11 Lung diseases in coal workers

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INTRODUCTION

Coal is not a pure mineral. It is formed by the accumulation of vegetable matter covered by sedimentary rock (thereby sealing it from air) and subjected to pressure and temperature over the ages. This causes the physical and chemical properties of the matter to change. The matter dries, becomes warmer, and loses oxygen content, all the while increasing the relative carbon content (Stahl, 1989).

The first step in this conversion of vegetable matter to coal is the formation of peat, a moist spongy material. This transformation of organic deposit can occur in a stagnant water bed relatively quickly (at a rate of approximately 1 foot (30 cm) per 100 years). An approximately 100-ft (30-m) accumulation of peat compresses to form a 1 ft-wide coal seam. In the most simple terms, coal is comprised of moisture (which lessens with time), pure coal (carbon) and mineral matter (Thomas, 1992).

The process of conversion (coalification) from organic matter follows a transformation of wood → peat → lignite → bituminous coal → anthracite coal. The terms brown coal and black coal refer to coals of low and high rank, respectively. *Rank* describes the extent of change from vegetation to mineral-free coal. Rank is directly related to the percent of carbon in coal, the completeness of the transformation from vegetation to coal, and the geologic age of the deposit, with new deposits forming above the old. Indeed, Hilt (1873) observed that 'in a vertical sequence, at any one locality in a coal field, the rank of the coal seam rises with increasing depth'. The *type* of coal relates to the plant materials which form the coal. The *grade* refers to the purity of the coal – or the amount of inorganic material (including ash and sulfur) released in the burning of coal. Sulfur is a frequent contaminant of coal and is derived from sulfur in the plants which have formed the coal or as a result of infusion of water containing sulfur during the formation of coal. In today's world, sulfur released as a by-product of coal burning is an important air pollutant and boiler corrosive, making coal with a high sulfur content less desirable.

Other important descriptive terms include a quantitative measurement of coal *moisture* (the percentage of water that is contained in coal and released with moderate heat) and the amount of *volatile matter* (that percentage of substances, mainly gases and coal tar, lost when a sample is well heated). The residue of combusted material remaining after a coal sample is completely burned is coal *ash*. The ash content has two sources. The first is the inherent minerals in the decaying wood and the second is material which was blown or entered the swamp while the peat was being formed (Haught, 1955). Coal is classified primarily by rank (Table 11.1).

The composition of coal mine dust varies with the coal seam. Most of the dust is composed of carbon, although in some seams it may only approximate 60% of the dust (Coal data, 1995), with more than 50 different elements and their oxides (Coates, 1981). Dusts of high rank typically have more silica than dusts of lesser rank because the anthracite seams often have roofs and floors of quartz which contaminate the coal during mining (Mutmansky, 1984)

During this century coal has become less important as an energy source, with petroleum and natural gas

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Table 11.1 Coal characteristics

Coal rank	*Volatile matter (%)*	*Carbon (%)*	*Ash (%)*	*Moisture (%)*	*BTU*/pound*
Peat (dried)	70	25	5	10	8000–12 000
Lignite	41	36	9	14	5500–7000
Bituminous	32	55	10	3	12 000–14 000
Anthracite	5	87	5	3	> 13 000

* One BTU (British thermal unit) is equivalent to the amount of heat necessary to raise the temperature of 1 lb water from approximately 4° to 5° F (1 BTU/lb°F= 4.1868 kJ/kg°C).

Source: adapted from Haught (1955).

used increasingly as a substitute. In 1900, 94% of the world's energy requirements were met by coal. By 1940, coal accounted for 75% of the world's energy consumption. In the 1950s and 1960s, cheap oil became available and this further curtailed coal use. By 1967, coal utilization had fallen to 40%, and by 1978, coal was only responsible for 31% of fuel use (Stahl, 1989).

In the US, electric power generation accounts for the utilization of more than 80% of the coal produced each year. Coal can also be processed and made into liquid and gaseous fuels. These products have the potential to compete with natural gas and petroleum-derived fuels depending on the cost. Nowadays, natural gas and petroleum-derived fuels are cheaper, and the use of liquid and gaseous coal fuels is not cost effective. Coke, necessary for the production of steel, is also an important use of coal, as is coal tar, a hydrocarbon by-product released by the formation of coke. For about 75 years, until the end of the Second World War, a great percentage of the organic chemical industry used coal tar as the substrate to form additional agents. More recently, the great majority of organic compounds have been derived from petroleum-based products, although some specialty chemicals are still made from coal tar (National Research Council, 1995).

A BRIEF HISTORY OF THE STUDY OF LUNG DISEASE IN COAL MINERS

As one might expect, the earliest coal mines were outcroppings of coal along the banks of rivers. As such, they could be described as surface mines. The drive for coal production on a large scale was associated with the manufacture of iron and derived from the development of the steam engine in the early part of the 18th century. It was not until 1762 that a steam engine was used to work a colliery in Scotland (Meiklejohn, 1951). Yet, even underground mining grew quickly as proper methods for ventilating fresh air and toxic gases from the mines, as well as effectively pumping water from the mines, were put into place. As of 1866, in Scotland, there were 472 coal mines, 41 000 miners and 12 million tons of coal produced yearly.

Meiklejohn (1951, 1952a,b) has provided a wonderful summary of the history of the health of United Kingdom coal miners. Lung disease in miners has been referred to as miners' asthma, phthisis, anthracosis, and in Scotland, miners' black lung. In 1831, the first report of 'black lungs' attributed to employment in coal mines was published (Gregory, 1831). He described a 59-year-old man who had been employed as a miner for 10 or 12 years and was hospitalized for generalized anasarca. He soon died from progressive heart failure. The lungs were examined, and a picture consistent with progressive massive fibrosis (PMF) with cavitation of these large lesions on a background of simple coal workers' pneumoconiosis (CWP) was described.

> *'At necropsy, the lungs were universally adherent to the chest wall; the pleura was thickened and in places ossified. When cut into, both lungs presented one uniform black carbonaceous color, pervading every part of their substance . . . The left lung did not appear to contain any cavities, but was condensed and loaded with black serum. Some minute hard points could be felt in various parts of both lungs'.*

Opinions regarding the explanation for this black pigment included gunpowder, inhalation of lampblack, or soot from the oil lamps of the miners. Marshall (1833–1834), just several years later, concluded that: 'The true explanation of the origin of this disease in colliers seems to be, that it is in consequence of the inhalation of fine coal dust, and its deposition in the substance of the lung. That coal may float through the air in particles sufficiently fine to be inhaled without immediate irritation and that it is thus inhaled is a matter of common observation'.

Yet, for the rest of the 19th century, there was considerable controversy and disagreement regarding the impact on coal dust inhalation on the survival of miners. Overall, data showed the mortality of coal miners only minimally exceeded the mortality of the most healthy men, such as farmers or agricultural laborers. This lack of coal dust effect on survival was thought to be attributable to better mine ventilation and lesser exposures to coal dust. Yet, even then, the relatively less fibrogenicity of coal dust compared with silica was well recognized (Ogle, 1885; Meiklejohn, 1952a).

Early in the 20th century, the understanding of lung disease in miners again changed. Collis (1915) opined that coal dust did not produce pneumoconiosis (and even served to protect against tuberculosis) but was a marker which showed the path by which dust enters and travels in the lung. He attributed dust disease in miners to silica inhalation (associated with working of thinner seams with likely silica contamination) and reported the disease that developed to be silicosis. Yet, this view failed to take into account increasing mechanization in the mines and deepening of the mines, with the resulting increase in coal dust generation. The adverse effects of these factors more than compensated for the positive effect of increased mine ventilation (Meiklejohn, 1952b) (Figure 11.1).

It was not until washed coal, free of silica, was recognized to produce dust disease in the lungs in stevedores, those employed loading and leveling coal in the holds of ships, that CWP was widely accepted as being pathologically distinct from silicosis (Collis and Gilchrist, 1928; Gough, 1940; Hepplestone, 1947). Later, King *et al.* (1956) showed that the radiologic and histologic severity of pneumoconiosis in coal miners was related to the total amount of dust and not the silica content of the coal. Although it was recognized that mining was associated with lung disease, only with the development of specialized techniques such as chest radiography, the discovery of the tubercle bacillus, and the application of the histologic examination of lung tissue that the specific causation of respiratory diseases affecting miners was identified.

With the development of medical tools necessary to make an accurate diagnosis of pneumoconiosis, the emphasis from the 1930s to the present time has been on epidemiologic studies of the prevalence of pneumoconiosis and attempts to understand the basic mechanism of both CWP and silicosis.

HOW COAL IS MINED TODAY

Safe mining requires close attention to numerous aspects of coal production. Although we are primarily interested in the respiratory consequences of coal mine dust inhalation, mining is inherently dangerous with recognized risks of underground fires and explosions, toxic gas excess and rock falls. Therefore, safety measures must emphasize adequate ventilation so that methane is removed and the risk of explosion and fire

Figure 11.1 The approach to mining in the earlier part of this century where coal was removed with a pick and shovel and hand loaded into a shuttle car.

minimized; protection from roof falls; and the control of dust. Each of the different methods of mining presents their own challenge in controlling the workplace environment.

Coal may be found in outcroppings and in seams that are sometimes just a few feet below the surface. In these deposits coal can be readily obtained by simply scraping away the surface or overburden and extracting the coal with earth-moving equipment. This is called surface or strip mining. Dust levels in the air at surface mines are generally considerably lower than in underground mines with few notable exceptions. In

Figure 11.2 (a) Typical rock drilling for overburden removal at an above ground mine in West Virginia. Highly respirable dust is generated, often with a high silica content. The operator is required to stand near the drill to control the drilling operation. (b) A damaged rubber skirting which surrounds the drill at the level of the ground. This is used for local dust control. In this instance, the skirt is damaged and dust generated by drilling is not captured. Such exposures may be hazardous to the drill operators and the driller helpers. (c) The operator standing in an intense dust cloud while operating the drill. (d) A driller helper, equipped with a hard hat and hearing protection, but without respiratory protection, standing near an on-going drilling process. (Figure 11.2 (a–d) are courtesy of Joseph C. Cocalis, P.E., CIH.)

surface miners and coal plant cleaning workers in the anthracite mining area of the US, the mean FVC, FEV_1 and peak flow rates was not related to the number of years worked in coal-cleaning plants (Amandus *et al.*, 1989). Yet, there is a group of these workers at special risk for pneumoconiosis. Workers who operate the large drills (drillers and driller helpers) to make holes in which explosives are placed at surface coal mines so that the overburden might be removed are exposed to silica and are at risk for the development of silicosis rather than CWP (Banks *et al.*, 1983) (Figure 11.2 (a–d)).

To a certain extent, workers in some exclusively above-ground operations may be exposed to coal dust. These are the workers at the tipples where crushing, sizing, washing and blending of coal is done; at surface coal mine sites away for the drilling operations; and at locations where coal is loaded into ships, railroad cars or river barges. Although each case needs to be evaluated on an individual basis, the great majority of these exposures are relatively small and insufficient to cause disease.

On hillsides where the coal seam may be many feet below the surface of the hill but some outcropping occurs from the side of the hill, a feasible method of mining coal is to bore into the outcropping with an auger. Depending upon how far into the hillside the seam extends, this method of mining is a combination of surface and underground mining (Figure 11.3).

Where coal seams are buried deep, typically greater than 200 feet underground, it is not economically feasible to strip away the overburden. The only practical way of mining the coal is to sink shafts from the surface to the coal seam and then follow the seam with a series of more or less horizontal tunnels. This has been the most prevalent method of mining coal in this century (Figure 11.4 (a–d)).

Figure 11.3 An auger fracturing overburden at a hillside mine.

Modern underground coal mining methods are categorized as room and pillar mines or long-wall mines. A room and pillar mine is a series of parallel rooms where mining has occurred separated by pillars of unmined coal. As mining goes on this looks increasingly like a honeycomb. Two types of mining are performed in room and pillar mining. The first is *conventional mining*, and is the least mechanized, accounting for less than 10% of US coal mine production (Coleman, 1992). Here, incisions are cut in the sides and bases of the seam and charges inserted through holes drilled in these areas. The roof is first supported, then the charges are exploded, and the coal loaded and removed from the face. The cycle is typically performed sequentially in adjacent areas to maximize coal production (Taylor and Thakur, 1993).

Alternatively, *continuous mining* is a mechanized process which accounts for approximately 60% of US coal production. This approach uses a machine with a rotating head to break coal from the face. The operator advances this machine through the seam, moving the cutting head of the machine up and down to maximize coal breakaway from the face. A conveyor, incorporated into the continuous mining machine, moves the broken coal from the face to waiting underground shuttle cars. The continuous miner may include roof bolter units located just behind the rotating cutter head or have a roof bolting machine nearby. Workers remain here while mining is conducted.

In this method of mining, between one-and two-thirds of the coal remains within the pillars after the rooms are mined. To recover the coal within the pillars, each pillar is mined using timber for temporary roof support. The timber is then removed, thereby allowing the roof in that area to collapse. This approach is called retreat mining because after mining of the pillars begins, no attempt is made to go back into the block. The roof caves in and the mine is abandoned (Stout, 1980).

Longwall mining is the most mechanized and the most productive means of underground mining. The number of underground mines employing longwall mining techniques continues to increase in the US (and now is used in approximately 100 mines). This accounts for approximately 30% of US coal production. Longwall mining produces up to 40 tons of coal per minute, and in some instances, nearly 4000 tons per work shift. In this process, the continuous miner cuts out a longwall of coal which may be 600–800 feet (180–250 m) wide and 5000–12 000 feet (1500–3600 m) long. When the wall is formed, the hydraulically powered roof supports, the cutting

(a)

(b)

(c)

(d)

Figure 11.4 (a) A coal cutter. This is an integral part of room and pillar mining. The 'saw' may be 15–20 feet (4–6 m) in length. The operator sits behind the saw in the motorized open cab. (b) A cutting 'ball' operating at the edge of a coal seam. Frequently, a conveyor is placed at the seam to capture and remove the cut coal. (c) Multiple cutting heads on a coal cutting machine. The operator sits behind the machine in close proximity to the freshly fractured coal. (d) Roof supports with jacks and preparation for additional roof bolting. This underground job is associated with silica exposure and a potential for the development of silicosis. The risk for silicosis in an underground coal miner is primarily related to the extent of roof bolting, and underground 'drilling' in general.

machine or shearer (often with built-in water sprays to minimize dust generation), and the conveyors are put into place. The shearer moves across the coal face in a bi-directional manner with a depth of cut of about 2 ft (0.6 m). A conveyor belt, which runs the length of the longwall, captures the newly excavated coal. As each pass is made by the shearer (which requires one or two workers to operate and travels up to 60 ft/min), the roof supports and the conveyor are advanced. As these supports are advanced, the roof behind these supports caves in, another example of retreat mining (Euler, 1981).

Finally, it should be noted that longwall mining has several disadvantages. First, if the cutting machine or any machine necessary for the process fails, production of coal from the entire system stops. Second, a coal face of 600–800 feet (180–250 m) may have numerous different characteristics. Attempting to mine this extensive block may lead to mining difficulties. Third, developing and setting up a longwall requires considerable time and worker effort. Finally, the large number of roof supports for a longwall are very costly. Using fewer supports would cut costs. This has led operators to combine the roof support system of the longwall mine with the flexibility of the continuous mining machine. This has been named *shortwall mining*. With this approach, the face is shortened to about one-third of the length of the longwall, production is increased relative to room and pillar mining, and the miner works under the hydraulic roof support the entire time, thereby lessening the hazards of roof falls. Coal production has increased for several decades in the US, while the number of working miners has decreased (NIOSH, 1995) (Figure 11.5 (a–c)).

CLINICAL AND EPIDEMIOLOGIC FEATURES OF COAL-INDUCED LUNG DISEASE

Coal inhalation can result in industrial bronchitis and CWP, presenting as either simple pneumoconiosis or complicated CWP (PMF). In addition, there has been considerable discussion regarding the relationship between coal dust exposure and clinically significant airways obstruction in the recent literature. This relationship between inorganic dust exposure, including inhalation of coal dust and airways obstruction is discussed in detail in Chapter 5.

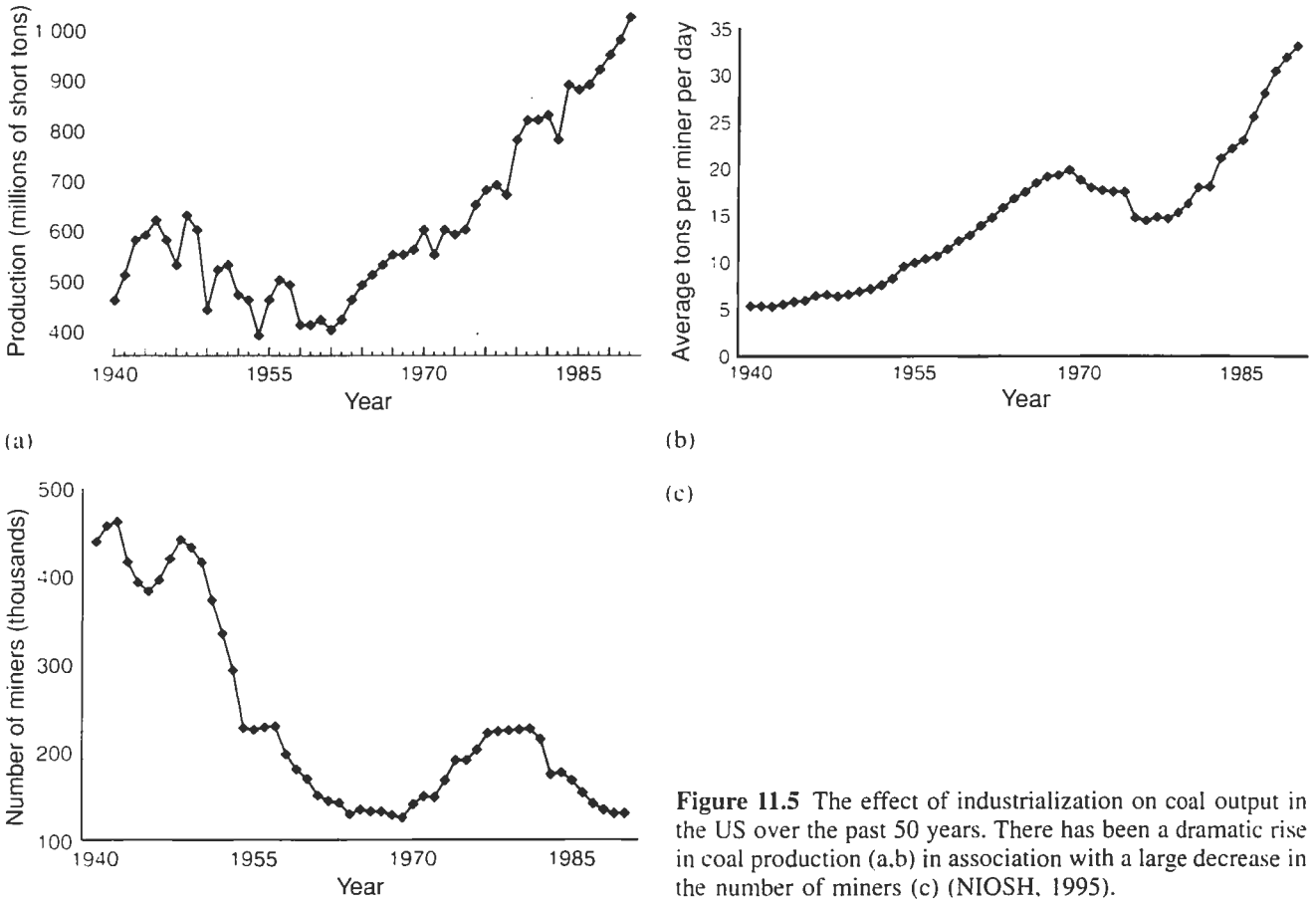

Figure 11.5 The effect of industrialization on coal output in the US over the past 50 years. There has been a dramatic rise in coal production (a,b) in association with a large decrease in the number of miners (c) (NIOSH, 1995).

Industrial bronchitis is a common diagnosis among workers exposed to dusts, including coal dust (Morgan, 1978). It manifests as a productive cough which persists for at least 3 months per year for at least 2 years (chronic bronchitis) associated with workplace dust exposure. The relationship between coal mining and bronchial mucous gland dimensions was presented in an autopsy study by Douglas *et al.* (1982). Coal dust exposure resulted in an increase in the maximal gland : wall ratio independent of smoking, but no relationship between mucous gland size and lung dust or pneumoconiosis was found. This suggested that pneumoconiosis is related to respirable dust exposure, and mucous gland enlargement is related to the inhalation of larger (non-respirable) dust particles which present a chronic burden to the mucociliary escalator and act as irritants to the airway.

Early reports showed that the prevalence of bronchitis in coal miners varied by smoking history, age and job (Kibelstis *et al.*, 1973). In all reports, smoking miners had bronchitis more frequently than non-smokers, and when studied, showed bronchitis to increase with age (a surrogate for years of dust exposure in the workplace). Marine *et al.* (1988) identified 543 lifetime non-smokers among 14 888 British coal miners (3.6%) studied between 1953 and 1967 who were less than 65 years of age, worked at the coal face in most instances, did not have PMF, and who had participated in three surveys over 10 years. Of these, 17% had bronchitis at the third survey. In another report of coal miners, 16% had bronchitis (Seixas *et al.*, 1992). There is little information on the natural history of chronic bronchitis in non-smoking miners once they are removed from dust, but it is thought to resolve when dust exposure ends. In those who develop chronic bronchitis due to dust, there is often a measurable, but not clinically significant, decline in FEV_1.

CWP results from the inhalation and deposition of coal mine dust, and the lung's reaction to its presence. Three criteria are necessary for this diagnosis (Balaan *et al.*, 1993). They include:

1. A chest radiograph consistent with the features of CWP.
2. A work history (typically that of underground coal mining) which is sufficient in exposure and latency to result in pneumoconiosis.
3. The absence of other illnesses which may mimic CWP.

Therefore, the diagnosis of CWP is a radiologic diagnosis which can be made with confidence without histologic confirmation, by an adequate history of coal dust exposure and a characteristic chest radiograph. Clinical features such as dyspnea, cough and sputum production are important in addressing the degree of a miner's overall respiratory impairment, but are not a part of the diagnostic criteria. The radiographic appearance of CWP is similar to that of silicosis, and miners may develop disease from exposure to both dusts during their employment as a coal miner.

Although numerous radiographic classifications have been used, the first attempt at an international consensus regarding classification of the radiographs of pneumoconiosis was put forth by Cochrane and colleagues in 1951. Since then, a series of approaches have been taken, and although this is recognized to be an imperfect system (Wagner *et al.*, 1993), the International Labour Office 1980 classification is the most widely accepted (ILO, 1980). This is discussed in detail in Chapter 9.

Typically, CWP is described as simple pneumoconiosis or PMF. The radiograph in simple pneumoconiosis shows small opacities, ranging in size from pinhead to 1 cm in diameter. Rounded nodules predominate and tend to appear first in the upper zones and then the middle and lower zones as the number of opacities increase. With prolonged excessive exposure, these small opacities may coalesce and form larger opacities, recognized as PMF lesions and characterized by one or more large opacities greater than 1 cm in diameter. As the disease progresses, the upper zone nodules begin to coalesce and distort the lung architecture, a feature which gradually becomes more prominent. This typically presents as deviation of the trachea and major airways to the side of the most prominent area of coalescence, loss of upper zone lung volume, elevation of the hila, and basilar emphysema (typically of a panacinar type), attributable to traction placed on the lower lung zones by the elevation of the hila.

There are no symptoms or physical signs associated with simple CWP. The not infrequent presence of a chronic cough and sputum production, even in the presence of CWP, is attributable to 'industrial bronchitis'. Alternatively, these same clinical features in a smoking miner may be partially attributable to bronchitis caused by the inflammatory stimulus of cigarette smoke. Finger clubbing is not a feature of CWP and, if noted, should prompt further investigations.

When PMF is recognized on the chest radiograph, the worker frequently describes dyspnea, cough and sputum production, although it is well recognized that the degree of impairment or the presence or absence of symptoms does not always correlate well with the extent of chest radiographic abnormalities.

The effect of pneumoconiosis on the heart has been described by several investigators. Lapp *et al.* (1971) performed cardiac catheterization during rest and exercise in 43 miners with CWP (23 with airways

obstruction and 24 without). In seven of 12 miners, either chronic obstructive lung disease or PMF appeared to explain the increase in pulmonary artery pressures. The other five with increased pressures had the smaller type of pinpoint (p) opacity on their radiograph. It is likely pertinent that the pinpoint type of opacity is associated with diminished diffusion and a diminution of the vascular bed (Lyons *et al.*, 1967). A later autopsy study of 215 British coal miners (100 with simple CWP or no pneumoconiosis and 115 with PMF) was designed to address the relationship between coal dust exposure and right ventricular hypertrophy (RVH). In this population, the prevalence of RVH was the same in those with simple CWP as in those without pneumoconiosis (approximately 15%) and was related to the extent of airways obstruction. Overall, RV enlargement did not occur unless the coal miner was a smoker with severe airflow obstruction or had developed PMF (Fernie *et al.*, 1983).

CWP alone does not appear significantly to increase the risk for development of coexisting mycobacterial infection. Miners, however, often have exposure to silica in drilling, a job typically associated with silica, rather than coal dust exposure. Miners with silica exposure in the workplace may also be at risk for mycobacterial infection. The appearance of a cavity in a PMF lesion or an aggressive and unexplained rate of radiographic progression should prompt examination of the sputum for mycobacteria.

The chest radiograph in simple CWP correlates with the amount of dust in the lung at autopsy (Rivers *et al.*, 1962). This is not true of the complicated form of the disease, suggesting that inadequately defined host factors play a role in the development of this lesion. Hypotheses which might explain the differences in tissue response to coal dust in those who remain with simple CWP and those who progress to PMF include:

- inhalation of significant amounts of silica in addition to coal in mine dust;
- infection with *M. tuberculosis* or an atypical mycobacterial organism; and
- development of specific immunologic factors which are responsible for progression.

Yet, none of these explanations appears to explain satisfactorily the development of PMF. First, PMF has been reported in carbon electrode workers. This suggests that silica exposure is not necessary for PMF to occur (Collis and Gilchrist, 1928). Treatment of miners with simple CWP in Wales with anti-tuberculous drugs did not prevent the occurrence of PMF (Watson *et al.*, 1959). Autoantibodies reactive to lung tissue antigens have been demonstrated in the sera of miners, suggesting a role in the tissue response. There are factors driving the development of PMF beyond the amount of dust in the lung.

A special case of nodular lung reaction occurs in dust-exposed individuals who either have rheumatoid arthritis or who develop rheumatoid arthritis within the subsequent 5 to 10 years (Caplan, 1953; Caplan *et al.*, 1962). This leads to discussion regarding the potential role of immunologic factors in mineral dust pneumoconioses. The nodules of Caplan's syndrome vary in diameter from 0.5 to 5 cm, and are usually multiple, bilateral and situated peripherally. Grossly, the lesions resemble a giant silicotic nodule. Microscopically, the amount of dust in the lesion is small, there is a necrotic area in the center, and there is a surrounding cellular zone infiltrated with lymphocytes and plasma cells. In many nodules there is a peripheral zone of active inflammation with neutrophils and a few macrophages. This observation was further enhanced when similar radiographic appearances in miners without arthritis, but in whom circulating rheumatoid factor was demonstrated, was described. An increased incidence of circulating positive rheumatoid factor among miners with PMF has also been reported. In a group of 60 consecutive cases of scleroderma, Rodnan *et al.* (1967) described 26 with a history of employment as a coal miner or employment in an occupation where silica exposure was encountered. Although the course of scleroderma did not vary in the two groups, it appears that chronic fibrogenic dust exposure may be a risk factor for the development of scleroderma.

The clinician may be presented with the diagnostic dilemma of attempting to distinguish a primary or metastatic neoplasm from an unusual presentation of PMF. When large opacities of PMF occur bilaterally on a background of simple CWP, one can be reasonably confident that the lesions are less likely to represent neoplastic disease. When there is a sparse background of simple CWP or none, or there are crops of nodules (as in Caplan's syndrome), the differentiation from neoplasm may indeed be difficult.

EPIDEMIOLOGY

A series of studies in the 1960s showed that the prevalence of CWP varied by region of the country, the type (rank) of coal mined (Lainhart *et al.*, 1969), and the duration and intensity of exposure (McBride *et al.*, 1963, 1966). Among underground miners, those working at the face and exposed to higher concentrations of coal mine dust had a higher prevalence of CWP than surface workers or those whose jobs caused them to enter the face area intermittently (Jacobsen, 1971). For example, miners in Pennsylvania anthracite seams had a considerably greater prevalence of CWP than those

miners in the western plateau of Colorado and Utah who mined a lower rank of coal. The institution and enforcement of dust control measures has reduced the attack rate of CWP and may have lessened progression among those with radiographic opacities who continued to work in mines under the US dust standard which was begun in 1969 at 3 mg/m^3 and reduced to 2 mg/m^3 in 1972 (Coal Mine Health & Safety Act, 1969).

A series of prevalence studies of miners with CWP have been conducted by the US Public Health Service. The first, round one of the US coal workers' surveillance program, was performed on data collected from 1970 to 1973. The most recent, round five of this same program, was undertaken from 1987 to 1991. Data from these studies have been presented in summary form in an impressive series of reports (Attfield and Castellan, 1992; Attfield and Althouse, 1992; Attfield, 1992). The mines were chosen to represent different geographic areas, coal seams and coal mining methods. Briefly, although the data are complicated by chest X-ray reader variation and concerns regarding worker self-selection for examination, there appears to be a reduction in the prevalence of CWP in the miners who have participated in this study over time. When comparing studies from the mid-1970s with the mid-1980s, the number of cases with both simple CWP and PMF declined in all groups with at least 10 years of mining. In one group, miners with a mining tenure of between 15 and 24 years, the prevalence decreased to less than half. Similarly, in those with a lifetime of coal mining, 11% had category 2 simple CWP in round one compared with 2% in round 4.

The most important concern about these data, however, is whether the recent work is representative of the miners' respiratory health and whether this decline in pneumoconiosis reflects a true decrease or self-selection inducing bias. Most importantly, in the years 1970–1973, approximately three-fourths of the nation's 100 000 miners participated in the program. Participation decreased to less than 30% of the mining work force in the study performed from 1987–1991.

With this diminished level of participation, death certificate data may be the most accurate measure of the number of miners with CWP. Over the next decade, a decline in the number of death certificates showing CWP may confirm the conclusions suggested by epidemiologic studies (NIOSH, 1994). In this report, the number of US death certificates mentioning CWP appears to have initially increased from the numbers reported in 1968, to have changed little over 20 years, and to have perhaps begun a minimal downward trend. This leads one to be concerned whether the current coal dust standard is sufficient to block the development of CWP in miners with coal dust exposure remains a concern (Figure 11.6).

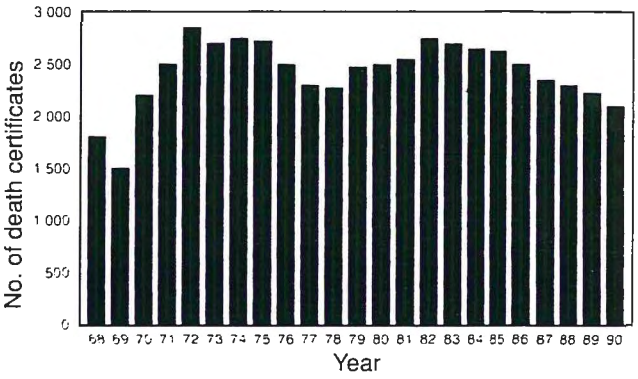

Figure 11.6 Numbers of death certificates in the US with mention of coal workers' pneumoconiosis by year. (Adapted from the NIOSH Work Related Lung Disease Surveillance Report, 1994.)

Data from British studies have also clearly shown that the attack rate (incidence of new cases) and the probability of progressing to a higher category of simple CWP were related to the mass of respirable dust to which the miner was exposed during his lifetime (McLintock *et al.*, 1971). Yet, once the miner with simple CWP leaves dust exposure, the chest radiograph is not likely to progress. The same cannot be said for the PMF. The rate of progression to PMF appears to be influenced chiefly by the age at which the miner begins to show radiographic changes of CWP. Progression may also be influenced by the presence of a rheumatoid diathesis (Davies and Mann, 1949).

Does the chronic inhalation of coal dust increase the risk for a premature death? A number of studies of mortality in coal miners CWP have been primarily conducted in Europe and the US. Perhaps the best to summarize the rates of mortality in the first part of this century was Enterline (1972), who noted that it was not until 1950 that relatively accurate death certificate data which described an individual's usual occupation were available. In US Public Health Service data collected from death certificates for the year 1950 (Enterline, 1964), there was a gross excess in mortality for all ages for coal miners compared with the general population. For example, in miners aged 45–64 years, the Standard Mortality Ratio (SMR, the ratio between the actual number of deaths and the observed number of deaths) approximated 2.0.

To emphasize further the mortality risk of being a coal miner between the years 1949 and 1963, the Society of Actuaries (1967) published SMRs on 44 US occupations using life insurance policy data. Of all of these occupations, coal mining had the highest SMR. The SMR for coal miners was 1.5. The SMR for death attributable to respiratory disease exceeded 40 using death certificate data and 11 using life insurance policy

information. Overall, most SMRs for both the death certificate data and the life insurance data for all of the causes of death were increased in miners, with the only clear consistency being the discrepancy between the death certificate study showing an SMR of 1.7 for deaths due to cancer compared with only 0.8 in the insurance policy study. Both the SMR from death certificates which measured mortality attributable to accidents at work and the SMR from insurance company data due to accidents and homicides exceeded 6.0. Yet, even when accidents and respiratory disease were eliminated as causes of death, the SMR for coal miners ranged between 1.4 and 1.7 times the death rate for all working men. Even as late as 1979, a coal miner was recognized to have a two-fold risk of being killed at work compared with the risk of the average worker (Editorial, 1979).

The mortality experience of more than 6000 miners and ex-miners residing in the Rhondda Fach (Cochrane *et al.*, 1978; Atuhaire *et al.*, 1985), a mining community in South Wales, also showed an excess mortality. In the absence of pneumoconiosis, or when simple pneumoconiosis, or even PMF of category A was present, the SMR approximated 1.2. The number of deaths did not vary by pneumoconiosis category unless category B or C large opacities were present. When this was the case, the SMR was almost double that of the comparison group. Chronic bronchitis was identified twice as often as a cause of death among the miners. Additionally, carcinoma of the stomach increased with pneumoconiosis category, a feature frequently reported in other studies. Ortmeyer *et al.* (1973) reviewed mortality for compensated Pennsylvania miners by category of opacity on the chest radiograph and recorded similar results.

Stocks (1962) was one of the first to present the relationship between mining and gastric cancer, but his report made it clear that it was necessary to compare the mining risks for illness with local controls. He showed that the rate of gastric cancer among miners in the United Kingdom varied, with it being most frequent in South Wales, intermediate in northern England, and least in the Midlands. The relationship between this malignancy and coal dust exposure was also suggested in a study of gastric carcinoma in Utah, where gastric malignancy occurred nearly three times as often in residents of two Utah coal mining counties, leading the authors to speculate that swallowed coal particles (containing polyaromatic hydrocarbons, including benzpyrene) increases the carcinogenic risk (Matolo *et al.*, 1972).

Further understanding of the mortality experience of US coal miners on the rolls of the United Mine Workers Health and Retirement Fund from 1959 to 1971 was presented by Rockette (1977). Of the 23 232 men enrolled, 7741 died. The overall SMR for this population was 1.02, a dramatic improvement compared with the above-cited death certificate study (Enterline, 1964). Yet, the SMRs for several specific categories of illness remained elevated. Specifically, the SMR for non-malignant respiratory disease was 1.6 and that for accidents was 1.8. Although mortality due to all cancers was not increased, the SMRs for stomach and lung cancer were excessive (1.4 and 1.1, respectively).

In a mortality report of Appalachian coal miners and ex-miners followed from 1963 to 1971, the mortality of current miners was 7% less than expected, although ex-miner mortality was 24% greater than expected (Ortmeyer *et al.*, 1974). Simple pneumoconiosis did not affect life expectancy, while the presence of PMF did.

Meijers *et al.* (1991) postulated that coal workers have a greater risk of lung cancer due to their exposure to coal mine dust, a material containing various potentially carcinogenic organic (i.e. hydrocarbons) and inorganic (i.e. trace elements such as cadmium and chromium) compounds. In support of this hypothesis, crystalline silica – a component of coal dust – had been declared a probable carcinogen (IARC, 1987). The cause of death of 334 miners from a cohort of 5400 miners with CWP employed in 11 Dutch coal mines from 1956 to 1960 was evaluated in 1983. Data showed a higher than expected mortality, primarily attributed to stomach and large and small intestine malignancy, as well as more frequent non-malignant respiratory diseases.

Exposure data were available in 26 363 coal miners from 20 collieries in England and Wales who first attended medical screening between 1953 and 1958 (Miller and Jacobsen, 1985). A chest radiograph, respiratory questionnaire and coal mining history was recorded and estimates of exposure made. An assessment of the causes of 8489 deaths was begun in 1980. First, the general mortality was 13% less on average than in English and Welsh men in the same region. The survival of miners with category A large opacities was less than that of men without pneumoconiosis, yet the mortality rate did not increase with increasing category of simple pneumoconiosis. Mortality due to all causes, as well as that attributable to pneumoconiosis, bronchitis and emphysema, increased with cumulative dust exposure. Furthermore, in those with greater dust exposures at the start of the evaluation, particularly those of an older age group, a shorter survival was reported. There was no association between lung cancer and coal mining, but the risk for cancer of the digestive system approached significance. Similarly, there was no relationship between gastric mortality and survival in Australian coal miners (Armstrong *et al.*, 1979).

The mortality experience of US coal miners with exposure estimates was reported by Kuempel *et al.* (1995). In this population, the mortality experience of

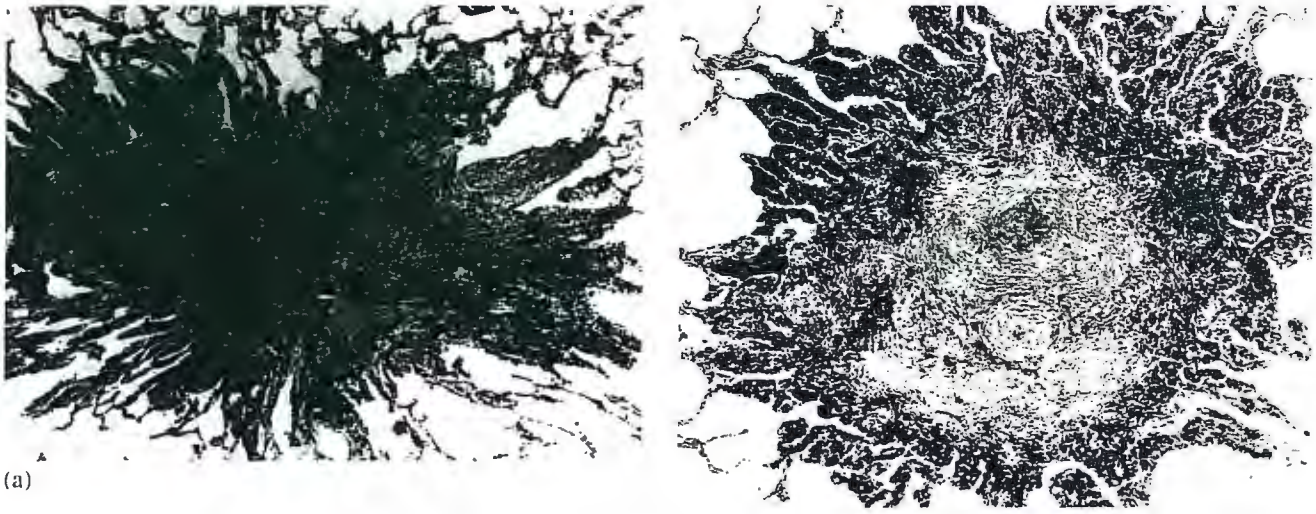

Figure 11.7 (a) Photomicrograph showing a coal macule. There is coal dust, reticulin fibers, and coal dust-laden macrophages, with minimal fibrosis. The periphery of the nodule shows air space enlargement. (b) Photomicrograph of a lung biopsy from a roof bolter with histologic evidence of both a coal macule and a silicotic nodule. This reflects the coal dust and silica dust exposures which have occurred over his working lifetime. On the periphery, there is coal dust and coal-laden macrophages. Centrally, there are whorls of fibrosis characteristic of a silicotic nodule. The cellularity is most prominent in the periphery of the lesion. (Figure courtesy of V. Vallyathan, PhD.)

793 men among a population of 8878 miners enrolled between 1969 and 1971 and followed until 1979 was evaluated. Overall, the SMR for all causes was 0.85, the SMRs for bronchitis, emphysema, lung cancer and stomach cancer were not increased, but the SMR for pneumoconiosis was 3.7. No statistical relationship between deaths due to lung cancer or stomach cancer was reported.

In summary, a series of mortality reports have not convincingly shown that simple CWP is associated with premature mortality, but that PMF adversely affects survival, most apparent when category B and C large opacities are present. Although there appears to be no clear difference in the SMR for lung cancer between miners and non-miners (Vallyathan *et al.*, 1985), a number of studies show an increase in the SMR for digestive cancer in general, and stomach cancer in particular.

PATHOLOGY OF COAL WORKERS' PNEUMOCONIOSIS

As the normal dust clearance mechanisms of the lung are overwhelmed, dust deposition increases. With the initiation and progression of fibrosis, the lung lesions increase in size and number. A focal collection of coal dust in pigment-laden macrophages around dilated respiratory bronchioles which tapers off towards the alveolar duct is initially apparent (Hepplestone, 1954). This is the coal macule, the characteristic lesion of CWP (Figure 11.7). A fine network of reticulin within this collection of cells may be visible early on. Focal emphysema is a specific entity that is an integral part of the simple lesion of simple CWP (Figure 11.8). It is characterized by enlargement of

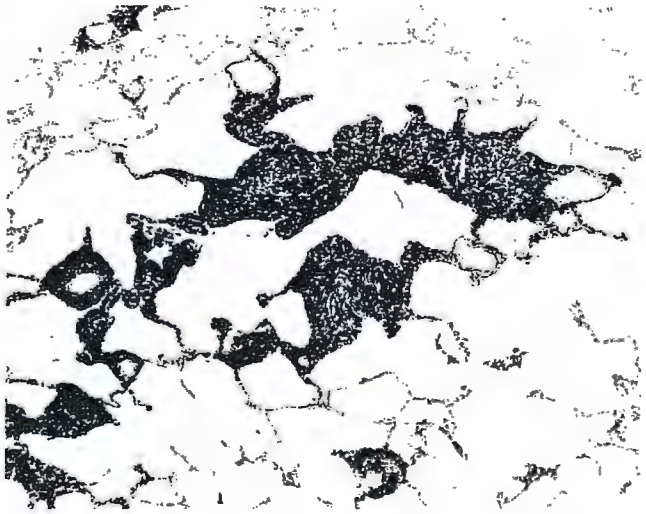

Figure 11.8 A coal macule surrounding a respiratory bronchiole. As the macule enlarges, the smooth muscle atrophies, and the bronchiole enlarges. Of interest in this photomicrograph are the enlarged air spaces surrounding the macule, a feature described as focal emphysema. (Figure courtesy of V. Vallyathan, PhD.)

the air spaces immediately adjacent to the dust macule (Kleinerman *et al.*, 1979).

What impact this has on lung function has been the subject of considerable discussion. In non-smoking, non-obstructed miners, Morgan *et al.* (1971) showed an increase in the residual volume (RV) in miners. In miners without CWP, the RV was 105% of a group of non-miners. This increased to 108% and 114% of controls in miners with category 1 or category 2 and 3 simple CWP. Although Morgan considered that this may be attributable to focal emphysema, he thought that unlikely because pathologic disruption of the bronchioles was absent. Rather, he considered this hyperinflation to be the result of coal macules narrowing the airways with alteration of the peripheral flow rates by and increasing resistance.

On gross examination of the lung, larger collections of dust are described as coal nodules, classified as micronodular if they are 7 mm in diameter or less, and macronodular if they are larger than this. These are palpable whereas coal macules are not.

PMF is diagnosed when one or more nodules attain a size of 2 cm or greater in diameter, typically on a background of simple CWP (Shennan *et al.*, 1981; Hodous, 1990) (Figures 11.9 and 11.10). The 2 cm diameter is an arbitrary choice of a minimal diameter that has allowed better correlation with clinical and radiographic measurements. Gross examination of the lung in PMF reveals a solid, heavily pigmented lung which is rubbery-to-hard in texture. These features are most common in the apical posterior portions of the upper lobes or the superior segments of the lower

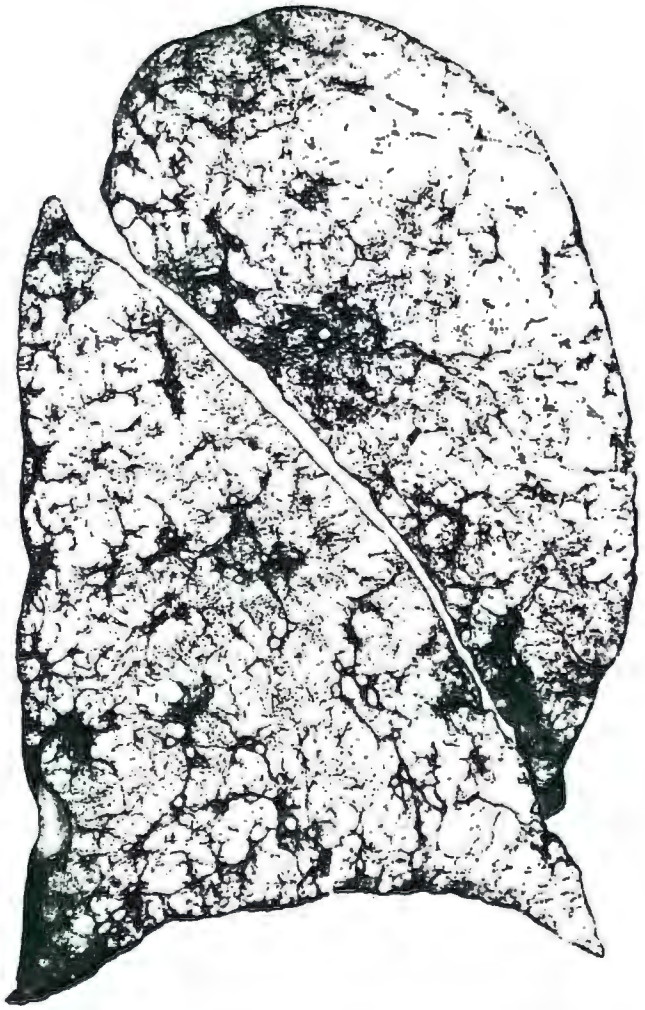

Figure 11.9 Simple Coal Workers' Pneumoconiosis (category 1) on a background of diffuse emphysema. The black deposits are the coal macules. The degree of emphysema is widespread and does not appear associated with the coal macules. (Figure courtesy of V. Vallyathan, PhD.)

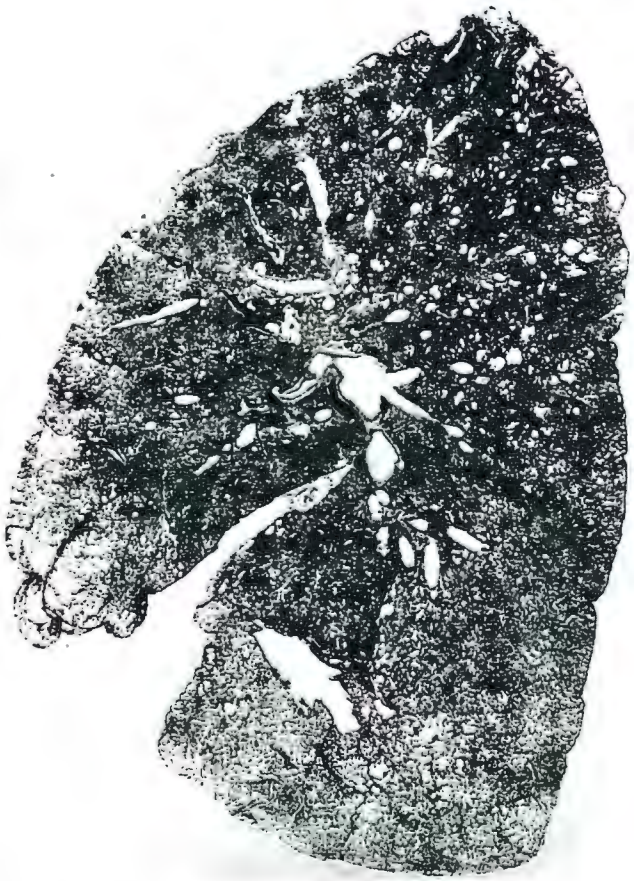

Figure 11.10 Progressive Massive Fibrosis in the upper zone on a background of coal macules, a few of which show focal emphysema. (Figure courtesy of V. Vallyathan, PhD.)

lobes. These lesions tend to occur asymmetrically, occasionally showing first in one lung and then the other, leading to a suspicion of malignancy. When these lesions are ashed, they appear to be composed of varying amounts of coal, silica, calcium and other substances. About one-fourth of the proteinaceous material in the center of these lesions is collagen (Wagner, 1972). These lesions may also cavitate and the worker may expectorate an ink-like fluid (a clinical sign described as melanoptysis), or when these cavitary lesions are cut, they may drain ink-like fluid. Airways and vessels adjacent to the lesions are distorted and destroyed within the lesions.

Silica exposures, in surface coal mine drillers, can also induce massive fibrotic lesions and result in a miner's death because of cor pulmonae and respiratory failure (Figure 11.11).

Figure 11.11 A Gough section of the lung taken from an autopsy of a 46-year-old West Virginian surface coal miner. His primary job was hard rock drilling to remove overburden. He died of silicosis with cor pulmonale and respiratory failure. The section demonstrates massive scarring with secondary emphysema and parenchymal distortion attributable to the pulmonary response to silica inhalation.

HOW CWP DEVELOPS: CELLULAR AND IMMUNOLOGIC FACTORS

CWP is the result of coal dust-induced cell damage with activation of the fibrotic process. Lapp and Castranova (1993) provided a well-outlined approach to addressing how coal dust causes lung damage. The potential mechanisms include:

- direct cytotoxicity of coal dust;
- release of oxidants, enzymes and cell membrane constituents from alveolar macrophages in association with cell death due to coal dust exposure; and
- stimulation of cytokine release from alveolar macrophages to recruit effector cells (other macrophages or neutrophils) and stimulate fibroblast proliferation and collagen synthesis in the area of coal dust deposition.

Coal dust is much less fibrogenic than silica. As an example, a mixture of 10% silica and 90% coal is far more cytotoxic to macrophages than pure coal dust (Adamis and Timlar, 1978). However, both dusts, when cleaved, show surface radicals by electron spin resonance spectroscopy. The free radicals generated by crushing anthracite coal are more numerous than those generated from crushing bituminous coal, leading to speculation regarding free radical release from different coal ranks and the suggestion that exposure to anthracite coal increases the risk for the development and progression of disease (Dalal *et al.*, 1989).

Long-term coal dust exposure in animals increases the number of alveolar cells recovered by lavage (Castranova *et al.*, 1985). In addition, an elevation in the number of blood monocytes and an increased rate of mitosis has been recognized in cells from animals undergoing chronic coal dust inhalation (Adamson and Bowden, 1978). This suggests that there was recruitment of cells into the lung from the alveolar capillaries and the interstitium of the lung. Recruited 'young' macrophages appear to be more phagocytically active than older macrophages. This may imply a more effective clearance of particles (Castranova *et al.*, 1985).

Exposure of alveolar macrophages to particles can result in the release of proteolytic enzymes, reactive oxygen species [i.e. hydrogen peroxide (H_2O_2), the hydroxyl radical ($OH^{\bullet}$) and superoxide anion (O_2^-)], and leukotrienes via breakdown of arachadonic acid in the cell membrane. Again, silica is a much stronger stimulus to the release of these agents than coal dust (Vallyathan *et al.*, 1988). Excessive release of these reactive oxygen species have the potential to overwhelm the naturally protective antioxidant system within the lung and begin the process of inflammation and fibrosis.

Activated macrophages secrete a wide variety of mediators which can attract neutrophils into the affected area and then stimulate them to release reactive oxygen species and enzymes (Borm and Henderson, 1990). Similarly, the macrophage-derived inflammatory factors can act as chemotactic agents for neutrophils (e.g. tumor necrosis factor, interleukin-8 and leukotrienes), and increase neutrophil adherence and reactive enzyme release (platelet-derived growth factor and platelet activating factor). The presence of these factors escalates the inflammatory process. Finally, secretion of many of the above factors by the activated macrophages also enhance fibroblast growth and/or stimulate the production of collagen.

Although alveolar macrophages in animals are activated after chronic exposure to coal dust, there is little lung damage. Protein and lysosomal levels in the acellular lavage fluid of these animals are not increased (Castranova *et al.*, 1985). However, when as little as 2% silica is added, the fibrotic process begins (Ray *et al.*, 1951).

Development of bronchoalveolar lavage (BAL) as a means for sampling lung cells and fluid has provided an opportunity to study the lung's response to inhalation of various dusts. Studies of the mechanisms of inflammation and fibrosis using BAL-derived material have opened the possibility for studying at least some of the mechanisms in the pulmonary reactions in CWP.

Lapp *et al.* (1991) compared 12 asymptomatic, lifelong non-smoking coal miners without pneumoconiosis with a mean of 17 years of underground mining exposure using BAL with the results from 18 controls. There was no difference in BAL mean total or individual cell counts, IgA or IgG, or spontaneous or PMA stimulated chemiluminescence. The BAL protein levels and particle-stimulated chemiluminescence was less in the miners. Although scanning electron microscopy showed a marked increase in alveolar macrophage cell surface ruffling, and transmission electron microscopy showed particles in macrophages consistent with coal, alveolitis was not present. These data are best explained by recognizing that the workplace dust exposures were insufficient to overwhelm the underlying clearance mechanisms.

Rom *et al.* (1987) studied 15 symptomatic, nonsmoking coal miners with simple CWP by BAL. They found no significant difference between miners with CWP and controls in the number of cells recovered, the percentage distribution of cells, and in the release of superoxide anion or hydrogen peroxide. This contrasted with the findings in subjects with asbestosis and silicosis, whose values for spontaneous release of oxidant superoxide and hydrogen peroxide were significantly greater than those of controls. BAL levels of fibronectin and alveolar macrophage-derived growth factor (AMDGF) were elevated but not different from the values obtained in subjects with asbestosis and silicosis. While fibrosis has not been a major part of the pathology of simple CWP, there is evidence of the presence of fibronectin in pneumoconiotic lesions (Wagner *et al.*, 1982). Wallaert *et al.* (1990) demonstrated a significantly increased number of total cells in the BAL recovered from miners with simple and complicated CWP, as well as increased percentages of alveolar macrophages, lymphocytes and neutrophils. Alveolar cells from miners with simple CWP and PMF spontaneously released significantly more superoxide, in contrast with the levels generated in the comparison group. However, when these alveolar cells were stimulated with phorbol myristic acetate (PMA), only the cells recovered from the miners with PMF showed significantly increased amounts of superoxide release.

There have been few studies of the peripheral lymphocytes in coal miners. Dauber *et al.* (1976) examined lymphocyte function of 15 miners with anthracosilicosis. They found decreased numbers of both T and B lymphocytes in the peripheral blood in the miners compared with controls. Lymphocyte functional capacity, as determined by response to stimulation by concavalin A, was less in miners with PMF compared with the response in miners with simple CWP or controls.

Overall, coal dust is much less fibrogenic than inhalation of silica dust, yet coal dust is a sufficient stimulus for the secretion and release of macrophage products. These activated macrophages can release enzymes, reactive oxygen species, cytokines and factors which cause fibroblast proliferation. All are important factors in inflammation and the development of CWP.

A less clear-cut relationship occurs when one addresses the role of the immunologic system in CWP. Soutar *et al.* (1974) reported circulating ANA and RF levels among 109 miners with radiographic evidence of pneumoconiosis. The authors did not identify the region of the country from which the miners came, or in which mines they worked. They found positive ANA in 17% and RF in 10% of the miners. The expected prevalence in the male general population of a positive ANA approximates 2–3%. The prevalence of ANA was least (9%) in simple CWP and 27% in those with category C (PMF). A similar but less striking trend was seen with RF, ranging from 6% in simple CWP to 18% in category C. Combining both ANA and RF resulted in prevalence of positive results in 13% of the miners with simple and 45% of those with category C PMF.

In 1973, Lippmann *et al.* (1973) reported a prevalence study of circulating antinuclear factor (ANA) and rheumatoid factor (RF) among coal miners in the

United States. Sera from 207 coal miners were examined. Of the 196 miners with pneumoconiosis, nine were positive for RF and 34% had positive ANA. There were regional variations in ANA that seemed to parallel the prevalence of radiographic changes; namely, a higher prevalence in the anthracite miners and a lesser prevalence in bituminous miners. These authors did not find the increased prevalence of RF in miners with PMF compared with simple CWP that Wagner and McCormick (1967) reported in Welsh miners.

Benedek *et al.* (1976) were unable to confirm the high prevalence of serum immunoglobulins and rheumatoid factor among US miners reported by European investigators. These authors matched 55 pairs of coal miners with rheumatoid arthritis with non-miners with rheumatoid arthritis. The miners and controls did not differ in respect to serum concentrations of IgG or IgA. Elevated levels of IgM were more frequent among the miners but did not reach significance. Latex agglutination test reactions were positive in 82% of miners and only 64% of non-miners ($P < 0.05$), but there was no correlation with category of pneumoconiosis.

Studies of serum immunoglobulins were also conducted by Hahon *et al.* (1980) among 155 US coal miners with chest radiographs demonstrating simple pneumoconiosis, Caplan's Syndrome and PMF. They found significantly higher serum concentrations of C3, alpha-1-anti-trypsin, and IgA and IgG in anthracite miners than in bituminous miners with PMF. There were few differences in these serum proteins among the miners with only simple CWP. They did find that compared with normal controls, the miners' C3, alpha-1-anti-trypsin and IgG values were elevated. The authors did not find an association between the elevated immunoglobulins and FEV_1.

There has been some attention paid to the role of tissue autoantibodies in CWP. Burrell (1972) used an anti-globulin consumption test to identify autoantibodies in the sera of coal miners directed at lung collagen and in the reticulin in the parenchyma and basement membranes of the lung. The lung autoantibodies tend to reside in the serum IgA. It is not clear at this time whether these autoantibodies participate in the CWP reaction in the lungs or simply represent epiphenomena. Animals injected with purified lung autoantibodies and then challenged with *M. tuberculosis* demonstrate much more extensive lesions of tuberculosis than animals receiving the *M. tuberculosis* without the prior injections of lung autoantibodies.

It has been suggested that constitutional differences may help to explain the variation in response to inhaled dusts, especially the attack rates and progression of CWP. One study looked at the prevalence of histocompatibility antigens in coal miners (Heise *et al.*, 1979). So far it has not been possible to confirm any definite association between these factors and CWP.

MANAGEMENT OF CWP

There is no proven therapy for CWP. The primary prevention of lung disease in miners must include continuing efforts at reducing coal mine dust exposure. Management is best directed at prevention, early recognition and treatment of complications. The major challenges to the physician are the recognition and management of airflow obstruction, respiratory infection, hypoxemia, respiratory failure, cor pulmonale, arrhythmias and pneumothorax.

Improved mining methods and lower dust levels appear to be reducing exposures and new cases of both simple and complicated pneumoconiosis in the US. Medical surveillance programs, using chest radiographs, allow the early recognition of workers with simple pneumoconiosis. Workers with simple pneumoconiosis should be encouraged to transfer from jobs with high dust exposure to jobs with low dust exposure. Any worker with the unexpected finding of PMF should be carefully advised about the hazards of further dust exposures.

Workers presenting with respiratory symptoms should have careful evaluation. The initial history and examination should be supplemented by chest radiography, spirometry with bronchodilators, diffusing capacity, electrocardiogram and resting arterial blood gas measurement as indicated. A thorough initial database allows accurate assessment of the worker's respiratory health and serves a starting point for observing the response to therapy or progression of disease.

Smoking cessation is important regardless of symptoms of respiratory disease, chest radiograph abnormalities or pulmonary function status. Physician encouragement to stop smoking should be supplemented by support from smoking cessation groups, use of nicotine substitutes, and behavior modification techniques.

Symptomatic reversible airflow obstruction should be treated with inhaled bronchodilator therapy as indicated. Patients with severe obstruction and inadequate improvement from the usual measures should be considered for a trial of glucorticoids.

Hypoxemia can be a serious complication in those with PMF. It is typically present first during exercise, but can occur with rest and during sleep. Chronic hypoxemia can lead to the complications of polycythemia, pulmonary hypertension, cor pulmonale and cerebral dysfunction. Therapy with low flow oxygen is

indicated when the arterial oxygen tension is less than 55 mmHg or when clinical evidence of cor pulmonale is present.

Workers with significant airflow obstruction or PMF should receive appropriate immunization with influenza and pneumococcal vaccines. Bacterial and viral episodes of bronchitis or pneumonia should be promptly recognized and appropriately treated. Similarly, miners with concomitant exposure to silica dust (most often roof bolters, drillers and motormen) deserve special attention with regard to mycobacterial infection. Symptoms of weight loss, fever, sweats, a change in sputum production, or malaise should be promptly investigated with a chest radiograph and examination of the sputum through stain and culture for AFB. Occasionally, the sputum may be negative and the infecting mycobacterial organisms can only be obtained by fiberoptic bronchoscopy with brushings and washings. Active tuberculosis in this population can be usually successfully treated with the usual drug regimens, provided that rifampin is one of the drugs used (Ball *et al.*, 1969; Dubois *et al.*, 1977). In coal miners with a significant history of concurrent silica exposure, the treatment for tuberculosis may need to be more aggressive, and long-term follow-up is indicated in view of reports of recurrent pulmonary tuberculosis in patients with PMF after completion of apparently adequate therapy (Morgan, 1979).

Pneumothorax can be particularly troublesome events in miners with pneumoconiosis. Those with bullous disease in the presence of advanced complicated pneumoconiosis appear to be at the greatest risk. Typically, once the lung collapses, it is difficult to expand, a feature attributable to the decreased compliance associated with interstitial lung disease. Therapy with one or several chest tubes is often therapeutic; however, recurrent pneumothorax which cannot be expanded may require an open procedure and pleurodesis.

Respiratory failure may complicate advanced PMF as it does in other chronic respiratory diseases. Ventilatory support measures are indicated when the failure is precipitated by a treatable complication. The application of ventilatory support measures should be discussed with the patient before the need arises. In general, miners with advanced pneumoconiosis are poor candidates for long-term mechanical ventilatory therapy.

PREVENTION STRATEGIES FOR COAL WORKERS' LUNG DISEASES

The control of coal workers' lung diseases in both the developed and developing world requires comprehensive prevention strategies, including exposure control, medical surveillance, research and education. Example approaches include:

- Major efforts must be directed to improving work methods or work practices, including engineering controls, to progressively reduce dust exposures to acceptable levels. These efforts are labelled *primary prevention*. Personal respiratory protection can be utilized as a secondary measure, to reduce exposures during short-term operations or unusual/emergency conditions, or while exposure control measures are being modified or improved. The use of respirators will only be effective when it is part of a comprehensive respiratory protection program, and should never be relied upon outside of such a program.
- Primary prevention should involve ongoing environmental dust monitoring, and assure mechanisms for feed-back to improve and modify working conditions if exposures above acceptable levels are observed.
- *Secondary prevention* through medical screening and surveillance should be designed to benefit the individual worker and future workers. Illness identified through medical screening represents a failure of primary prevention, and thus should trigger feedback to those involved in environmental monitoring and work practice evaluations.
- Education about the respiratory health hazards from uncontrolled exposures to silica and coal mine dust must be available to workers, employers, managers and health care providers.
- Information on the cumulative burden of disease should be monitored over time for CWP control.
- Research into mining-related lung diseases should be encouraged to improve recognition, monitoring, exposure reduction and therapy, and to increase understanding of pathogenesis. Research efforts should supplement, not displace, attention to dust control.
- Clinicains who recognize coal-related diseases in their patients should attempt to determine whether ongoing workplace exposures present a continuing risk to current workers, while maintaining the confidentiality of the patient–physician relationship. Assistance in this can often be obtained through local or regional health departments, occupational medicine groups or central governmental agencies. Reporting of occupational diseases is required in some jurisdictions.

SUMMARY

CWP is a disease distinct and separate from silicosis. Although the coal particle is not nearly as fibrogenic as the silica particle, excessive exposures over a period of

time can overwhelm effective clearance mechanisms and initiate chronic interstitial lung disease. Simple CWP is clearly related to the amount of dust deposited within the lungs. PMF most often occurs on a background of simple CWP and is the result of dust deposition plus other inadequately defined host factors. Immunologic and local cellular factors may contribute to the development of this form of the disease.

PMF is clearly associated with alterations in ventilatory, mechanical and vascular function of the lungs. These abnormalities in PMF contribute to the premature morbidity and mortality of this disease. Similarly, there is no specific therapy for CWP. Prevention remains the cornerstone of eliminating this occupational lung disease. The education of workers and employers regarding the hazards of coal dust exposure and measurement and effective control of dust exposure remains the sole means of eliminating this disease.

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