8-hour working day. This is expressed as weight of substance or number of particles per unit volume of air. Governmental bureaus and private occupational health organizations have established standards known as threshold limit values (TLVs) for many substances. The TLV is that amount that current knowledge indicates a worker could be exposed to for 8 hours a day for a normal working life without suffering significant injury. If the TWA is less than the TLV, it is assumed that no hazard is present. If the TWA is greater than the TLV, a potential hazard does exist and control measures must be instituted.

SILICOSIS

Many persons do not understand the meaning of words such as silicon, silica, and silicates, which are used in discussions of the disease silicosis. The word *silicon* refers to the element silicon, the second most common and widely distributed element in the earth's crust; only the element oxygen is more common. Silicon does not occur naturally as a free element but as various compounds, most commonly as silicon dioxide (quartz, cristobalite, and tridymite, depending on the crystalline form) and also as various silicates. Many artificially produced compounds containing the element silicon are used in industry. The term *silica* refers only to one specific compound of silicon, silicon dioxide. The terms silica and silicon dioxide are used interchangeably in articles and discussions about silicosis. The uncombined forms of silicon dioxide are often collectively referred to as *free silica* to distinguish them from *silicates,* which contain cations and occur in chains and other complex structures. The term *silicosis* refers to one specific disease that is produced by exposure to silica and not to any other specific compounds of silicon. Some of the naturally occurring silicates can cause other pneumoconioses, but not silicosis.

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Epidemiology

Because silica is so abundant and widely distributed, hazardous exposure can occur in a wide variety of occupations. Traditionally, silicosis has been a disease of miners. Many rocks contain silica and some, such as quartz and sandstone, are virtually pure silica. Thus occupations that involve mining or tunneling in rock may result in exposure to free silica. Quarrying, chipping, grinding, and polishing silica-containing rock are also hazardous. Sandblasting, which is used extensively in ship building operations and in the maintenance of offshore oil rigs, can lead to severe exposure to silica of nearby workers as well as to the person doing the sandblasting. In parts of the United States and in Western Europe, "sandblasting" is usually performed with abrasives other than sand, a measure that has helped prevent silicosis.

In previous years the foundry and pottery industries produced many cases of silicosis. Finely ground sand was used in foundries as a parting compound. Many clays contain silica, and ground flint was once used in the pottery industry. Although the hazard in these industries has been substantially reduced by abandoning the use of the finely ground sand and flint, potential hazards still exist, especially to workers doing grinding and polishing operations. Ordinary brick does not represent a silicosis hazard, but heat-resistant, or "fire," brick contains considerable silica; thus the cutting or manufacture of this type of material can produce silicosis. Heating diatomaceous earth produces high concentrations of free silica of a particularly dangerous variety. Recognition of the hazards of silicosis resulted in the elimination or control of exposure in many industries, but despite its being a preventable disease, new cases of silicosis continue to occur.

Pathogenesis

When respirable particles (mainly 1 to 5 μm) of free silica are inhaled, some reach the terminal airways and alveolar surface. There, they are ingested by macrophages, which are promptly killed by the cytotoxic effects of the silica. More macrophages enter the area to ingest the liberated silica particles, but these phagocytes also die. This silica-induced death of macrophages results in the release of lysosomal enzymes and appears to induce the progressive formation of collagen, which eventually hyalinizes. Many of the silica particles, either free or in macophages, travel in the lymphatic system to become trapped in lymph nodes in the hilum; some silica may reach the mediastinal or even the supraclavicular nodes. Particles may extend to the pleural surfaces and cause fibrotic adhesions between the visceral and parietal pleura, often with eventual obliteration of the pleural space.

The parenchymal granulomatous reaction develops into fibrotic nodules. *Simple silicosis* typically consists of small nodules, generally less than a few millimeters in diameter. These nodules are scattered through the lungs but tend to be more numerous in the upper lung zones. Eventually, some nodules may become calcified. In some cases *complicated silicosis* occurs: large conglomerate masses of fibrotic tissue develop and cavitation may occur. Complicated silicosis previously resulted from the additional insult of coexisting infection by *Mycobacterium tuberculosis*. Now that these infections are less common, it is recognized that

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a similar response can be produced by other infections (e.g., other mycobacteria, fungi, and *Nocardia*) and from immunologic reactions.

In almost all cases, roentgenographically detectable disease develops only after 20 or more years of constant daily exposure. There is a reasonable correlation between the extent of fibrosis and the quantity of silica recoverable from the lungs. Unexposed persons have less than 0.25 gm, whereas patients with silicosis have 15 to 20 gm of silica in their lungs. Thus some silica is likely to be present in normal lungs and even a modest excess above normal (doubling or tripling) does not produce significant disease. Under certain circumstances, acute silicosis can develop after only 1 to 3 years of exposure and accelerated silicosis after 4 to 10 years of exposure. When the onset is earlier and the rate of progression faster than expected, probably either extraordinary work conditions (lack of protection or massive exposure) or coexisting infectious or immunologic complications are present.

Clinical, Laboratory, and Roentgenographic Findings

The clinical, laboratory, and roentgenographic abnormalities in patients with silicosis depend on the extent and duration of exposure and the presence of coexisting infectious or immunologic complications. Patients with roentgenographically detectable disease may be completely asymptomatic or may be extremely disabled from respiratory failure and/or cor pulmonale with heart failure.

CLINICAL FINDINGS

Most cases of simple silicosis have few or no symptoms or abnormalities in routine pulmonary function tests. However, some patients with silicosis have coexisting pulmonary problems that make it difficult to evaluate the specific contribution from silicosis. Many miners are heavy cigarette smokers and have the usual pulmonary problems associated with this habit; there also appears to be an increased incidence of chronic bronchitis among miners, which may be another effect of their exposure to dust. The most common symptoms in patients with silicosis are cough and sputum production; these are presumably caused by smoking or dusts other than silica. The chief symptom of "pure" silicosis is shortness of breath of varying severity.

Category 1 disease (see the ILO U/C classification, described earlier) produces no symptoms and no measurable functional abnormalities. Many patients with category 2 disease and some with category 3 also lack significant symptoms or functional abnormalities. As simple silicosis becomes more severe, especially in category 3 patients, there is a greater incidence of symptoms of dyspnea. In addition, some pulmonary function abnormalities can be detected, generally decreased FVC and FEV_1, decreased pulmonary compliance, and decreased arterial Po_2 during exercise.

A sequence of events seen in many patients is the appearance of few or no symptoms during the patient's normal working years followed by the development of symptoms of dyspnea, with no change in the chest roentgenographic abnormalities, when the patient is 60 or 70 years of age. What probably is occurring is that the silicosis has lowered the respiratory reserve but not enough

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to be noticeable until other complicating factors, such as increased age, cardiac disease, and other disorders, begin to appear. It is also likely that there is slow progression of fibrosis for several years after exposure has ceased.

Severe effort intolerance is virtually always related to the presence of complicated silicosis: contraction of the lungs, sometimes with cavitation, is detectable by roentgenographic studies and there are corresponding physiologic abnormalities. Tests of ventilation reveal obstruction, restriction, or mixed disturbances with decreased pulmonary compliance. Diffusing capacity is also decreased and, when severe, is reflected by exercise-induced hypoxemia. In advanced cases, restrictive impairment and hypoxemia may be profound.

A frequent cause of complicated silicosis is the development of tuberculosis. Traditionally, tuberculosis was believed to be especially prevalent in patients with silicosis, and "miners' consumption" was greatly feared in the prechemotherapy days because it responded poorly to the treatment available then and was often fatal. The silicotic lung appears to be less resistant to tuberculous infection than the normal lung, possibly because of impaired macrophage function; in addition, much of the increased incidence of tuberculosis in miners was attributable to the enhanced spread of droplet infection in the close, confined conditions in underground mines.

Tuberculosis from *M. tuberculosis* in patients with chronic silicosis is much less common today than before but remains high in those with more acute forms of the disease; these patients also develop infections with other mycobacterial organisms. Approximately 25 per cent of patients with accelerated silicosis may develop mycobacterial infections; of these, approximately half are caused by *M. tuberculosis* and, depending on geographic residence, *M. intracellulare* or *M. kansasii* comprise the remainder.

Another cause of complicated silicosis and accelerated deterioration is the development of collagen vascular disease, particularly scleroderma, rheumatoid arthritis, and lupus erythematosus (see Chapter 33). The exact relationship between pneumoconiosis and connective tissue disorders is unknown, but autoimmune phenomena manifested by serologic abnormalities have been demonstrated in as many as 44 per cent of sandblasters with silicosis.

LABORATORY FINDINGS

There are no specific laboratory findings in patients with silicosis. Results of routine hemogram, urinalysis, and blood chemistries are within normal limits in patients with simple silicosis; those with the complicated variety may be polycythemic if their hypoxia is severe. Values of gamma globulin tend to be higher than normal, and tests for autoimmunity, such as the presence of antinuclear antibody (ANA), are often positive in patients with complicated, accelerated, or acute forms of silicosis.

ROENTGENOGRAPHIC FINDINGS

The diagnosis of silicosis is made by roentgenographic examination and a history of exposure in nearly all cases. Clinically significant disease does not occur in the absence of unequivocal roentgenographic abnormalities, which are usually quite characteristic, and can be differentiated from other types of pulmonary fibrosis and, with one exception, from other pneumoconioses. The roentgeno-

Pneumoconioses and related diseases (1980)
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Figure 28–5. Simple Silicosis

Chest roentgenogram of asymptomatic retired hard rock miner, with 35 years' exposure to silica, showing numerous dense 2 to 4 mm nodules in both lungs. The predominantly upper lobe distribution is evident, particularly on the left.

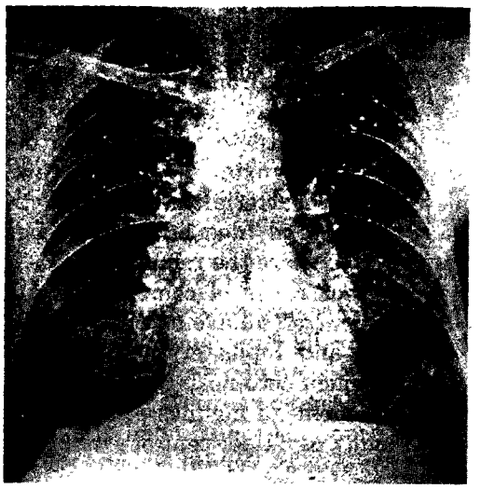


Figure 28–6. Simple Silicosis with Calcification

Chest roentgenogram of a retired hard rock miner, with 30 years' exposure to silica, showing bilateral calcified nodular densities, and bilateral hilar node enlargement with calcification.

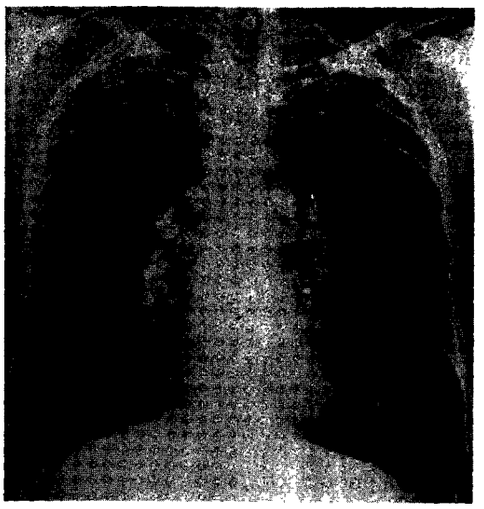


Figure 28–7. Egg Shell Calcifications

Chest roentgenogram of a 60-year-old man, who worked underground for 20 years in a hard rock mine, showing bilateral upper lobe infiltrations and bilateral enlargement of the hilar nodes with egg shell calcifications.

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-5. Simple

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showing num-
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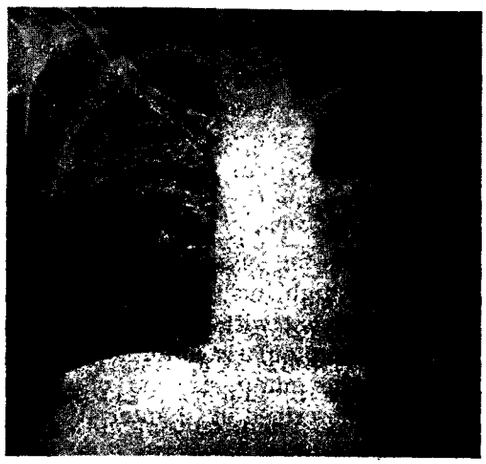


Figure 28–8. Complicated Silicosis

Chest roentgenogram of a 70-year-old former hard rock miner showing conglomerate densities characteristic of complicated silicosis. One lesion has cavitated (arrow).



graphic findings in coal workers' pneumoconiosis are often similar to those of silicosis, although the two diseases are otherwise quite different.

Simple silicosis consists of multiple nodules, usually several millimeters in diameter, scattered throughout the lungs but predominantly in the upper lung fields (Figure 28–5). The nodules may calcify (Figure 28–6). In addition, there is often enlargement of the hilar nodes, which in older cases often develop a rim of calcification producing the so-called egg shell hilar lymph nodes (Figure 28–7). When present bilaterally, egg shell hilar nodes are highly suggestive of silicosis, although the inflammatory diseases can result in similar abnormalities. As expected, the greater the exposure, the more profuse the nodules.

Complicated silicosis is manifested by large solid masses, some of which may become necrotic in the center and cavitate (Figure 28–8). These conglomerate lesions are usually bilateral and are located in the upper lobes. As they retract toward the hilum they produce the so-called "angels' wings" pattern, which may be confused with malignant disease. As upper lobe lesions conglomerate and fibrosis progresses, the upper lobes shrink and paracicatricial emphysema develops in the remaining lungs. When the latter becomes marked, the small silicotic nodules present are pulled apart and tend to become inapparent roentgenographically in the overexpanded lung. At this stage, a roentgenographic diagnosis of silicosis may be difficult because all that is seen is evidence of fibrotic destruction of the upper lobes and emphysema in the lower lobes. Rheumatoid pneumoconiosis, or Caplan's syndrome, an unusual clinical-roentgenographic constellation of findings described later in the chapter, may also occur in patients with silicosis.

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Treatment and Prognosis

The diagnosis of silicosis is nearly always made on the basis of characteristic roentgenographic abnormalities and a history of exposure. Although exposure is usually known to the patient, a detailed occupational history may be necessary to discover the source of free silica. Open lung biopsy, not transbronchial biopsy, may be required in patients whose roentgenogram or clinical course is atypical; this is much more likely to occur in the accelerated than in the chronic varieties.

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Lung tissue can be examined by polarized light, scanning electron microscopy, or x-ray energy spectrometry. In patients whose course is deteriorating, complicating mycobacterial or other infections and evidence of autoimmune disease should be sought.

There is no specific therapy for silicosis, and all therapeutic efforts are aimed at the complications. Silicotuberculosis responds well to the usual antituberculosis drugs (see Chapter 12), and most cases become permanently inactive with this treatment. There is a tendency to treat patients with silicotuberculosis more intensely and for longer periods of time than other patients with tuberculosis. In view of the high incidence of atypical mycobacterial infections in these patients, initial treatment with three antituberculous drugs, including rifampin, is recommended. Symptomatic autoimmune phenomena, if present, may respond temporarily to the administration of corticosteroids. Chronic respiratory failure is treated as described in Chapter 40.

The prognosis in cases of simple silicosis is good, although there may be slow progression over a period of many years (decades). In contrast, the prognosis in cases of the accelerated and complicated varieties is poor, with a steadily deteriorating clinical course punctuated by intercurrent episodes of bronchopulmonary infection and respiratory failure.

ASBESTOS-RELATED DISEASE

Compounds containing silicon, oxygen, often hydrogen, and one or several metallic bases, such as calcium, magnesium, aluminum, and iron, are referred to as silicates. There are many naturally occurring silicates; many others have been created artificially. Most of these compounds are harmless and do not produce pneumoconiosis, a feature that enhances their commercial usefulness. There are, however, some naturally occurring silicates that do produce biologic effects when inhaled and these few can lead to pneumoconiosis and other serious problems. This section deals with pulmonary and extrapulmonary diseases related to exposure to asbestos. Pneumoconiosis from nonasbestos silicates is discussed in a later section. According to common usage, the term asbestosis refers *only* to asbestos-induced interstitial fibrosis and not to other asbestos-related diseases; this policy is observed in this chapter.

Epidemiology

Asbestos is not a single substance. The word refers to a number of different silicates that have similar characteristics. They are fibrous in nature and are remarkably resistant to chemical and physical influences, especially high temperature—characteristics which make asbestos useful for a large number of industrial purposes. In recent years, the use of asbestos has increased greatly; as much as a million tons may be used annually in the United States.

Of the different types of asbestos, by far the most commonly used is chrysotile, which is mined principally in Canada, the Soviet Union and South Africa. Crocidolite and amosite are mined in South Africa and anthrophyllite is mined only in Finland. There are probably subtle but important differences in both

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fibrogenic and carcinogenic potential among the various types of asbestos. Some of these differences may be accounted for by variations in the diameter and the length of the fibers, which effect penetration and deposition in the lung. However, in the usual physician's practice, and for the purposes of this chapter, the differences in biologic behavior of the various types of asbestos can be ignored.

Although man has used asbestos for thousands of years, extensive mining and use of the mineral has only occurred in the last 100 years (Figure 28–9). The disease asbestosis has only been recognized in the twentieth century; the realization of the real potential hazard of pulmonary fibrosis did not begin until the late 1920s and 1930s, and the potential risk for carcinogenesis was not established until the 1960s. Initially, asbestos-related disease was a recognized hazard only for workers engaged in mining and milling. The hazard for workers and other persons who use or are exposed to finished asbestos products has only recently been appreciated by industry. The main reason for this delayed understanding of the true risk of asbestos-induced disease is the fact that clinical manifestations do not usually occur until many years after exposure. Hence, the discovery of numbers of cases of asbestos-related disease could not occur for at least 20 or 30 years after a particular use became widespread.

The mining and especially the milling of asbestos is an obvious source of asbestos-related disease. The other principal sources occur in the insulation and construction industries. In the past, asbestos was widely used for insulation of various kinds of pipes, ducts, boilers, and furnaces. Many workmen in these occupations, whether or not they handled asbestos directly, had hazardous exposure. Insulation frequently needs to be cut or sawed to fit the parts being insulated, and often this is done in small enclosed areas with poor ventilation, leading to exposure to high levels of asbestos dust. Another source of respirable asbestos particles occurs during the repair or demolition of asbestos-insulated objects. The ripping out of old asbestos insulation produces especially high con-

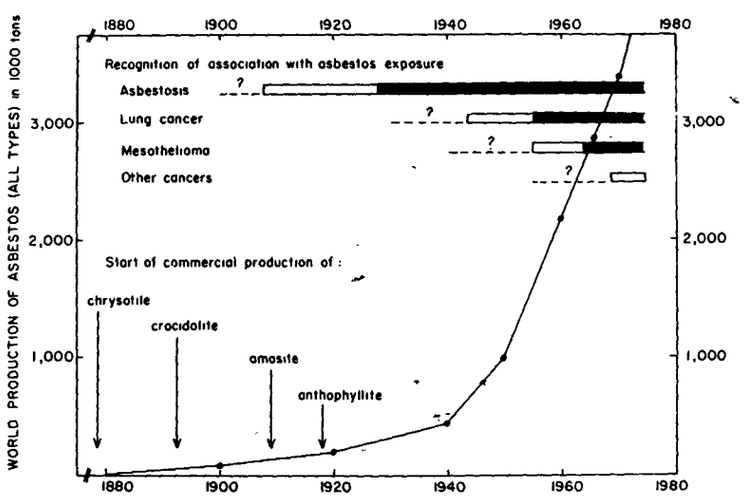


Figure 28–9. Production of Asbestos and Disease

Diagrammatic representation of the production of asbestos and the recognition of associated biologic effects. Symbols: ? = suspected; □ = probable; ■ = established. (From Becklake, M. R.: Am. Rev. Resp. Dis. *114*:187, 1976; by permission.)

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centrations of respirable dust. Nonoccupational exposure to asbestos can also occur. The pleural lesions resulting from asbestos exposure and possibly even pulmonary fibrosis may occur in contact cases, such as in (1) family members with direct exposure to asbestos workers who bring large amounts of dust home on their clothing or (2) persons living near a plant or a mill. Because asbestos is so widely used, most persons have had some contact with it, but the amount is too small to lead to any clinical disease, with the possible exception of malignancies. There has been considerable speculation that casual exposure (e.g., to the asbestos in brake linings) may produce malignancies in the general population; however, there is no scientific evidence to support such a contention. The malignancy that occurs with the least exposure to asbestos is mesothelioma and virtually all cases of this disease can be traced to obvious occupational, direct contact, or neighborhood exposures.

Asbestos appears to be indispensable for some industrial applications, but refined methods of dust disposal reduce or eliminate the risk to workers; furthermore, during the last few years, the use of asbestos for new insulation in the United States has been virtually abandoned. Thus the number of cases of asbestos-related disease should eventually decrease. However, there are still a great many workers who have had significant exposure in the past, and there is also a vast amount of old asbestos insulation in place, which can be a hazard when replacement or repair work is needed. Accordingly, the incidence of clinical asbestos-related diseases is not likely to be significantly reduced for decades.

Pathogenesis

The diameter of the fibers is the important determinant of whether or not a particular fiber penetrates to the distal airways and airspaces and is retained. Fibers with a diameter of 3 μm or less may be retained even if they are 20 μm or more long. Asbestos fibers are similar to silica particles in that both are likely to be ingested by macrophages, some of which are killed. However, the cytotoxic effect of asbestos is not as great as that of silica. Furthermore, there is a tendency for asbestos fibers to remain in place once they have reached the interstitial tissues. Thus asbestos particles are much less likely to enter the lymphatic system than those of free silica. The small number of asbestos fibers that get into the lymphatics seldom lead to enlarged hilar nodes, in contrast to silica particles. Other differences between silica and asbestos dust are that some asbestos fibers migrate outward to the pleura and their residence in the lungs is not as permanent as that of silica particles. Asbestos fibers tend to break up into smaller pieces that may be removed. In addition, asbestos, especially chrysotile with its high magnesium content, may be slowly dissolved within the tissue, reducing the dust burden in the lung.

A small percentage, estimated to be approximately 25 per cent, of the inhaled fibers become coated by macrophages, forming so-called "asbestos bodies." The coating over the fibers consists of ferritin granules and proteinaceous material. These bodies are yellow-to-brown in color and have a characteristic rod-shaped appearance with clubbed ends (Figure 28–10). Similar microscopic structures can occur in the lung in response to other kinds of fibers; because not all such bodies are due to asbestos, these structures are properly called ferruginous bodies. Although nonspecific, it is believed that the majority are

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Figure 28–10. Ferruginous Body

Photomicrograph of a lung biopsy specimen showing typical ferruginous bodies in center (arrows) and fragments of others near by. (Courtesy of Dr. J. H. Manwaring.)

asbestos-related. The presence of these bodies serves as a marker of asbestos exposure, but their presence does not necessarily indicate actual disease. They may be found in the sputum of persons recently exposed to asbestos. It is believed that the coating inactivates the fibers so that they exert little or no fibrogenic reaction. About 75 per cent of the fibers are not coated, and these may cause fibrosis. Many of these fibers are very small and thin and are not visible with the light microscope. The use of electron microscopy has demonstrated the presence of large numbers of small uncoated asbestos fibers in areas previously thought, on the basis of light microscopy, to have few or none.

A number of theories have been advanced to account for the presence of pleural and/or parenchymal fibrosis. None of these can adequately explain all of the observed findings, and experimental proof of them is lacking. The proposed mechanisms include mechanical irritation by "sharp" fibers, chemical fibrosis induced by dissolved asbestos, immunologic responses, and release of enzymes by death of macrophages. It is possible that several or all of these may be important, but it is also possible that some other, as yet undiscovered, mechanism may also be involved.

Asbestos-induced disease may involve the pleura (pleural plaques, effusions, or mesothelioma), the lung (asbestosis or bronchogenic carcinoma), or extrathora-

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cic organs (peritoneal mesothelioma and other visceral malignancies). The clinical, laboratory, and roentgenographic findings depend on the location and kind of reaction. For convenience, non-neoplastic involvement of the pleura and lung and neoplasms of the pleura, lung, and extrathoracic organs are discussed separately in the next section.

Non-neoplastic Pleural Involvement

Asbestos-related pleural disease most commonly presents as an asymptomatic pleural plaque. Occasionally, symptomatic but benign pleural effusion occurs. The most serious form of involvement, although rare, is mesothelioma, which is sometimes benign but more often malignant (see subsequent section). The relationship among the non-neoplastic disorders is not definitely known, nor is the relationship among serous effusions, plaque formation, and benign and malignant mesotheliomas understood.

PLEURAL PLAQUES

A pleural plaque is an area of collagen deposition and thickening that is characteristically irregular in size and shape and often calcified. Plaques are most likely to occur over the lower ribs and are especially common in the diaphragmatic pleura. The plaques involve only the parietal pleura and do not produce pleural adhesions.

Pathologically, plaques are mostly collagenous connective tissue, usually containing granular deposits of calcium along the collagen fibers. Coated asbestos fibers have not been observed in pleural plaques, but electron microscopy reveals

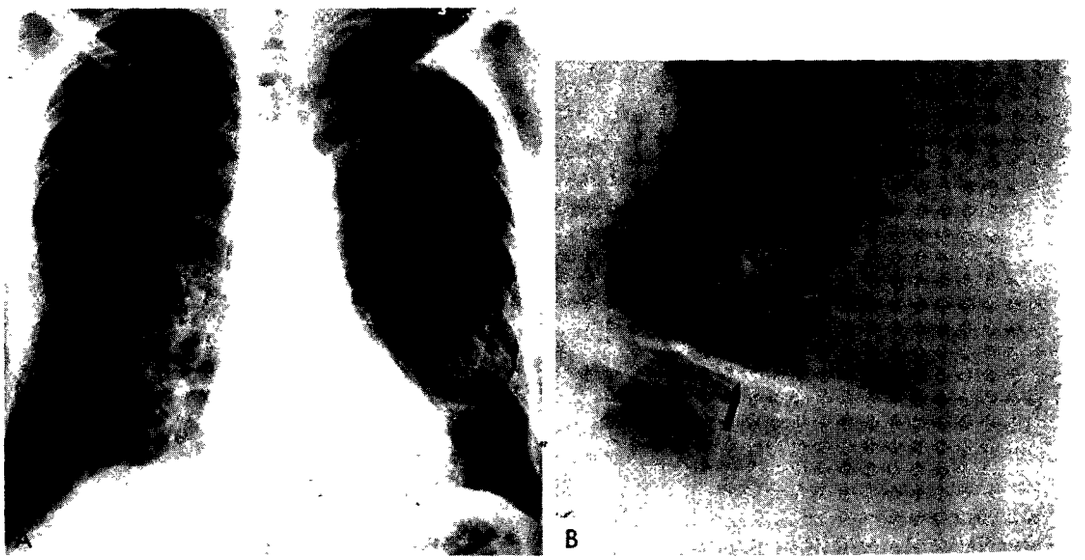


Figure 28–11. Calcified Pleural Plaques

Chest roentgenogram (*A*) of a 50-year-old shipyard worker, with 25 years' exposure to asbestos, showing calcified pleural plaques. Lateral projection (*B*) showing detail of one lesion (arrow).

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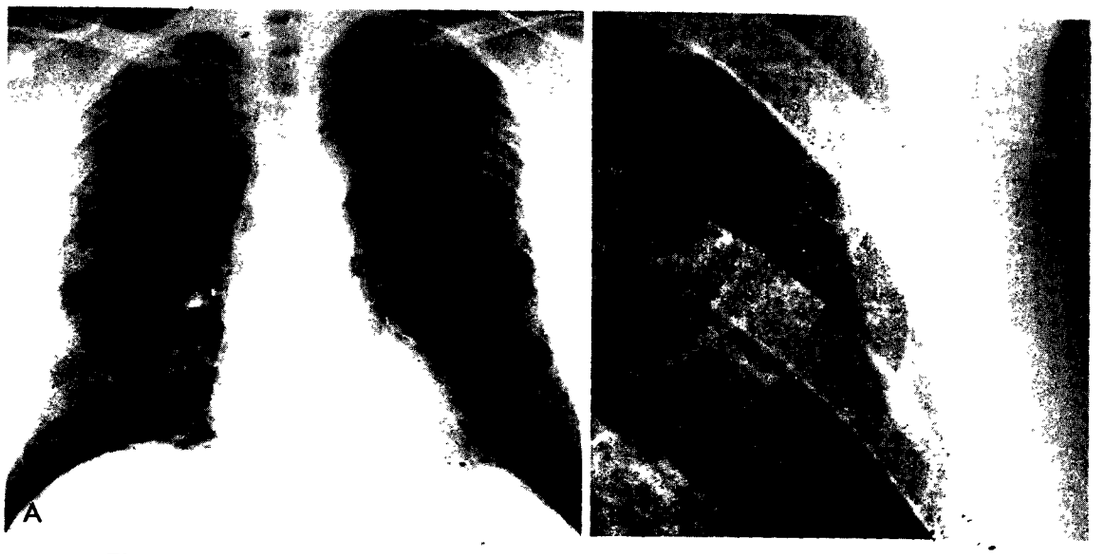


Figure 28–12. Pleural Plaques

Chest roentgenogram (*A*) of 55-year-old insulator, with 28 years' exposure to asbestos, showing bilateral pleural plaques. Close-up (*B*) showing detail of left-sided lesion (arrows).

many small uncoated fibers that are more concentrated in calcified regions. Ordinarily, pleural plaques do not produce symptoms and no significant functional impairment can be attributed to them. The chief importance of pleural plaques is that they serve as a marker of exposure to asbestos.

The roentgenographic appearance of calcified pleural plaques is usually quite characteristic (Figure 28–11). They may appear as extremely irregular areas of increased radiodensity. Most plaques are visualized best in the outer portions of the lung fields because they are seen partly on end and hence are more radiodense. Calcification is most commonly seen in the diaphragmatic pleura, particularly on the lateral projection (Figure 28–11*B*). It is probable that only calcified regions are seen on the roentgenogram because sites that do not contain calcium are too thin to produce detectable differences in radiodensity. If the patient is followed for a period of years, plaques may seem to enlarge and become more numerous; however, at least some of this change represents increasing calcification rather than actual growth of the lesions.

Bilateral ovoid or lenticular-shaped pleural plaques of uniform density are a common roentgenographic manifestation of exposure to asbestos (Fig. 28–12). Inflammatory disease and traumatic injury to the pleura with hematoma formation can produce similar appearing calcified lesions, but these are almost always unilateral and localized (see Figure 36–9). The presence of typical bilateral widespread pleural plaques is virtually pathognomonic of exposure to asbestos. Bilateral diaphragmatic calcifications add a further degree of certainty to the diagnosis.

PLEURAL EFFUSION

Exudative pleural effusion, which may occur in association with other asbestos-related lung diseases, particularly asbestosis and mesothelioma, may be the sole or at least the primary clinical manifestation of exposure. Chest pain of

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acute or chronic onset is the most common symptom. Recurrent pleurisy is frequent and may be unilateral, bilateral, or move from side to side. The fluid is usually an exudate, which is apt to contain red blood cells (5,000 to 50,000 per cu mm). The clinical course is variable, ranging from a benign transient disorder to a chronic process with disabling pleural thickening requiring decortication.

The diagnosis of asbestos-related pleural effusion may be difficult to establish because the patient's contact with asbestos may have been brief and many years earlier. Furthermore, asbestos fibers are usually not identifiable in the fluid, although they may be present. For this reason, patients with asbestos-induced pleural effusion are often said to have "idiopathic" pleural effusion (see Chapter 36).

Interstitial Fibrosis (Asbestosis)

Asbestosis or asbestos-induced diffuse interstitial fibrosis, which may vary in magnitude from minor involvement of the lung bases to extensive scarring with contraction and honeycomb changes, is a characteristic finding after prolonged and/or extensive exposure to asbestos dust. The progression of asbestosis from none to mild and from mild to moderate appears to be dose-related; subsequent worsening seems to relate in part to other as yet unidentified factors.

The microscopic appearance of the lung in the early stages of asbestosis resembles that of other forms of interstitial pneumonia, with varying degrees of fibrotic organization of the interalveolar septal walls and an infiltration of mixed leukocytes into the interstitium and of macrophages into the alveolar spaces. In addition, the pleura overlying the affected regions is nearly always fibrotic and thickened, sometimes to a considerable extent. According to some investigators, the basic lesion of asbestosis is peribronchiolar fibrosis, an observation that correlates with some of the earliest detectable changes in pulmonary function. Asbestosis cannot be diagnosed solely on the basis of the character and location of the fibrosis



Figure 28–13. Asbestosis

Chest roentgenogram of a 66-year-old retired construction worker, with 30 years of intermittent exposure to asbestos, showing diffuse interstitial infiltrations, more marked in the lower lung fields, and bilateral pleural thickening.

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because it is not significantly different from other forms of interstitial fibrosis. The diagnosis is supported when many asbestos bodies are present; however, sometimes these are few in number and occasionally ferruginous bodies may be seen in lungs of persons without asbestosis. Electron microscopy should reveal many small uncoated asbestos fibers and particles.

The chief symptom of patients with asbestosis is dyspnea, which in severe disease can greatly limit activity. Characteristic showers of dry rales during inspiration can be heard in patients with asbestosis as in patients with other kinds of interstitial fibrosis. These sounds do not represent fluid in the alveoli but are believed to be caused by the sudden opening of small airways in the dependent portions of the lung. Clubbing of the fingers may be present. In advanced cases,

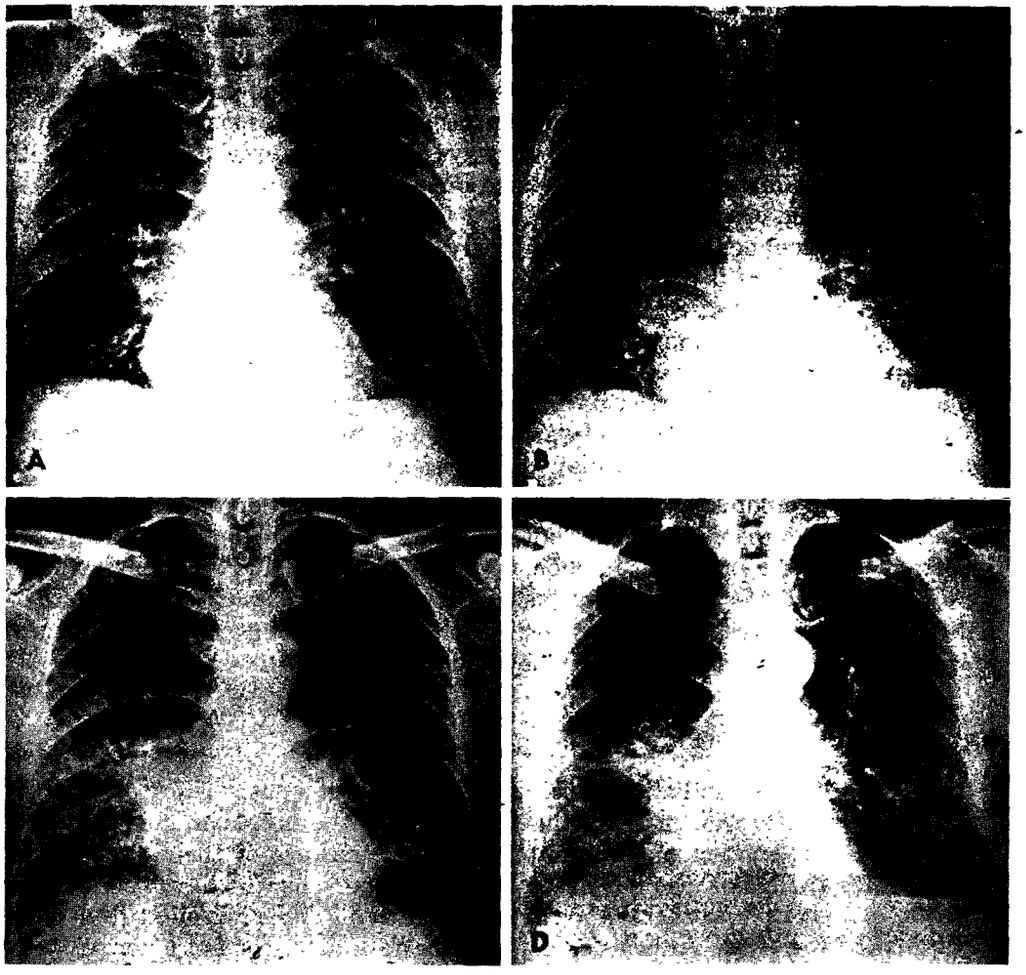


Figure 28–14. Progression of Asbestosis

Series of chest roentgenograms of a 38-year-old (at time of initial study) pipe fitter, who used asbestos for insulation. *A*, At age 38, showing slight prominence of basal bronchovascular markings. *B*, Seven years after *A*, showing progression to bilateral interstitial infiltrations. Exposure ceased at this time. *C*, Six years and *D*, 14 years after *B*, showing progressive fibrosis and development of bilateral pleural thickening.

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cyanosis, plethora (from polycythemia), and signs of cor pulmonale are sometimes present.

The roentgenographic appearance of the interstitial fibrosis due to asbestosis is the same as that of other types of interstitial fibrosis. Abnormalities may be minimal or even absent in the early stages because of the diffuse and fine nature of the fibrosis. The diagnosis of asbestosis can be substantiated from chest roentgenograms when pleural changes are also present (Figure 28–13). The type of pleural reaction involving both layers is typically said to produce fuzziness and irregularity in the cardiac and diaphragmatic borders. These changes are difficult to see and to evaluate, and their importance has been considerably overemphasized. Substantial asbestosis can be present without roentgenographically apparent associated pleural reaction, although it is present pathologically.

Characteristic abnormalities in pulmonary function in established cases of asbestosis are those of a restrictive ventilatory defect: decreased FVC and FEV_1, relative preservation of the ratios of FEV_1/FVC, and slight decrease in residual volume. Diffusing capacity and arterial Po_2 (especially during exercise) are usually decreased. Evidence of slight airways obstruction, resulting from an asbestos-induced peribronchial fibrosis or from nonspecific airway responses to the dust exposure, may also be present. Severe airways obstruction, except in patients with obviously severe disease, is not likely to develop unless other factors, such as cigarette smoking, are present.

Asbestosis commonly continues to progress even though exposure has ceased (Figure 28–14). There is no specific therapy for the basic disease and only the complications can be treated. Symptomatic support includes treatment of intercurrent infections, hypoxia, and congestive heart failure (see Chapter 40).

Neoplasms

The disease-producing potential of asbestos includes not only pleural and parenchymal inflammation and fibrotic reactions, but also neoplasms. The most important of these are mesotheliomas of the pleura and peritoneum and bronchogenic carcinoma. In addition, asbestos workers have an excess incidence of malignancies of several other organs: esophagus, stomach, colon-rectum, oropharynx, larynx, and possibly the kidney and breast (Table 28–2).

MESOTHELIOMAS

The majority of mesotheliomas occur in persons with a past history of asbestos exposure; it is likely that many of the patients with the neoplasm but without a known history of contact have forgotten or were unaware of their exposure to asbestos. Mesotheliomas also have been reported in persons who have been family contacts to asbestos workers and in persons living near a plant that has contaminated the air with asbestos fibers. Asbestos-induced mesotheliomas most commonly originate from the pleura but a considerable number involve the peritoneum. To date, there is no evidence of an increased incidence of mesotheliomas due to the random exposure that occurs as a result of living in an industrial society that uses a large amount of asbestos.

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TABLE 28–2. DEATHS AMONG 17,800 ASBESTOS INSULATION WORKERS IN THE UNITED STATES AND CANADA JANUARY 1, 1967–JANUARY 1, 1977: ANALYSIS BY DURATION FROM ONSET OF EMPLOYMENT. (From Selikoff, I. J., and Hammond, E. C., CA, *28*:87, 1978; by permission.)

TOTAL MEN	12,683	12,051
MAN-YEARS OF OBSERVATION	89,466	77,389

	BEFORE 20 YEARS FROM ONSET		20 OR MORE YEARS FROM ONSET	
	*Expected**	*Observed*	*Expected**	*Observed*
Total deaths, all causes	283.93	324	1,377.01	1,946
Cancer, all sites	42.65	83	277.25	911
Lung cancer	12.03	36	93.94	449
Pleural mesothelioma	**	2	**	64
Peritoneal mesothelioma	**	3	**	106
Cancer of esophagus	0.66	1	6.35	17
Cancer of stomach	1.56	1	12.67	21
Cancer of colon-rectum	4.07	4	33.79	55
Asbestosis	**	8	—	154

*Expected deaths are based upon white male age specific mortality data of the U.S. National Center for Health Statistics for 1967–1975 and extrapolation to 1976.

**These are rare causes of death in the general population.

There is typically a long interval between the onset of asbestos exposure and the appearance of a mesothelioma. This latent period is almost always at least 20 to 30 years and may be longer. There is poor correlation between the intensity and duration of asbestos exposure and the development of mesothelioma, suggesting that the neoplasm is not related to the total dose of asbestos fibers. Many patients with mesotheliomas had only brief and/or minimal exposure to asbestos decades previously. This is quite different from the findings in cases of bronchogenic carcinoma and asbestosis, in which a much clearer dose-response relationship exists. Mesothelioma also is not related to cigarette smoking, unlike cancer of the lung in asbestos workers.

Mesotheliomas are much more likely to be malignant than benign. Pleural involvement is usually so widespread that the lung is encased in a thick neoplastic shell. Exudative and usually blood-tinged pleural effusion is common, and may precede the development of overt tumor. Peritoneal mesotheliomas usually present with exudative ascites; constriction of abdominal viscera is not a common feature.

Malignant pleural mesothelioma may be strongly suspected on the basis of clinical and radiologic findings: the presence of pleural effusion and diffuse pleural thickening, often with chest pain, in a patient with a past history of exposure to asbestos. A pleural biopsy is needed to confirm the diagnosis; however, the histologic appearance may be difficult to interpret in some cases because mesothelioma cannot always be differentiated confidently from metastatic malignancy involving the pleura. Once the diagnosis of malignant mesothelioma is

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established, most patients die within a year (mean of 7 months). It cannot be cured surgically, and the response to radiation and chemotherapy is extremely poor.

BRONCHOGENIC CARCINOMA

A number of studies have shown that there is a considerable excess of cases of bronchogenic carcinoma in workers exposed to asbestos. Analysis of these data has shown that this increased incidence is almost entirely in persons who are also cigarette smokers. The incidence of lung cancer in nonsmokers with asbestosis is low; in contrast, among smokers with asbestos exposure the incidence is much higher than among other groups with the same smoking habits but without asbestos exposure. There appears to be a synergistic carcinogenic effect between asbestos and cigarette smoking. The actual incidence differs greatly among various reported studies. Some of this variation may be due to differences in both the type of fiber and the size of the fibers that are inhaled. There is a dose-response relationship both for the degree of asbestos exposure and for the amount of cigarette smoking. Heavy smokers who have had heavy asbestos exposure may have as much as 90 times the incidence of lung cancer as nonsmokers without asbestos exposure. Those with less asbestos exposure and those with less heavy cigarette smoking histories have a lower incidence, but one that is always much higher than in comparable groups of cigarette smokers without asbestos exposure.

All types of lung cancers occur but there may be a slightly increased incidence of adenocarcinoma (scar cancer) and a decreased proportion of small cell undifferentiated lesions. Although most patients with asbestos-induced bronchogenic carcinoma also have asbestosis, there have been cases with very little or no pulmonary fibrosis.

The treatment of asbestos-related lung cancer is qualitatively the same as for other comparable lung cancers and the results of a particular treatment are probably similar. However, resection may be impossible because of pulmonary insufficiency from asbestosis. All patients with current or past exposure to asbestos should be warned of the marked danger of cigarette smoking. Those who are still smoking need to be fully informed of the tremendous risk of dying from lung cancer if they continue to smoke. Current or past heavy smokers with significant asbestos exposure probably should have regular routine chest roentgenographic and cytologic examinations to look for early stages of lung cancer (see Chapter 19 for further details).

OTHER MALIGNANCIES

Careful epidemiologic studies of asbestos workers have revealed a greater than expected risk for many malignancies. This is most clearly expressed in the excess rate of bronchogenic carcinoma and mesothelioma. However, other organs may also be involved, although the incidence is not as excessive as for tumors of the lung or pleura. Exposure to asbestos is associated with cancer of the gastrointestinal tract (esophagus, stomach, and colon-rectum), larynx, and possibly the kidney and breast (Table 28–2).

The mechanism of malignant transformation is not known. Awareness of the increased risk of these cancers improves the chance of early diagnosis, prompt treat-

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ment, and survival. Periodic surveillance for pulmonary, gastrointestinal, and laryngeal cancers is warranted in all persons with significant exposure to asbestos.

RESPIRATORY DISEASE OF COAL WORKERS

Large scale underground coal mining began in the early nineteenth century, and soon after that, reports of occupational lung disease began to appear. A number of descriptive names were used, such as miners' asthma and miners' phthisis, which actually included a number of diseases besides coal workers' pneumoconiosis, such as silicosis and tuberculosis. When chest roentgenograms and other improved diagnostic tests became available in the twentieth century, the various disease processes could be differentiated.

Two quite different diseases can be attributed specifically to the inhalation of coal dust. One is coal workers' pneumoconiosis, or CWP, and the other is chronic bronchitis. These two may occur in the same person, but some workers have only one of these. Patients with CWP are further classified as having simple CWP, complicated CWP (also known as massive pulmonary fibrosis), and Caplan's syndrome. As in patients with other pneumoconioses, complicating infections such as tuberculosis and other mycobacterioses may also occur in coal miners and lead to further radiologic and functional changes. Nonspecific terms such as the currently popular "black lung disease" are meaningless and should be avoided.

Although the prevalence of smoking in coal miners is less than in other occupations, some miners smoke heavily and the deleterious effects of cigarette smoking often complicate their occupationally induced disease. In many patients, the effects of cigarettes are more important than those of inhaled coal dust.

Epidemiology

Coal is widely used as a fuel. Although many workers and other persons are involved in transporting, handling, and using coal once it has been removed from the mine, the amount of dust that such nonminers are exposed to is relatively small. Respiratory disease of coal workers is essentially confined to those who actually work in underground coal mines.

A number of different jobs are involved in extracting coal from a coal seam and getting it to the surface from an underground mine. An important part of the occupational history is to ascertain which jobs a particular patient had because these affect the likelihood of acquiring certain pneumoconioses. Coal is removed from what is known as the coal face by cutting or blasting. The fragments are then loaded onto a train or shuttle system to be removed from the mine. The workers at the coal face have by far the greatest exposure to dust. Those who work on the transportation system do not have as much exposure to coal dust, but the sand used to improve traction on the rails can lead to substantial exposure to silica. Maintenance and other underground workers have intermediate exposure to coal dust, and those who work on the surface have even less. One worker may hold different positions at different times; usually, as a miner grows older, he is moved to

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jobs further away from the coal face. A heavy dust load is usually needed to produce detectable disease, hence CWP and chronic bronchitis generally occur only in those miners who were underground for 20 or 30 years and is more common in those who worked for many years at the coal face.

Coal Workers' Pneumoconiosis

The pathogenesis of all three varieties of CWP—simple CWP, complicated CWP, and Caplan's syndrome—depends on the inhalation and retention of large quantities of coal dust. The additional factors, constitutional or acquired, that transform simple CWP into the other, more serious forms are not well understood; some of the prevailing theories will be described in the following sections.

SIMPLE COAL WORKERS' PNEUMOCONIOSIS

The first defense against respirable coal dust that is deposited on the alveolar surface is phagocytosis by alveolar macrophages. The phagocytes containing ingested coal particles move to the terminal bronchiole and are removed via the mucociliary escalator. When exposure to coal dust is heavy, macrophages increase in number, but the clearance mechanisms may be overwhelmed and the coal-laden cells are retained in the terminal respiratory units, especially in the respiratory bronchioles. When retention is prolonged, fibroblasts invade the region and deposit a thin layer of reticulin around the macrophages. Lysis of the macrophages and release of proteolytic enzymes, which is especially likely to happen if the coal dust also contains silica, enhances the formation of connective tissue. By this process, respiratory bronchioles and the branches from them become slowly incorporated in the coal macule, an aggregate of coal dust, dead and dying macrophages, fibroblasts, and connective tissue. These basic lesions of all forms of CWP occur throughout the lungs but are more common in the upper lobes.

Simple CWP is said to exist in the presence of roentgenographically detectable opacities 1 cm in diameter or less, according to the ILO U/C classification (see earlier section on Roentgenographic Classification and Figures 28–1 to 28–3). The abnormalities are further subdivided into categories 1, 2, and 3 depending on the profusion of small opacities. Regardless of category, simple CWP is a benign disease if complications do not develop. Respiratory symptoms of cough, expectoration, and dyspnea are frequently present, but these also occur with equal frequency in coal miners with normal chest roentgenograms. Because the presence or degree of symptoms correlate poorly with the extent of roentgenographic involvement, severe symptoms, if present, are probably caused by factors besides simple CWP. Focal emphysema in the central portion of the lobule may develop, but widespread emphysema of clinical importance does not occur as a result of simple CWP.

A slight decrease in FVC and FEV_1/FVC may be found by studying large groups of miners with simple CWP. However, the changes are small and not of clinical significance for the individual patient. There is also a small increase in residual volume, and there may be minor abnormalities in distribution of inspired air and gas exchange. A few coal workers have a slight decrease in diffusing capacity and lung compliance; a more common abnormality, but still unimportant clinically, is the presence of frequency dependence of compliance. The magnitude of the ordinary changes in pulmonary function tests in simple CWP is so small that they cannot

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account for significant symptoms and disability; if these are present, other abnormalities must coexist. If other diseases are present, such as chronic bronchitis, then symptoms may occur through a combination of adverse effects.

Chest roentgenograms reveal small round opacities throughout both lung fields, especially in the upper regions. In the early stages of the disease (category 0), they may not cast detectable shadows because of their small size and/or insufficient number. Comparison of serial chest roentgenograms taken at least five years apart may reveal the appearance of new lesions or progression of old ones.

COMPLICATED COAL WORKERS' PNEUMOCONIOSIS

Complicated CWP is diagnosed when a large opacity of 1 cm or more in diameter is observed in the chest roentgenogram of exposed persons. The term progressive massive fibrosis is also used to describe this condition. The occurrence rate of complicated CWP is approximately 2 per cent in miners with category 2 or 3 simple CWP, but it occasionally occurs in persons with category 1 disease or even in those in category 0. In most but not all patients, the process, once underway, tends to be progressive, but additional dust exposure does not seem to affect further progression. In some miners, complicated CWP may not occur until after exposure has ceased; in others, roentgenographic lesions may remain stationary for long periods.

Pathologically, complicated CWP is characterized by large masses of black-colored fibrous tissue. These are most likely to occur in the posterior segment of the upper lobes and the superior segment of the lower lobes. Large lesions may cavitate, either as a result of ischemic necrosis or secondary infection, often tuberculous.

A number of theories have been proposed to explain why only a small percentage of miners at risk develop complicated CWP: (1) that progressive massive fibrosis is due to silica deposited in addition to the coal dust; (2) that the fibrosis is due to acute pulmonary infection or to chronic infection with *M. tuberculosis* or other pathogenic mycobacterial organism; and (3) that altered immunologic



Figure 28–15. Complicated Coal Workers' Pneumoconiosis

Chest roentgenogram of a 70-year-old man, who had mined coal for 38 years, showing conglomerate densities in medial portions of both lungs; the two on the right have cavitated and there is an air-fluid level in the lower one.

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mechanisms are involved. None of these theories is presently generally accepted and serious objections can be raised to each of them. Presently available data seem to favor the immunologic theory, but other as yet undiscovered influences may well play a role. For the present, the cause should be considered largely unknown.

In patients with category A disease (i.e., the roentgenographically large opacity or opacities have a combined diameter between 1 and 5 cm), there are usually no clinically significant symptoms or functional abnormalities. In contrast, patients with category B and especially category C disease (i.e., the combined diameter of opacities exceeds 5 cm but occupies less than or more than one third of the lung fields, respectively) may be severely dyspneic. Complicating pulmonary hypertension and cor pulmonale may occur also but generally only in the late stages of the disease.

Pulmonary function testing reveals decreased FVC and FEV_1/FVC; the airways obstruction probably is a direct result of the distortion and narrowing of large airways by the fibrotic lesions. The residual volume is often increased and the diffusing capacity is reduced. Other findings that can be expected are abnormalities of the distribution of inspired air, decreased pulmonary compliance, and decreased arterial Po_2.

Complicated CWP is chiefly a roentgenographic diagnosis because it depends on the identification of one or more large densities over 1 cm in diameter (Figure 28–15). As indicated, category A, B, or C involvement defines the size and extent of the large lesions, not the small lesions of associated simple CWP. In category C disease, there may be extensive shrinking of the involved lobe with compensatory overdistention and paracicatricial emphysema of the remaining lung tissue. Usually category 2 or 3 simple CWP is also present, but in advanced progressive massive fibrosis the emphysema and overinflation may make the small opacities difficult to detect or seem to be less profuse.

CAPLAN'S SYNDROME

In 1953, Caplan described rounded nodules seen on the chest roentgenograms of coal miners who also had rheumatoid arthritis. This is now commly known as Caplan's syndrome or sometimes as rheumatoid pneumoconiosis. Like complicated CWP, the diagnosis of Caplan's syndrome depends on roentgenographic findings in patients with clinical or serologic evidence of rheumatoid disease. Scattered nodules 1 to 5 cm in diameter are seen, usually in the peripheral portions of the lung, that may develop rapidly over a period of several weeks and may cavitate. Although roentgenographic densities closely correlate with the finding of rheumatoid factor in the patient's serum, their appearance does not necessarily correlate with manifestations of arthritis; both may occur simultaneously, or either may precede the other by many years.

The pathogenesis of Caplan's syndrome is believed to depend on the changes that underlie the development of rheumatoid lung disease (Chapter 33) interacting with those provoked by the pneumoconiosis. The same type of reaction has also been described in patients with silicosis and asbestosis. Several but not all studies have shown an increased incidence of positive rheumatoid factor in coal miners.

Rheumatoid pneumoconioses produce interesting roentgenographic findings and further study of patients with these abnormalities is of considerable importance in defining the immunologic aspects of the reaction to inhaled dusts. It is known that there are immunologic reactions involved in all pneumoconioses, but much work still needs to be done before these reactions can be characterized completely.

Pneumoconioses and related diseases (1980)
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In coal workers, Caplan's syndrome is more likely to occur in those with category 0 or 1 pneumoconiosis than in those with more advanced disease. The pathology of the lesions is characteristic. There are concentric layers of collagen with necrotic centers. Vasculitis is also present and the periphery contains an area of active inflammatory reaction.

Coal Workers' Chronic Bronchitis

Coal miners are more easily studied than most groups of workers because they are relatively numerous, they tend to stay in the same occupation for many years, and they are concentrated and usually remain in the same geographic areas. Careful surveys have shown that there is a definitely increased incidence of chronic bronchitis with the usual symptoms of chronic cough and expectoration of sputum in coal miners. Furthermore, there is a tendency for the bronchitic symptoms to increase with increasing amounts of exposure to dust. No necessary correlation exists between the bronchitis and the pneumoconiosis of coal workers. Some patients have both, but often one occurs without the other. Chronic bronchitis is, of course, also strongly associated with cigarette smoking, but the increased incidence in coal miners is still evident after correcting for smoking habits, and the disorder also occurs in nonsmoking workers.

Small decreases in ventilatory tests such as FEV_1 have been attributed to exposure to coal dust; however, large groups of workers must be studied to obtain statistically significant differences and the effect in individual persons is inconsequential. Similarly, apart from the presence of cough and sputum, it has not been possible to associate significant clinical disability with the chronic bronchitis of coal workers.

OTHER PNEUMOCONIOSES

Silicosis, asbestosis, and coal workers' pneumoconiosis are the chief occupationally related lung diseases in terms of prevalence, disability, and socioeconomic cost. There are, however, several other pneumoconioses that are important not only from a clinical point of view but also in terms of epidemiologic and preventive considerations. This section deals briefly with the pneumoconioses from nonasbestos silicates, tungsten carbide, graphite, aluminum (Shaver's disease), and diatomaceous earth, and the pulmonary effects of inhalation of iron, tin, and barium dust.

Pneumoconiosis from Nonasbestos Silicates

Several nonasbestos silicates are capable of causing pneumoconiosis. The major abnormality from these substances is diffuse pulmonary fibrosis similar to asbestosis. Pleural plaques may occur, especially with talc, but the carcinogenic potential is, at present, uncertain.

TALC

Talc is milled soapstone, which chemically closely resembles asbestos. The milled product of talc is a mixture of particulate and fibrous material. The latter is

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much like asbestos and some talc also contains silica. Because of the complex nature of the commercial product, there is some uncertainty regarding what component of talc is responsible for the pulmonary fibrosis. There is good reason to believe that the asbestos-like fibers are the most important fibrogenic portion of talc.

Talc is used in the manufacture of a number of materials such as ceramics, paint, asphalt, and roofing materials and was used as a dusting powder on human skin. Workers who mine and mill the material are at highest risk, but the disease has been reported in persons using refined products.

The clinical, radiologic, and pathologic features of talc pneumoconiosis closely resemble those of asbestosis but there is more of a tendency to develop nodular lesions in talc pneumoconiosis. Calcified pleural plaques like those that occur after exposure to asbestos are also seen.

KAOLIN

Kaolin, also known as China clay, is a hydrated aluminum silicate which is used in the ceramic, paper, and cement industries. Heavy exposure usually in the milling and processing of kaolin, can cause severe pulmonary fibrosis, which is similar to that seen after exposure to talc.

FULLER'S EARTH

Fuller's earth is a silicate clay with absorbent properties that is now used chiefly in refining oils and in foundry molding. There have been several cases of pulmonary fibrosis (Fuller's earth pneumoconiosis) due to this material, but the disease is generally a relatively benign one.

CEMENT

Cement is a complex man-made substance containing calcium silicate. There is no evidence that exposure to cement produces pneumoconiosis, but workers with heavy exposure to cement dust are reported to have a higher prevalence of airways obstruction and respiratory symptoms than unexposed persons.

FIBERGLASS

Fiberglass is a silicate made from molten glass. Although glass is virtually inert chemically, there has been concern that it may produce fibrosis by mechanical means. Epidemiologic studies of fiberglass workers have failed to show an increased incidence of respiratory symptoms or chest roentgenographic or ventilatory abnormalities. In addition, autopsy studies have not shown pathologic findings attributable to fiberglass. Thus although there is some disagreement, present evidence strongly indicates that fiberglass does not produce pneumoconiosis.

OTHER SILICATES

A few cases of pneumoconiosis have been attributed to other silicates such as mica, nephelene, and sillimanite, but the number of workers with sufficient exposure is small. Mica, like asbestos and talc, may produce pleural plaques.

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Tungsten Carbide Lung Disease

An especially hard metal used for cutting and drilling is prepared by blending finely powdered tungsten and carbon with cobalt as a binder. Two types of reactions may occur from exposure to tungsten carbide: (1) diffuse interstitial chronic pulmonary fibrosis, which is similar to other pneumoconioses and (2) acute airways obstruction, which resembles byssinosis in its clinical manifestations and time course. Both of these reactions are believed to be hypersensitivity-type reactions, and it appears that cobalt, not tungsten or carbon, is the sensitizing agent.

Graphite Pneumoconiosis

Pure graphite is crystallized carbon. The naturally occurring material often contains a small amount of silica as a contaminant. There is evidence that some exposed persons may develop fibrosis from either the silica or a combination of the graphite and the silica. Other workers develop a disease that is essentially the same as coal workers' pneumoconiosis.

Shaver's Disease

Electrically heated furnaces used in the manufacture of synthetic abrasives (corundum) emit fumes of complex composition as they fuse the ore known as bauxite. The fumes contain extremely fine particles of alumina and silica (0.02 to 0.5 μm) that may be inhaled by the workmen who feed the furnaces. The resulting pneumoconiosis was first described by Shaver in 1947. Similar lung disease has been reported to occur in workers exposed to powdered aluminum.

Exposure to bauxite fumes may produce a rapidly progressive pulmonary fibrosis with severe emphysema and large subpleural blebs that tend to rupture. Dyspnea, which may develop rapidly, even within a few months of the first exposure, is the main symptom. In most cases, however, symptoms are minimal, even after many years of exposure. The first manifestation may be sudden severe dyspnea resulting from a spontaneous pneumothorax, which may be bilateral. In advanced cases, the usual symptoms of emphysema are present, such as dyspnea, anorexia, weakness, and weight loss. Secondary polycythemia has been noted in cases of prolonged duration.

In the early stages of the disease, the chest roentgenographic changes are slight and easily overlooked. The fibrosis is fine and delicate in texture and is usually not nodular. Large emphysematous blebs are sometimes present. Spontaneous pneumothorax, which may be localized because of pleural adhesions, may be seen in roentgenograms of patients who have no symptoms. Mediastinal widening is often present. The diaphragm is depressed and irregular, as in patients with other types of emphysema. The hilar structures are prominent and with the increased vascular markings may simulate chronic passive congestion. Pleural reaction may be marked in those patients who have experienced repeated spontaneous pneumothorax.

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Diatomaceous Earth Pneumoconiosis

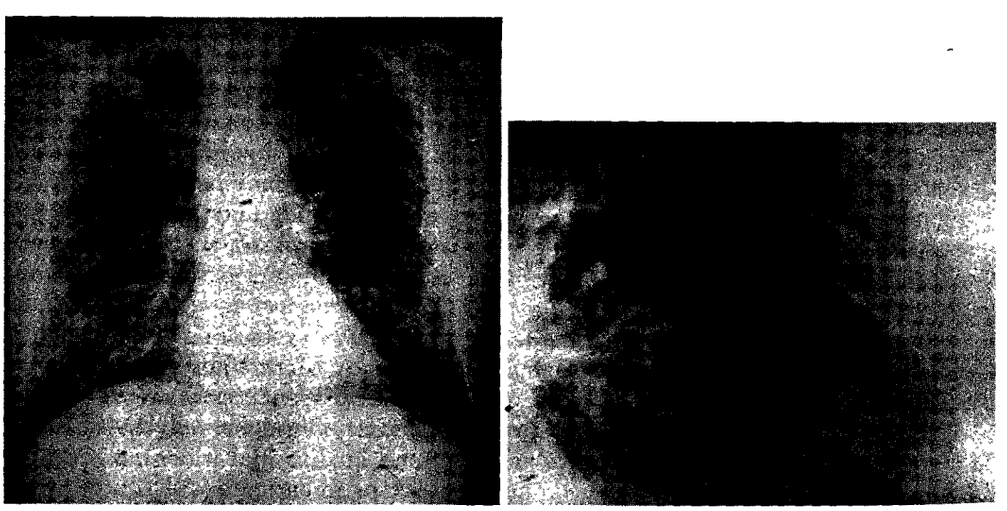
Diatomaceous earth consists of the remnants of skeletons of unicellular organisms deposited in large quantities on the bottom of the sea for eons. In its pure form, it consists of silicon dioxide, but clay is often present in the commercial material. Diatomaceous earth is used for a number of purposes, primarily filtration. Considerable amounts of dust may be created when the material is in a finely divided form and when it is transported by blowers; accordingly, it is necessary to provide elaborate protective equipment to ensure the safety of workmen.

Diatomaceous earth is capable of producing diffuse pulmonary fibrosis. The fibrosis is not necessarily of the nodular character seen in patients with typical silicosis. Linear stranding, which subsequently becomes coarse and massive, may develop within a year or two if the exposure is heavy. Dyspnea may be severe and some patients have developed bullous emphysema with spontaneous pneumothorax similar to that seen in patients with Shaver's disease.

The roentgenographic findings are variable and have not been well classified in relation to the type of material inhaled and to the duration or intensity of exposure. Often, there is merely accentuation of the vascular marking, but in severe cases massive linear fibrosis with or without nodulation is observed. Conglomerate densities may also occur, probably indicating complicating infection such as tuberculosis.

Siderosis

Siderosis is a benign condition resulting from accumulation of iron particles in the lung. It is important to recognize siderosis because of the frequency with which



A B

Figure 28–16. Siderosis

Chest roentgenogram (*A*) and detailed view (*B*) of an asymptomatic foundry worker and arc welder suspected of having simple silicosis. Lung biopsy established diagnosis of siderosis.

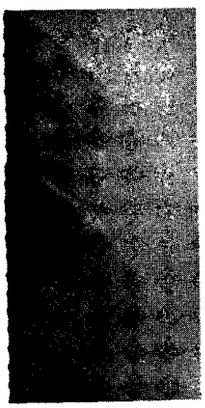
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it has been confused with silicosis; although the two diseases produce similar chest roentgenographic abnormalities, the deposition of pure iron, in contrast to free silica, does not cause fibrosis.

Siderosis is encountered most frequently among workers who weld ferrous metals. The fumes emitted from welding contain particles of iron oxide of respirable size and also pulmonary irritants of several types. In electric arc welding, electrodes are fused by an electric current to form a bond between the objects being welded. The electrodes are coated with various materials designed to form an inert vapor mantle around the operation to minimize oxidation of the molten metal. These coatings often contain pulmonary irritants such as fluorides and cadmium. Symptoms of cough and expectoration effected by these irritants may lead to roentgenographic examination of the chest that reveals nodular accumulations of iron pigment in the interstitial tissues that resemble the changes of silicosis (Figure 28–16). However, the opacities of siderosis are rather dense and more sharply outlined than those of silicosis. Foundry workers and hematite miners may be exposed to both iron and silica, which further complicates the evaluation of the roentgenogram and the diagnosis. A substance called "rouge," used as a polishing agent in the jewelry industry, contains finely powdered iron and may also cause siderosis. Siderosis produces no symptoms and no disability; it does not predispose to tuberculosis or to malignancy.

Stannosis and Baritosis

Stannosis results from the deposit of tin oxide in the lungs of tin miners. Stannosis produces no symptoms unless silicosis is also present. Baritosis, or the deposition of barium salts in the lungs, has been reported in workers in industries manufacturing glass, insecticides, dyes, rubber, and ink. Baritosis, like siderosis and stannosis, produces no symptoms. However, because the retained metal in the lung in these three conditions is quite radiodense rather spectacular roentgenographic abnormalities may be present despite the absence of a fibrotic reaction.



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