19.10 Respiratory Diseases of Coal Miners

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HISTORY

Coal production

The growth in coal mining was virtually coincident with the Industrial Revolution. Although shallow mining of coal seam outcrops is reported to have occurred since the 9th century, the 18th century brought increased demand for coal as well as the technology to pursue the mining of seams well below the earth's surface. By the early 1800s coal mining had become an important industry both in the United States and abroad. Employment in coal mining peaked about 100 years later in 1923, when over 800,000 coal miners were working in US mines. From that point, although production and consumption of coal continued to increase, mechanization progressively reduced the size of the work force. In 1999, average employment in coal mining work in the US was 108,244, down from 132,535 six years earlier. Between 1993 and 2002, annual US production remained stable at about 1 billion short tons of bituminous and 1.5 million tons of anthracite coal. Currently, about two-thirds of coal production is at surface mines, while about 57% of miners are employed at underground mining operations.[1]

The principal US coal deposits are shown in Figure 19.10.1. Coal production has recently been fairly evenly divided between Eastern and Western coalfields, but the proportion of production has been increasing in Western states and now exceeds that of the Appalachian coalfields.

Health effects

In Europe, recognition of the adverse health consequences of coal mining followed the marked increase in the number of miners. Wedel, in 1672, wrote of 'miners asthma', but was probably referring to hard rock miners. According to Kerr, the term was first applied to coal miners in 1822. Laennec described the black pigment in the lungs of coal miners as 'melanosis' in 1806, and by 1819 clearly differentiated the condition from malignant melanoma. Several years later, the term 'miners' black lung' was used to describe the disease in Scotland. In 1919, silicosis became a 'certifiable disease' in the UK, and British miners with pneumoconiosis became eligible for certain benefits. Based on studies among South Wales coal workers, a high prevalence of pneumoconiosis became evident in the 1930s, and the condition 'coal workers' pneumoconiosis' (CWP) was differentiated from silicosis in the early 1940s.

Unfortunately, this awareness of CWP failed to cross the Atlantic. A mine explosion in Farmington, West Virginia, on November 20, 1968, was widely reported on television, and graphically illustrated to the nation the plight of coal miners in the US. This tragedy added momentum to the movement to improve coal mine health and safety conditions. 'Black lung associations' were organized in the coalfields. A 3-week long strike in the West Virginia coal mines, one of the largest and longest strikes ever called on the single issue of occupational health, ended when the state legislature passed a bill in 1969 making CWP a compensable disease. By the end of 1969 the Federal Coal Mine Health and Safety Act had been passed into law. Dust control and safe practices in coalmines were mandated and backed up by inspectors with the threat of fines or closures, and the right of the miners' representatives to participate in inspections.

MINING JOBS

An understanding of mining techniques is useful in the evaluation of respiratory diseases of miners, since different activities involve exposures to different degrees of risk. Activities at coalmines are generally classified into face, non-face, and surface work. By virtue of multiple activities, some workers spend portions of their workday in two or more of these mine locations.

Face workers

Miners working at the edge of the unmined coal seam, or coalface, are engaged in the actual removal of coal from the working 'section' of the mine. Face miners are generally exposed to the highest concentrations of respirable airborne dusts, particularly carbon, but may also be exposed to silica dust when the cutting bits strike silica-bearing rock immediately adjacent to or within the coal seams. There are three primary methods of mining coal underground that have been employed in the US. They are discussed below.

Continuous mining

Room and pillar mining with 'continuous mining' machines is one of the commonly used coal mining techniques in the US today. Continuous miner operators and their helpers direct the rotating bits on the machine into the coal seam to rip out the coal. The loosened coal is pushed to the back of the machine, where a loading machine operator and helpers use a large scoop to load the coal into a rubber-tired buggy (shuttle car), or directly onto a conveyor line.

Pillars of unmined coal are left at the sides of the advancing tunnel to help support the roof. As the continuous mining machine advances, roof bolters and their helpers install steel plates to strengthen the unsupported roof of the tunnel. Holes are drilled into the hard rock above the coal seam and long steel bolts inserted, along with glues, to anchor these plates. Silica exposure is common in roof bolting, and there may also be exposure to resins and plasticizers in glues. Timber men install wooden posts to further stabilize the roof of the tunnel. Brattice men install curtains or tubing to direct the flow of ventilation at the working face. Shuttle car ('buggy') operators transport newly mined coal in rubber-tired vehicles (Fig. 19.10.2). Scoop car oper-

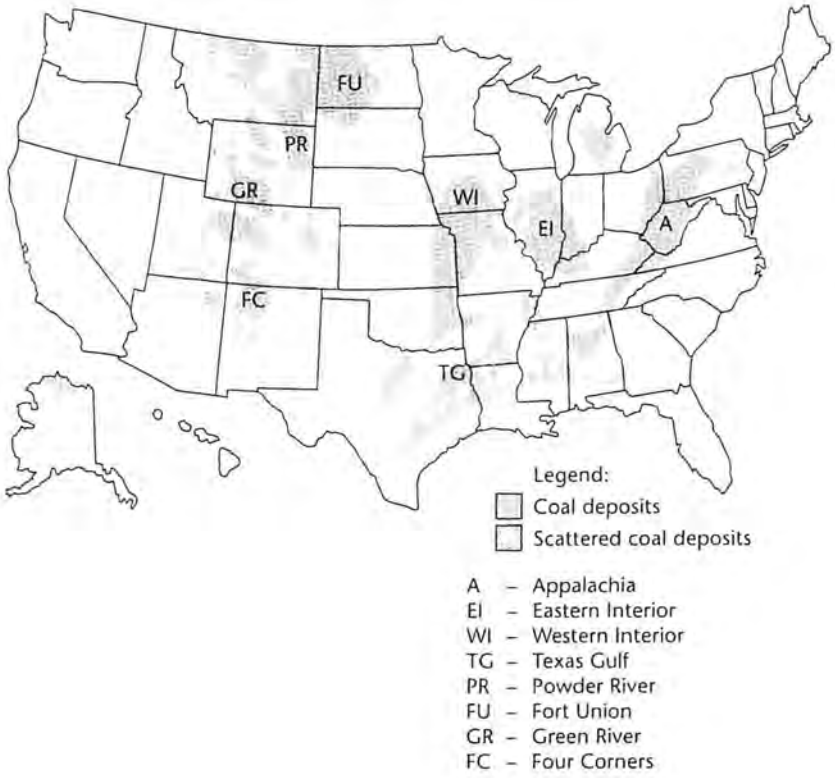


Figure 19.10.1: Map of United States coal deposits.

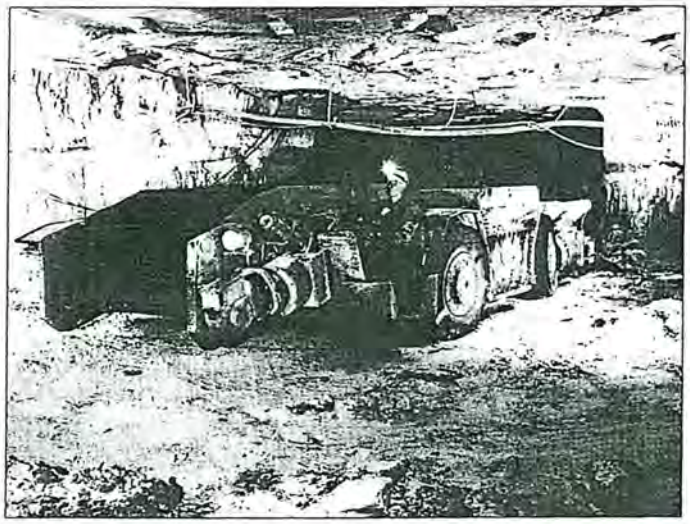


Figure 19.10.2: Coal miner operating rubber-tired shuttle car.

ators run battery-operated vehicles throughout the mine, transporting coal or mining supplies. Mobile bridge operators operate portable conveyor lines to move coal away from the face. Section maintenance workers repair and maintain the underground heavy equipment at the face in all mining methods.

Longwall mining

'Longwall mining' is currently the most cost-efficient and highly productive method of underground coal mining. Production using this technique is increasing as an alternative to room and pillar mining. The longwall machine spans several hundred to a thousand feet or more along the active coalface. A rotating drum (shear) or a plow moves back and forth along the face, breaking coal loose from the seam and allowing it to fall onto a conveyor line. A series of hydraulic 'jacks' support the roof along the face, and are advanced as the coal is removed. Behind the longwall device, the unsupported roof is simply allowed to fall. This minimizes the loss of the coal in pillars and the need for roof support activities, as in room and pillar mining. To develop the mining section in advance of the longwall machine, one or two continuous miner crews are generally operated. However, to run the longwall section itself, a smaller crew of longwall operators, mechanics, headgate operators, and jack setters is required.

Respirable dust exposures in longwall mining have been difficult to control. In recent years, nearly 40% of reported dust measurements on longwall sections have been over the 2 mg/m^3 respirable dust standard, compared with 10–15% on continuous mining sections.

Conventional mining

Little coal is mined in the US today using the 'conventional mining' technique, although many current miners have used this method in prior jobs. In this system, cutting machine operators and helpers make a deep cut in the coal face at the bottom of the seam. 'Shot firers' drill deep holes in the area of the cut and place explosive charges to blast the coal free. 'Hand loaders' or loading machine operators shovel the coal into vehicles, which then transport it away from the face. In addition to dust, important exposure to nitrogen oxides from the explosives may occur using this technique.

Non-face workers

Several underground mining activities relate to reducing dust or methane gas hazards. Masons construct plastic, cloth, or block barriers to keep the flow of air directed across the working face. Rock dusters scatter hygroscopic powdered limestone along the cut faces of the mine tunnels, to moisten and trap loose dusts and reduce the danger of explosion. Although this job may involve considerable visible dust and irritation, the lung toxicity of the limestone is considered to be lower than the other mine dusts. Electricians, mechanics, and welders may work with equipment throughout the mine.

Workers on non-face transportation have several types of exposure. The coal is loaded at the working face (see above) and transported to the main haulageways, where it is dumped into train cars or a conveyor belt and taken out of the mine. Motormen operate the train locomotives. To improve traction on the steel rails, motormen drop sand or other abrasives on the rails. The materials are fragmented into respirable particles and resuspended by passing trains, creating a mixed dust or silica exposure hazard. Belt men patrol the conveyor belts, assuring the continued movement of coal, and shoveling up spilled coal. In some mines, diesel exhaust exposure may be present from diesel-powered vehicles.

Surface workers

With a few important exceptions, surface workers generally are exposed to lower levels of respirable dust. Surface maintenance workers frequently perform welding and cutting on equipment, and may be exposed to welding fumes as well as asbestos from shields and gloves. Tipple operators and preparation plant workers are frequently exposed to higher levels of dust during the cleaning, processing, and loading of the coal. Lampmen and others at the mine portal often have little dust exposure.

Equipment operatives at surface coal mines, particularly high-wall drillers and their helpers, and to a lesser extent bulldozer operators, frequently experience important silica exposures while drilling and removing overburden. Surface coal miners have dust exposures that are generally lower than those of underground miners, but a risk of both simple and complicated pneumoconiosis remains.[2]

In summary, modern mining techniques bear little resemblance to the pick-and-shovel techniques of the early 1900s. Highly mechanized equipment allows mining more coal with far fewer miners. Unfortunately, the high production intrinsic to these operations may lead to dust levels that are difficult to control, and strict attention is necessary to avoid unhealthful conditions.

TERMINOLOGY

Overview

When dealing with respiratory diseases of coal miners, it is important to be familiar with disease definitions. Differing terms and different definitions have been used for clinical, epidemiological, pathological, and legal or legislative purposes.

In the clinical arena, use of the term 'pneumoconiosis' tends to be restricted to the radiologic or pathologic appearances relating to the accumulation of lung dust deposits and the associated tissue reactions. This usage is often interpreted to exclude other lung abnormalities, such as those associated with bronchitis or emphysema, although, as noted below, focal emphysema is often considered an integral component of the pathologic lesion of coal workers' pneumoconiosis. Such distinctions between the different mining-related disease processes may facilitate the diagnosis, treatment, and study of disease, but has tended to lead to fragmented understanding and assessment of the totality of lung disease associated with exposure to coal mine dust.

Generic definitions of pneumoconiosis, which include all dust-related effects on the lung, are also widely used. Some of these definitions are employed more in the non-clinical sphere, particularly with regard to legal/legislative activities, and include dust-related effects that may not be radiographically apparent, such as dust-induced chronic airflow obstruction. The lay term 'black lung' is a generic term used by miners for lung disorders associated with their work.

Since the radiographic pattern of CWP may be quite distinctive, pneumoconiosis in coal miners often has been defined based upon a radiographic appearance. For example, in research publications, confirmation of CWP may require that several readers concur in the finding of a certain shape and profusion on a chest film (for example, 1/0 rounded opacities using the International Labor Office system of classification of radiographs for pneumoconiosis) (for more detail, see Chapter 19.1). Although this approach increases precision, it may reduce sensitivity for several reasons: (1) it is recognized that readings of 0/1 rounded opacities also correlate with mine dust exposures; (2) the profusion of *irregular* as well as rounded opacities seen on the chest radiograph increases with increasing mining exposures; (3) the routine chest radiograph may be normal in the presence of clinically important and pathologically identifiable interstitial lung disease; and finally (4) several dust-related diseases, including bronchitis and emphysema, may not be apparent radiographically.[3] In spite of this, in the United Kingdom, for example, airflow obstruction among coal miners is compensable only for miners who also have radiographic evidence of pneumoconiosis.

Post-mortem lung pathology has shown that nodular radiographic changes correlate quite well with pathology: ILO q-type opacities on chest films are associated with coal macules and micronodules, while r-type opacities are associated with macronodules. The profusion of p opacities, and to a lesser extent q-type opacities, has been found to reflect the dust content of the lung at autopsy. With regard to large opacities, about two-thirds of those seen on chest films are subsequently confirmed on pathology. However, routine chest x-rays are not entirely sensitive for detecting CWP lesions, which may be found pathologically or on high-resolution computed tomography in miners with

normal routine chest films. Among miners whose lungs revealed progressive massive fibrosis (PMF) on pathologic examination, 22% had prior chest x-rays showing no radiographic large opacities.[4,5]

In summary, for clinical purposes, coal miners with typical radiographic or pathological findings of pneumoconiosis are properly diagnosed as having coal workers' pneumoconiosis. For legal and compensation purposes, a broader definition of 'pneumoconiosis' is used, which includes dust-related effects that may not be radiographically apparent.

Specific medical terms relating to respiratory diseases in coal miners

Simple CWP. This lesion is defined clinically in miners showing multiple radiographic shadows up to 10 mm in diameter. These dust-related shadows are usually rounded, although irregular shadows may also be noted in combination with rounded opacities, or occasionally alone. The pathological correlates of the chest radiograph are discussed in the text.

Complicated CWP. This lesion is also defined clinically based on a chest radiograph showing a dust lesion or lesions over 10 mm in diameter. Complicated CWP is found most often on a background of smaller rounded opacities, with the risk increasing with increasing profusion. Progressive massive fibrosis (PMF) is a term often used interchangeably with complicated CWP, although not all of the larger rounded shadows seen in coal miners will progress with time.

Silicosis (see Chapter 19.9). This is the chronic interstitial lung disorder caused by the inhalation of respirable crystalline silica. Radiographically, silicosis is also characterized by small rounded opacities, which may coalesce to form shadows larger than 10 mm (complicated silicosis or PMF). Although silicosis is pathologically distinct from CWP, the two disorders often cannot be distinguished radiographically. The lungs of coal miners frequently show lesions consistent with both disorders.

Bronchitis (see Chapter 19.4). Sometimes referred to as industrial bronchitis, this dust-related disorder is characterized by excessive cough and sputum production. Sputum production is deemed excessive when it occurs on most days for at least three months a year for two or more years. The pathology of this disorder has been less well studied.

Focal emphysema. A form of airspace enlargement with tissue destruction, found in association with, and an integral part of the macular lesion of simple CWP.

LUNG PATHOLOGY

Pigmented lesions

The most characteristic and striking pathologic changes in the respiratory system of coal miners are associated with the accumulation of dark pigment, primarily located in lung macrophages. Localized dust macules are considered pathognomonic of CWP.[6] They are located at the level of the respiratory bronchioles, and are generally associated with deposition of reticulin, and with destruction of adjacent alveolar walls (focal emphysema), but with only minimal collagenous scarring (Fig. 19.10.3). With increasing dust deposition, the pigmented macule progressively enlarges, becoming solid and palpable. Adjoining macules may coalesce. At this stage, the lesions show clear collagen deposition, and are labeled nodular lesions. Destructive vascular lesions are commonly associated with nodules.

With further progression of the disorder, coalescence of nodules can result. The massive lesions of complicated CWP (progressive massive fibrosis) may also appear. These lesions are greater than 1–2 cm in diameter, and are usually seen in upper lobes or superior segments of lower lobes of lungs with extensive pigment deposition (Fig. 19.10.4). PMF lesions may be unilateral, bilateral, and multiple, and may show cavities containing black liquid. The lungs of miners with complicated CWP also usually show pathologic changes of bronchitis and extensive emphysema.

Figure 19.10.3: Coal dust macule.

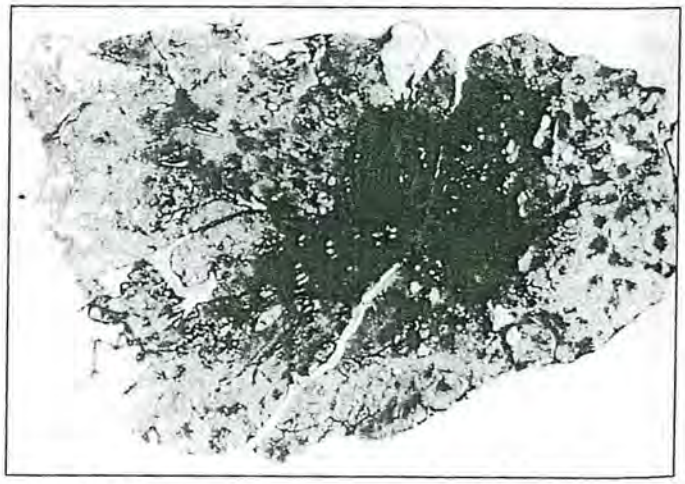


Figure 19.10.4: Gross pathology of complicated coal workers' pneumoconiosis

Cardiovascular changes consistent with cor pulmonale can also be seen. Right ventricular hypertrophy has been correlated with the thickening of pulmonary vessel walls seen in association with increasing severity of CWP, and emphysematous changes in miners' lungs.[7,8]

Factors facilitating progression to complicated CWP are not entirely understood. Excessive lung dust deposition is clearly the basis for the condition. The greatest risk factor for subsequent development of complicated CWP is the miner's radiographic category of simple CWP, with an additional effect of increasing age. The risk of progression to PMF in a miner with category 2 simple CWP is 3–4 times that of a miner with category 1. Exposures at a young age increase the duration of residence of the dust in the lung and also the risk of PMF.[9] In the past, mycobacterial infection was frequently associated with PMF, and was considered a precursor to its development. It is now accepted that PMF lesions often develop in the absence of infection. The role of excessive silica exposure is still debated, although PMF can clearly develop in workers with scant silica exposure. Miners, particularly those with simple or complicated CWP, have an increased frequency of autoantibodies and other serologic abnormalities. However, no clear role in disease pathogenesis of these findings has been defined.

Although it is rarely reported in the United States, dust-exposed miners with rheumatoid arthritis may develop a syndrome known as rheumatoid pneumoconiosis, or Caplan's syndrome (see Chapter 23.5). Features include multiple large (up to 5 cm) lung nodules developing rapidly, often over several months, with little or no background profusion of simple pneumoconiosis. The pulmonary lesions may cavitate, and occasionally will precede the onset of the joint disorder. Pathologically, the lesions are distinct from the typical lesion of PMF, and are similar to rheumatoid nodules.

Even in non-miners, the extent of pigment in the lungs generally increases with age. It is also greater in cigarette smokers than non-smokers. However, in the absence of occupational exposures, the deposition of pigment will very rarely be sufficient to form macular lesions.

Typical lesions of silicosis are described elsewhere (see Chapter 19.9). Overall, classical silicotic nodules have been reported at autopsy in the lung of 12.5% of coal miners. Higher prevalences are observed in certain jobs, such as motormen (25%), and in miners with complicated CWP (over 50%).[10]

Emphysema

The occurrence of emphysema in CWP has been recognized since the time of Osler, and is considered by some observers to be an integral component of the dust macule. Cigarette smoking may also result in emphysema of the centrilobular type, which differs from the emphysema due to coal dust only in the extent of lung involvement and the absence, in non-miners, of associated dust pigment. Generalized emphysema can be seen in the lungs of both smoking and non-smoking coal miners. After taking tobacco use into account, the extent of emphysema correlates with prior mine dust exposure, as well as the amount of dust retained in the lung. Opinions differ among pathologists as to whether the emphysema associated with the dust macule is different in any way from that related to tobacco use. However, the severity of airflow obstruction measured during life correlates significantly with the extent of pathologic emphysema in the coal miners' lungs at autopsy, suggesting an important functional effect.[11]

Bronchitis

The prevalence of chronic cough with sputum production is elevated in miners, increasing with mine dust exposure. It is generally accepted that coal mine dust deposition in the airways over prolonged periods of time leads to mucous gland enlargement and proliferation of goblet cells. However, few investigations have been performed regarding the histology of the airways and mucous glands in coal miners. The lungs of miners with complicated CWP frequently show the changes of chronic bronchitis. Some autopsy studies of miners have also shown a significant correlation between prior coal mine dust exposure and the proportion of mucous glands in the bronchial walls (the Reid Index). This is considered evidence that dust exposure contributes to the pathologic changes, as well as to the clinical symptoms, of chronic bronchitis. No significant relationship was found between airway mucous gland changes and the severity of pneumoconiosis.

In British coal miners there is a markedly different geographical distribution of mortality from pneumoconiosis versus chronic bronchitis and emphysema, suggesting that the exposures and mechanisms for these responses are different.[12]

Mycobacterial infection

Tuberculosis and non-tuberculous mycobacterial infections are seen in coal miners. The risk of these infections is generally considered to be increased with heavier dust exposures, particularly in miners with PMF. Some miners also have lesions of silicosis, which represent a greater risk factor than CWP alone for these infections. As in the general population, response to appropriate chemotherapy is usually good for tuberculous infections, and less satisfactory for most atypical mycobacterial disease. Tuberculosis is no longer considered an important factor in the development of complicated CWP in most countries, but is still associated with PMF onset and results in increased mortality among coal miners in some countries (e.g., China).[13]

Other conditions

Coal deposits also contain other variable components, including minerals such as crystalline silica and kaolin, as well as a polycyclic aromatic hydrocarbons (PAHs). In 1997, the International Agency for Research on Cancer determined that inhaled crystalline silica is a human carcinogen.[14] Thus, there has been an ongoing concern for a potential lung cancer risk among coal miners. In contrast to the finding

Pigmented lesions:
Dust macule
Coal nodule
Progressive massive fibrosis (PMF)
Silicotic nodule
Centrilobular emphysema
Bronchitis
Rheumatoid pneumoconiosis

Table 19.10.1 Pathologic classification of coal mine dust-induced changes

with crystalline silica, no consistent increase in lung cancer has been observed in relation to inhalation exposure to coal mine dust. However, several studies have reported an increased risk of gastric cancer among coal miners (see Chapter 30.5).[15]

Extracting coal requires the application of large amounts of mechanical energy to the coal, and injuries remain a constant threat to miners. In 2000, the US rates of fatal and non-fatal lost-time injuries in coal mining were 0.040 and 5.18 per 200,000 employee work-hours, respectively, or about three deaths and 356 non-fatal injuries per 10,000 miners annually. Discussion of injury risks is found in Chapter 31.

In summary, several pathological lesions have been identified in the lungs and airways of coal miners (Table 19.10.1). Some are rarely seen in the absence of extensive inhalation of coal mine dust, and therefore are accepted as generally related to occupational exposure. Some lesions have been correlated with the appearance of characteristic changes of CWP on the chest radiograph. Other lesions, while similar to those found in the general population, are seen with greater frequency in workers exposed to coal mine dusts. In the lungs of underground coal miners, macules, nodules, and massive lesions, as well as the emphysema associated with these lesions, and silicotic nodules, are accepted as almost uniformly related to mine dust exposures. In contrast, overall emphysema scores, carbon pigmentation, and possibly Reid indices are increased in miners' lungs in relation to their mine exposures, but can also be increased by other inhaled agents, depending on the relative exposures to mine dusts and other materials such as tobacco smoke.[11]

PATHOGENESIS

Observations from both human and animal studies have implicated overloading of lung clearance mechanisms in coal mine dust-related disorders. Excessive exposures trigger impairment of alveolar macrophage (AM)-mediated lung clearance, progressive accumulation of particle-laden macrophages, and subsequent inflammatory changes in the lung.[16] Once deposited, dust particles are thought to trigger release of mediators, including reactive oxygen species and related antioxidant protection mechanisms, as well as cytokines, growth factors, and related proteins. Tissue damage and remodeling in the respiratory tract results from modifications of the extracellular matrix. A number of animal and human studies have addressed these molecular mechanisms of lung injury from coal mine dust inhalation. With dust exposure, increases in leukocyte recruitment as well as neutrophil adhesion have been observed, resulting in retention of inflammatory cells in the lung.[17]

Alveolar macrophages obtained from healthy subjects, exposed in vitro to coal dust particles, demonstrate release of tumor necrosis factor alpha (TNF-α) and interleukin-6 (IL-6). Alveolar macrophages from miners with CWP release higher levels of TNF-α and interleukin-1. Macrophages from dust-exposed workers with respiratory impairment also release increased amounts of oxidant species.[18] Bronchoalveolar lavage fluids from miners with CWP show an influx of mononuclear phagocytes, with an increased spontaneous production of oxidants, fibronectin, neutrophil chemotactic factor, and also of IL-6 and TNF-α. This spontaneous cytokine release is associated with an increased expression of cytokine messenger ribonucleic acid. Additional studies are needed to more fully characterize the cellular events and mediators responsible for the unique pattern of airway and parenchymal lung injury that is seen with coal mine dust inhalation.

RADIOLOGY

Radiographic changes in coal miners

Several patterns of abnormality on routine posteroanterior chest radiographs have been related to coal mine dust exposure. Most commonly, fairly discrete small nodular radiographic shadows are seen in the lung fields. These densities are usually rounded in shape, although they may be irregular, and are seen in greater numbers in the upper and middle, compared to the lower lung zones, with prominent lower zone involvement seen only occasionally. Typically the largest diameter of the small nodules categorized as simple CWP is 3 mm or less, but may be up to 10 mm. With increasing lung dust deposition, the number of opacities observed in a lung zone (profusion) increases. The normal vascular shadows of the lung become obscured.

Further progression may be indicated by a coalescence of the small opacities into a combined density, which may be quite homogenous. By convention, if a dust-related radiographic shadow is larger than 10 mm, it is categorized as complicated CWP or progressive massive fibrosis (PMF). Shadows of PMF are frequently bilateral, occurring in the upper and mid-lung zones. As they enlarge they may migrate toward the hilum, forming a sharp lateral margin delineated by a zone of emphysematous lung. Often the lesions are parallel to the chest wall, and are seen to have a greater diameter on the posteroanterior film than on the lateral view. If ischemic necrosis of the lesion occurs, a central cavitation or lucency may be noted.

Small irregularly shaped radiographic shadows are also observed in the lungs of miners. The finding of irregular densities, in contrast to small rounded densities, has more consistently been associated with a reduction in gas transfer and/or ventilatory lung function. The tissue pathology associated with these radiographic shadows is unclear. They have been correlated with increasing dust exposure, increasing age, and cigarette smoke, and are thought to

have multiple causes, including both pathological emphysema and dust-induced fibrosis.

Under the ILO classification scheme, the small, rounded type of radiographic changes seen early in CWP are usually classified as 'p' or 'q' type opacities. The larger 'r' type opacities are more commonly associated with silicosis. As mentioned, irregular opacities (ILO type 's', 't', or 'u') may also be noted. A typical radiographic appearance of simple CWP is shown in Figure 19.10.5. The detail shows the opacities to be less than 3 mm in diameter ('q' type), and the profusion was interpreted as category 2.

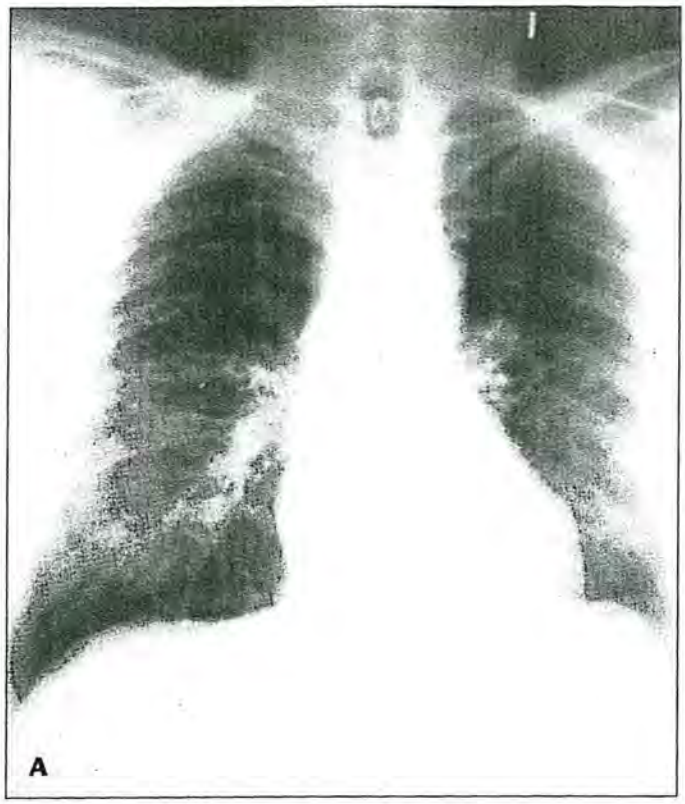
Course and progression

Much of the information on the course and progression of radiographic changes in coal miners has been derived from long-term epidemiologic studies of coal miners in Britain. These were greatly facilitated by the concurrent collection of dust exposure data. In the main, these studies have shown that the overwhelming determinant of simple pneumoconiosis is the extent of exposure to coal mine dust per se. In addition, different coals are categorized by their 'rank', a characteristic which appears to have some influence on disease development. Rank is a factor which is related to the hardness and degree of metamorphosis of the coal due to heat and pressure.

High-rank coals, such as anthracite, have been associated with a greater risk of CWP and lung function deficits than lower rank and softer coals, such as bituminous coal or lignite. Silica may also play a role in the development of radiographic changes in underground coal miners, especially when experienced at high concentrations.[19] No other environmental factor has been shown to have a major effect. Tobacco use appears to have little effect on the development of the radiographic findings of CWP.

The main recognized risk factor for development of PMF is the category of simple CWP, particularly ILO categories 1/2 or greater.[20] Hence prevention of simple CWP must remain a priority. Recently it has been shown that there appears to be an exposure–response relationship between PMF and dust exposure in miners without radiographic evidence of simple CWP. Other factors pertinent to PMF development are coal rank, age of the miner, and residence time of dust in the lungs. Even though clear and consistent exposure–response trends have emerged from the many studies undertaken in the United Kingdom and elsewhere, much unexplained variability remains for both simple and complicated CWP. Large variations between mines in prevalence of diseases are seen that cannot be explained by recourse to available information on dust levels and composition.

The current federal dust standard in the US is 2 mg/m^3 (but may be further reduced when silica levels are high). This limit was derived from early British work which indicated that progression to category 2 or greater simple CWP would be prevented at this dust level. By this means, it was expected that further progression to PMF would be eliminated. As an additional health measure, an x-ray screening program was set up to identify miners with signs of CWP, and to offer them the right to work in a reduced dust envi-



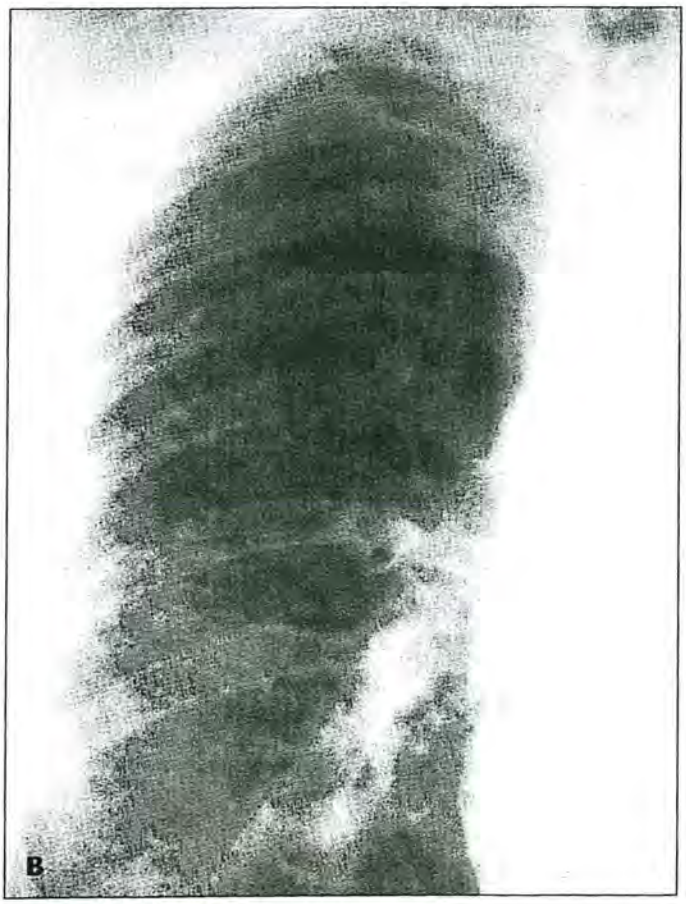


Figure 19.10.5: (a, b) Chest radiographic pattern of category 2 simple coal workers' pneumoconiosis with detail.

ronment, and have their work environment monitored more frequently. Dust-induced lung functional changes in the absence of radiographic changes were not considered during promulgation of the US standard.

Since the establishment of dust controls in the US, the prevalence of radiographically apparent CWP among underground miners has declined considerably.[21] Unfortunately, recent work from Britain and elsewhere suggests that the current US disease prevention strategy may be flawed. First, the more recent exposure–response models predict somewhat higher levels of disease than did the early work for the same dust exposure. In particular, 2 mg/m^3 is no longer associated with zero incidence of category 2 or greater (Fig. 19.10.6). Second, as noted earlier, it has been shown that PMF can develop when prior radiographs show no identifiable changes of simple CWP, and that this development is dose-related. Moreover, results from Britain have shown a substantial amount of PMF developing on a category 1 background. A survey of retired miners in Northern France also demonstrated the onset of simple or complicated CWP in 24% of miners who had normal radiographs at retirement. Finally, as discussed below, radiographic surveillance programs do not identify miners with dust-related reductions in ventilatory lung function that are not associated with radiographic changes of pneumoconiosis.

There is additional evidence that current US prevention strategies may not be entirely protective. Henneberger and colleagues found that, even after the establishment of a 2 mg/m^3 respirable dust standard, exposures among US coal miners were associated with an increased risk of respiratory symptoms.[22] As well, miners exposed for a working lifetime to the current US standard of 2 mg/m^3 respirable dust appear to have an elevated risk of dying from pneumoconiosis and COPD.[23]

In summary, there is no doubt that the current US health and environmental standard has led to a reduction in disease levels compared to those in the past, and there has been a significant decline in CWP mortality in the US, particularly among miners over age 45.[24] However, several recent reports have challenged the efficacy of the current US dust standard, and imply that it may be less protective than originally intended. A recent publication extensively reviewed current knowledge of the health implications of coal mine dust exposures.[25] Based upon a critical review of the published scientific studies, the authors calculated the excess health risk (beyond that due to non-occupational factors including tobacco smoking) during work at the current US exposure limit for respirable coal mine dust (2 mg/m^3). Utilizing published exposure–response studies, it was estimated that for miners of low-rank bituminous coal working at the current exposure limit over a working life (45 years), 11–16.5% will develop pneumoconiosis, with 2–3% developing progressive massive fibrosis. Lower risks were projected for Western coal fields, and higher risks during the mining of Eastern higher rank and anthracite coals. In a similar calculation, clinically significant airflow obstruction (FEV_1 less than 65% of predicted normal) is projected to develop in 1–2% of miners working at the current dust exposure limit. Based upon these and other findings, a reduction in permissible exposures to 1 mg/m^3 was recommended. Additionally, since health risks would not be eliminated by the exposure reduction, medical monitoring was recommended using questionnaires and spirometry, in addition to chest radiographs.[25] However, at the time of writing, neither the exposure reduction nor monitoring recommendations have been implemented (see below)

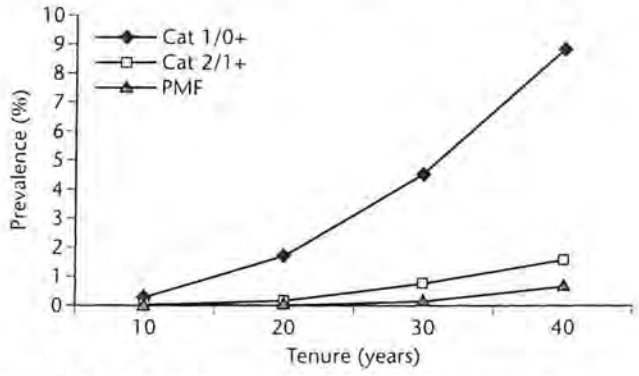


Figure 19.10.6: Predicted prevalence of radiographic coal workers' pneumoconiosis by mining tenure, assuming dust concentration of 2 mg/m^3, 83% carbon. Based upon British Field Research. (Adapted from Attfield[45] © 1992 American Public Health Association.)

Contemporary trends in CWP

Several radiographic surveys of selected mines, performed as part of the US National Coal Study, provide information regarding the prevalence of radiographic CWP in the US. The prevalence of the more advanced simple CWP (category 2 or greater) in miners with over 30 years tenure declined from about 11% in the first round of radiologic studies in 1971, to 8% in 1980, and to about 2% in 1988. The overall tenure-adjusted prevalence of CWP declined over the four surveys (Fig. 19.10.7). Some of the observed decline was found to be related to differences in the x-ray readers, as well as the use of a new version of the ILO chest

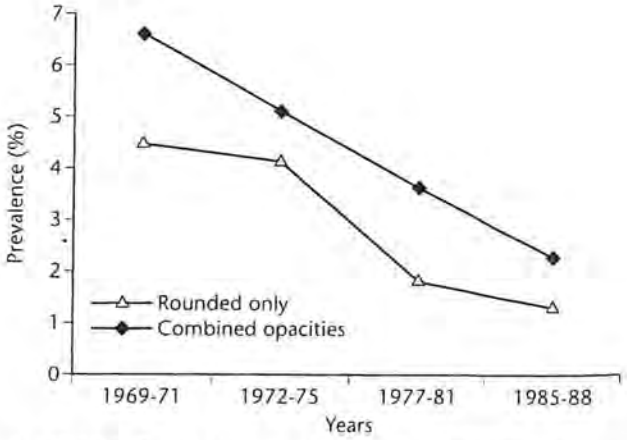


Figure 19.10.7: Trends in observed prevalence of radiographic category 1/0 or greater rounded only or combined opacities, adjusted to common job tenure. Based on the US National Study of Coal Workers' Pneumoconiosis. (Adapted from Attfield & Castellan[46] © 1992 American Public Health Association.)

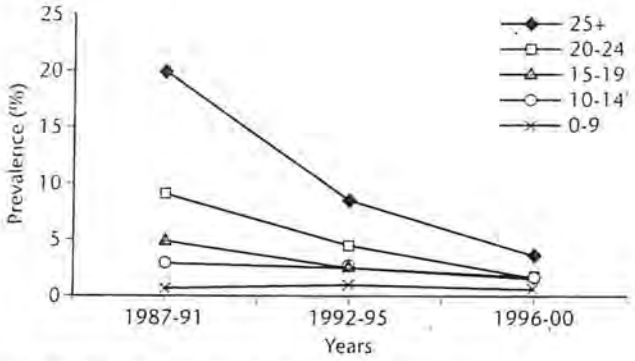


Figure 19.10.8: Recent trends in observed prevalence of radiographic category 1/0 or greater combined opacities by job tenure. Based on the US National Coal Workers' X-ray Surveillance Program. (Adapted from Wang ML, Petsonk EL: Pneumoconiosis prevalence among working US coal miners participating in federal chest x-ray surveillance programs: 1995 to 2000. Am J Epidemiol 153; 11:5127, by permission of Oxford University Press.)

radiograph classification system. When miners were grouped by mining tenure, and the proportion of miners with radiographic evidence of complicated CWP was analyzed, a clear decline was seen between the initial and final round of surveys. However, trends over the last decade were not as apparent.

In addition to the National Coal Study, an ongoing National Coal Workers' X-ray Surveillance Program is administered by NIOSH. Miners may participate in this program and receive chest radiographic examinations during the first 6 months and after 3 years of employment, and at approximately 3–5-year intervals thereafter. The classifications of radiographs taken under this program have been analyzed through 2000. The prevalence of category 1 or greater pneumoconiosis has generally declined for miners in each tenure category (Fig. 19.10.8). The proportion of participating miners showing complicated CWP also decreased since 1970 for all tenure groups. Thus, results of both the US National Coal Study and the US Coal Workers' X-ray Surveillance Program document a large reduction in the prevalence of radiographic changes of CWP in US coal miners since 1970.

Participation in the National Coal Study was over 90% in the first round, but was lower in later rounds. The US Coal Workers' X-ray Surveillance Program participation rates have been 50% or less. Although confidence in the analysis of trends is somewhat reduced by the participation rates, it does appear that the mandated programs of environmental and medical monitoring initiated under the Federal Coal Mine Health and Safety Act of 1969, in conjunction with transfer options and compensation of affected miners, have had an important effect in reducing the prevalence and incidence of CWP in US coal miners. Cases that continue to occur are more often noted in smaller mines and certain states.

Newer imaging techniques

In the past two decades, computerized radiographic techniques, gallium lung scanning, and magnetic resonance imaging have become widely available. Because traditional chest radiography is insensitive to early pathologic interstitial and emphysematous changes, interest has focused on these newer modalities in the non-occupational lung disorders. Limited evaluations of the usefulness of newer imaging techniques have been performed relative to the pneumoconioses. High-resolution computed chest tomography has been reported to be a more sensitive technique in identifying emphysema, as well as early pneumoconiosis among miners.[20] Coalescence of pneumoconiotic nodules and PMF lesions have been identified on CT scans when they were not apparent on routine radiographic evaluations.

Increased activity on gallium scanning of the lungs has been noted in several pneumoconioses, but this is nonspecific and may be less prominent in coal workers. Magnetic resonance imaging in the pneumoconioses has no currently demonstrated utility. Digital image processing has become widely used in the past few years for display, storage, and transmission of chest images. Computer-assisted pneumoconiosis interpretation of digitized images is being currently evaluated. However, at this time, additional studies are needed to define the role that digital processes should play in the evaluation of coal miners' lung diseases.

FUNCTIONAL CONSEQUENCES

Pathogenesis

The inhalation of sufficient coal mine dust affects the function as well as the structure of the lungs. Functional consequences can generally be divided into two categories: (1) effects related to the movement of gas into and out of the lungs (the 'bellows' function); and (2) effects related to the transfer of gases between the alveolar air and lung capillary blood.

Bellows function

Resistance to airflow into and out of the lungs of coal miners may be increased by bronchitic changes in the larger airways, by airway distortion, and by emphysematous destruction of elastic lung tissue at the level of the bronchioles and alveoli. These changes reduce the rate of maximal airflow, producing an obstructive ventilatory defect, demonstrated by reductions in the FEV_1% (FEV_1/FVC ratio) and the forced expiratory flow in the middle 50% of the vital capacity (FEF_{25-75}). In contrast, dilation of small air spaces can lead to overinflation and gas trapping, while fibrotic lesions, particularly of the massive type, reduce the volume of air contained in the lungs (total lung capacity).

Both of these latter changes can result in a reduction of the forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1), producing a restrictive or mixed restrictive and obstructive pattern of abnormality on spirometry. It can be seen that, based on the predominant pathology, miners may show obstructive, restrictive, or often mixed patterns of dysfunction on spirometry related to their dust exposure. Recent studies have

suggested that the loss of lung function due to dust exposure is not linear with time; rather, during the first months of exposure, there is an initial rapid loss of lung function, and subsequently a slower exposure-related decline.[27,28]

Gas exchange

Reduced ability to transfer gases in the lungs of miners can likewise result from multiple structural abnormalities. Dilation and distortion of small airways, as well as the fibrotic changes due to dusts, result in a heterogeneous delivery of inspired air into the alveoli. Destruction of capillaries and small vessels reduces the uniformity of lung perfusion. These effects combine to result in the mismatching of lung ventilation and perfusion and subsequent hypoxemia in the systemic arterial blood. The destructive loss of capillaries alone may also be sufficient to reduce the combined surface area of the alveolar-capillary membrane to an extent that gas transfer is impaired, particularly on exertion. In advanced disease, airflow obstruction may be so severe that alveolar ventilation is insufficient for metabolic demands. In this situation, hypoxia with hypercapnia results. An additional cause for hypoxia is reduced cardiac output, primarily seen in association with cor pulmonale and right ventricular dysfunction. Additionally, excessive ventilation on exertion has been associated with dyspnea in the presence of normal measures of lung mechanics.

INVESTIGATIONS OF COAL MINERS

The functional consequences of coal mine dust inhalation have been documented through both clinical and epidemiologic studies. Most studies of large groups of miners have used routine spirometry, evaluating the expiratory flows and forced vital capacity. Smaller groups have been studied utilizing other techniques. Series of miners have also been reported from clinics emphasizing disability/impairment evaluations. Although disability series are of interest, the representativeness of miners in these series is undetermined, exposure information is often scant or absent, and a control group is generally lacking. Their overall usefulness in the study of the effects of coal mine dusts on lung function is thus quite limited.

Epidemiologic studies

Most information regarding ventilatory effects of coal mining has been derived from large field studies of miners in the United States and the United Kingdom. Studies from other countries have been reported, and show generally very similar findings. Both cross-sectional and longitudinal evaluations of the US and British studies have been published. The lung function findings from both countries are quite consistent and are summarized below. The miners who were exposed to the dustiest environments show lower mean FEV_1 levels than those in less dusty mining jobs. More importantly, exposure–response relationships of FEV_1 with estimated dust exposure or with exposure surrogates such as tenure underground have been reported for miners in both countries. Other ventilatory function indices, such as FVC, FEV_1/FVC ratio, and flows at both higher and lower lung volumes have also been shown to be inversely related to estimated dust exposure.

Evidence of an effect on airflow at low lung volumes implies obstruction in small airways. In addition to the cumulative effects of dust, a large but non-progressive reduction in average lung function is seen in the group of miners who develop the symptom of a chronic productive cough. Groups of working miners from both the US and UK have been studied longitudinally over an approximately 11-year period. Results of these studies were quite similar, and showed that, as the miners' dust exposures increased, there was a resulting progressive decrement in lung function, as measured by the FEV_1, over the period of follow-up. Differences in the effects were seen between miners at different collieries. The effects on lung function associated with dust exposure were present after adjustment for smoking, and were typically observed to occur in all three smoking groups (never smokers, ex-smokers, and current smokers). The smoking and mine dust effects appeared to be independent and additive, but not synergistic. Thus, the average reduction in ventilatory function associated with exposure to mine dusts in smokers was similar to that in never-smoking miners. No disproportionate dust effects were noted in the smoking miners. In fact, the observed dust exposure effect, rather than being greater in current smokers, was often somewhat less.[29,30]

In miners who also smoke cigarettes, comparisons have been made between the excess reductions in lung function associated with mining exposures versus those associated with tobacco use. Since both effects are dose related, estimates of relative effect on lung function are conditional on the levels of exposure to tobacco smoke and to dust exposure chosen for the comparison. In the longitudinal study of US miners, for example, the average tobacco consumption in the smoking miners was 14 cigarettes per day. This was observed to result in an average excess loss of FEV_1 of 96 mL over 11 years in smoking miners compared to non-smoking miners. Eleven years of working at the coal face was associated with an average loss of FEV_1 of 84 mL in miners (non-smoking or smoking). For all miners, including those in less dusty work (average dust concentration 1.2 mg/m^3), the mean dust-related decline in FEV_1 over 11 years was 36 mL.

It has been suggested that although mean dust-related functional declines may be of similar magnitude to smoking effects, the tobacco and mining effects might be distributed differently. Under this hypothesis, tobacco effects among smokers are confined to large deficits in a small group of susceptibles, whereas mining effects are small but occur in most miners. Based on this hypothesis, equivalence in mean functional deficits attributed to dust and smoking could conceal very different functional consequences. However, several recent observations tend to contradict this hypothesis. Miners with non-specific airway hyper-responsiveness do show accelerated declines in lung function, but these miners do not appear to expe-

rience a disproportionate effect of dust exposure or smoking.[31] Miners with severe ventilatory deficits attributed to coal mine dust have also been identified, and studies of smoking and non-smoking miners have revealed similar increases in the proportions of severe impairments from dust- and smoking-related effects.[32,33]

Recent studies have demonstrated additional mine-related risk factors for lung function declines. Excess declines have been associated with exposures to explosive blasting and also to potentially contaminated water sprays used for dust suppression. Less steep rates of FEV_1 decline have been found among miners who use respiratory protective devices.[34] These findings suggest that additional health benefits can result from further improvements in the mine environment.

Miners' ventilatory lung function has also been compared to their chest radiographic changes of CWP, categorized using the ILO classification scheme. Higher categories of CWP, particularly complicated disease, are often associated with large reductions in FEV_1, and other functional consequences, as discussed below. Lower categories of simple CWP are not consistently associated with identifiable abnormalities on spirometry, after taking into account the functional losses associated with mine dust exposure, tobacco use, and the development of bronchitis. The relationship of the nodular lung disease of simple CWP and reduced lung function has repeatedly been investigated. Some studies observe a significant deficit in spirometry among miners who develop nodular simple CWP,[35] while others have not found an effect of nodular disease on lung function independent of the effects of emphysema.[36] Conversely, reduced lung function has repeatedly been observed among non-smoking miners without radiographic pneumoconiosis, and a negative x-ray does not exclude the possibility of occupationally related lung disease.[37,38]

Laboratory studies

Lung mechanics

Static lung compliance is often normal, but may be abnormally low or high in coal miners. High compliance, suggestive of emphysema, is the more common abnormality. Miners with PMF may have marked compensatory emphysema and high lung compliance; in others with PMF, the fibrotic process appears to predominate, and stiff lungs with low static compliance are seen. Miners with pinpoint ('p' type) opacities also appear to have dilation of peripheral airspaces.

Gas transfer

The transfer of gases in the lungs of coal miners has been evaluated using both the single-breath and the steady-state techniques to measure carbon monoxide diffusing capacity. Certain miners were found to have abnormalities on these tests. Reduced diffusing capacities are more commonly observed in miners with either pinpoint ('p' type) or irregular ('s' or 't' type) opacities of simple pneumoconiosis, and in those with complicated CWP, when compared to those with 'q' type opacities. Recent studies among Chinese coal miners have found simple CWP to be a significant contributor to decrements in pulmonary function, including diffusing capacity.[39]

The findings related to gas exchange on exercise in coal miners are less well defined. Many authors have reported on highly selected groups of miners referred for disability evaluation. Estimated dust exposures were often not determined. Diminished gas transfer and/or excessive ventilation on exercise have been found in some groups, but not in others. Miners with PMF often have shown severe gas exchange abnormalities on exercise. Those with increasing airflow obstruction also generally have corresponding defects in gas exchange on exercise. When the functional correlates of radiographic pneumoconiosis have been carefully evaluated in miners without important airflow obstruction, it appears that simple pneumoconiosis, particularly categories 2 and 3, can lead to identifiable abnormalities of gas exchange on exercise. These changes have been attributed to the emphysema that often accompanies the radiographic changes.

In summary, low categories of pneumoconiosis, particularly the 'q' type opacities, in the absence of airflow obstruction, often appear to be associated with little or no gas exchange impairment. In contrast, miners with a finding of airflow obstruction, higher category of CWP, and either irregular or pinpoint opacities may show impairment of gas exchange, particularly on exertion.

Pulmonary hemodynamics

Increases in pulmonary artery pressure, at rest and on exercise, have been documented in some coal miners. Abnormalities are more commonly seen when the miners have measurable airflow obstruction. When the hemodynamics were compared to radiographic findings, abnormalities were most commonly seen in miners with complicated pneumoconiosis, silicosis, or pinpoint ('p' type) opacities of simple CWP. In the absence of airflow obstruction or one of these radiographic features, pulmonary hypertension appears uncommon in coal miners.

Summary

Miners may show several functional abnormalities in relation to the inhalation of coal mine dusts. Radiographic changes in PMF are often associated with multiple abnormalities. Certain patterns of simple pneumoconiosis (irregular and rounded pinpoint opacities) appear to be associated with gas exchange impairment. Studies of miners vary regarding the relationship between ventilatory function and radiographic category of simple CWP. In contrast, deficits in expiratory flow and volumes on spirometry appear to be related to the intensity and duration of dust exposures, independent of radiographic category of simple CWP. An additional reduction in ventilatory function is often seen in miners with symptoms of bronchitis. Spirometric abnormalities measured during miners' lives are correlated with pathologic changes in emphysema, which in turn have been correlated with measures of both dust exposure and lung dust retention.

The extent of pathologic emphysema is strongly related to coal dust content of the lungs, as well as to age and smoking. The dust effects in miners who smoke cigarettes appear to be additive to the effects of tobacco use; no disproportionate effect has been identifiable among coal miners who smoke. In smoking miners, FEV_1 reductions associated with a year of working at the mine face are of similar magnitude to the average annual smoking effect. When work at less dusty jobs is included, the average dust-associated reduction in ventilatory function over one year appears to be smaller than the average effect of a year of smoking. In addition, evidence indicates that dust exposure leads to severe lung function changes in some miners.

MORTALITY

A number of studies of coal miners from the US, the UK, and the Netherlands have investigated mortality rates among miners compared to the general population. These studies have identified increased deaths from pneumoconiosis, bronchitis, and emphysema, as well as accidents, but reduced mortality from a number of causes, including lung cancer and ischemic heart disease. The deficits observed in cause-specific mortality rates have been attributed to the strict prohibition of smoking in underground coal mines and to the healthy worker effect. As mentioned above, several studies have observed an elevated risk among coal miners for death due to gastrointestinal cancer (possibly related to incidentally ingested carcinogenic aromatic hydrocarbons in coal).

When mortality has been studied in relation to dust exposures, increasing mine dust exposure has been associated with risk of death from all causes, pneumoconiosis, bronchitis, and emphysema, after accounting for smoking and age. Mortality rates are also increased with the presence of radiographic pneumoconiosis, particularly PMF.[23,40] Miners who have deficits in FEV_1 or who experience accelerated losses of ventilatory lung function have an increased risk of death from cardiovascular and non-malignant respiratory diseases. A study from the Netherlands found that, in addition to a high standardized mortality rate for coal workers' pneumoconiosis, Dutch miners had significantly increased mortality from chronic airflow obstruction, and this was particularly evident among those without pneumoconiosis. Spirometric impairment was predictive of COPD mortality.[41]

A recent study reported mortality and questionnaire follow-up for 634 US coal miners whose lung function had been monitored over an average of 11 years. When evaluated after an additional 10–18 years, the group of miners with excessive FEV_1 losses reported a greater onset of respiratory symptoms and illness, and had experienced increased mortality from cardiovascular and non-malignant respiratory diseases, compared to their colleagues with more stable lung function.[42] Overall, the findings from the mortality studies among coal miners emphasize the importance of early detection of both radiographic abnormalities and lung function declines, as well as the initiation of effective interventions to prevent disease progression and death in affected miners.

DIAGNOSIS

Coal workers' pneumoconiosis

Pathologic changes of CWP may be found in the absence of radiographic evidence of pneumoconiosis. However, the radiographic picture of coal workers' pneumoconiosis is often sufficiently distinctive that, in the presence of an adequate exposure history, a diagnosis can be made with reasonable certainty. A lung biopsy is rarely needed. The question of what constitutes an 'adequate exposure history' is determined by the timing, duration, intensity, and other characteristics of the reported occupational exposures. Underground work prior to the institution of federal dust control regulations in 1970–73 likely represents a high risk of exposure. In this setting, pneumoconiosis developed in some miners with less than 5 years of exposure. Several jobs, as discussed above, offer recognized risks for silicosis. Face work, particularly in longwall mining, is still occurring with exposures commonly over the 2 mg/m^3 respirable dust standard. In the absence of these higher risk settings, a careful workup should be performed before diagnosing radiographic CWP in a miner with less than 5–10 years of coal mine exposure. Stability of the radiograph, or slow progression over a period of 2–5 years, is expected.

More rapid change should also prompt a search for alternative processes. Hilar or mediastinal adenopathy and pleural effusion are also not likely to be due to CWP alone. For example, the appearance in a coal miner with 10 or more years of underground mining experience of bilateral small rounded radiographic opacities in low profusion and principally in the middle and upper lung zones should be accepted as diagnostic of CWP. Some miners with this presentation have complaints of cough and sputum. Basilar lung crackles are usually scant or absent. Infectious processes, other interstitial lung disorders, and metastatic neoplasm should be carefully considered if fever, weight loss, clubbing, chest pain, hemoptysis, or progressive dyspnea or malaise are present. A broad list of differential diagnoses should be considered if the initial radiograph shows unilateral disease, predominant irregular opacities, or a high profusion of small rounded densities. Finally, prominent gas exchange abnormalities are also unusual early in the course of simple CWP.

Of great diagnostic concern is the development of a large radiographic opacity in a coal miner. The differential diagnosis must include malignancies and mycobacterial or fungal infections, as well as complicated CWP. PMF, which typically develops in the upper lung zones on a background of simple nodular densities, is often bilateral. Stability or slow progression over several years is consistent with complicated CWP. If doubt exists, then an appropriate workup should be completed before accepting the diagnosis of a dust-induced lesion. Caplan's syndrome (rheumatoid pneumoconiosis) may present as multiple larger

nodules appearing without a definite background of simple CWP. The nodules may appear rapidly, i.e., over a period of weeks. A similar radiographic appearance may be seen with pulmonary metastases, but joint examination and serology will almost always reveal the confirming evidence of active rheumatoid disease.

MANAGEMENT

Medical screening and monitoring

Pre-placement medical testing is primarily intended to identify workers with existing medical conditions which would increase the health risk associated with coal mine employment. Periodic medical screening may utilize questionnaires, standard chest radiography, and spirometry. A program offering periodic chest radiographs to all underground coal miners in the US is currently mandated by federal regulations. This program serves to highlight potential areas of continuing medical and environmental problems. Due to the long latency of CWP, at times it can be difficult to relate contemporary environmental exposures to the cases identified. Miners with pneumoconiosis identified during screening qualify for administrative actions. They are entitled to increased frequency of personal environmental monitoring and offered the option to work in a reduced dust environment. This may require job transfer with maintenance of pay.

Periodic medical screening using spirometry is currently recommended, but not mandated. Pulmonary function testing is targeted toward identifying individuals with progressive airflow obstruction. To avoid the development of disabling lung impairment, workers with accelerated loss of lung function should be counseled regarding reducing current and future exposures to dust, if possible, as well as controlling other recognized risk factors (e.g., tobacco smoke).

General medical management

Medical management of a symptomatic coal miner with pneumoconiosis and/or airflow obstruction is similar in many ways to that of patients with non-occupational chronic lung diseases, but with emphasis on three principal areas: control of exposures, detection and treatment of complications, and compensation for disability. These are discussed below.

Exposure control

Control of symptoms in underground coal miners must address the mine environment, in addition to other potentially aggravating exposures, such as tobacco use. The miner should be encouraged to obtain and review the results of contemporary periodic air monitoring, to determine compliance of his or her work area with respirable dust and silica regulations.

A recommendation that the miner transfer to a surface job or a less dusty underground job is often the most viable option for reducing exposure to environmental dust. Miners, particularly those not close to retirement, who have identifiable airflow obstruction, accelerated longitudinal decline in lung function, or radiographic changes of pneumoconiosis should be counseled regarding transfer to a less dusty environment. Miners who develop symptomatic bronchospasm in dusty environments can at times suppress the symptoms through the use of inhaled medications and oral bronchodilators. However, transfer is often necessary in order to control symptoms without resorting to more hazardous forms of therapy, such as systemic corticosteroid therapy. Transfer rights with retention of wage rates and benefits are available only for underground coal miners with radiographic abnormalities identified through radiographic surveillance, under current US mining regulations. Radiographs may be submitted to NIOSH for an official interpretation to determine eligibility.

In contrast to the situation in the US, dust control is less well established in coal mines in other countries. For example, in Poland, recommended dust levels may be exceeded in over 90% of measurements.[43]

Engineering controls are accepted as the primary method of exposure control, and recommendations for continuous use of respiratory protective equipment are rarely practical in underground coal mining. Occasionally, however, a miner with prominent cough or bronchospasm may be able to continue working underground while participating in a formal respiratory protection program. There is evidence that use of respirators can diminish the adverse respiratory health effects from exposure to current levels of coal mine dust.[34]

Attention should also be paid to non-occupational respiratory hazards. In smoking miners, recommending smoking cessation is an initial step in exposure management. Referral to volunteer organizations or other formal cessation programs, prescription of nicotine delivery systems when appropriate, and unambiguous advice from the healthcare provider are effective approaches.

Detection and treatment of complications

Dust-induced fibrosis, emphysema, and chronic airflow obstruction are irreversible changes. Interventions should be targeted toward detecting, treating, and preventing complications. Management of conditions etiologically unrelated to mine dust exposure may also be complicated by lung impairments related to dust effects.

Periodic monitoring of spirometry and chest radiographs is useful to determine the rate of progression of the disorder and to detect onset of complications. An initial electrocardiogram may be helpful for subsequent comparisons. In miners who present with symptoms and signs of illness, assessment of gas exchange at rest and exercise may be indicated. The subsequent performance of these evaluations must be tailored to the clinical course of the individual miner. Infectious exacerbations, including acute bronchitis and pneumonia, may occur in affected miners. Broad-spectrum antibiotics and physical therapies are helpful in reducing symptoms during episodes of purulent sputum production.

The treating clinician should take care to exclude both tuberculosis and non-tuberculous mycobacterial infections. Particularly in miners with silicosis, the risk of these infections is increased and the response to chemotherapy is less satisfactory (see Chapter 19.9). Tuberculin reactivity should be tested in all miners with CWP, since over 10% may have silicosis, which confers a 30-fold increased risk for development of active tuberculosis. In tuberculin-positive miners with radiographic pneumoconiosis, active disease must be carefully excluded. Once this has been accomplished, isoniazid preventative therapy is recommended for those with positive skin tests and no contraindications or risk factors for isoniazid-resistant infection. Nine months of daily treatment reduces the subsequent risk of developing active disease. Shorter regimens of chemoprevention including rifampin have also been effective but are not considered the first choice for miners with abnormal chest radiographs and a low risk of INH-resistant strains.

Inhaled and/or oral bronchodilator therapy should be attempted in most symptomatic miners. Prolonged courses of inhaled steroids have been shown to control symptoms and reduce non-specific bronchial hyper-responsiveness. Short, tapering courses of systemic corticosteroids may be helpful for acute exacerbations. However, it is rarely, if ever, justified to use long-term systemic steroids to allow a miner to remain working in a dusty environment.

Whole lung lavage, reported for two miners with silica exposure and pneumoconiosis, resulted in considerable removal of inorganic dust from the lungs.[44] However, there is currently no evidence that the course of disease is improved by this drastic measure.

Cor pulmonale with right heart failure due to CWP is treated the same as cor pulmonale due to other etiologies.

A coordinated rehabilitation program, with attention to nutrition, emotional stresses, pharmacologic and physical therapies, and judicious exercise may improve the clinical status of the disabled miner. As in patients with other irreversible disorders, discussions regarding the use of life-support and mechanical ventilation are appropriate in miners with severe but clinically stable lung disease.

Compensation/disability

Miners with lung impairments may be eligible for compensation under the Federal Black Lung Benefits Program, as well as through state pneumoconiosis and workers' compensation plans. In advising individual miners, the healthcare provider should be familiar with the specific requirements of the programs. Particular note should be taken of the statute of limitations, since the date of filing a claim relative to the establishment of a diagnosis of work-related disease may be critical in determining eligibility for benefits. Legal counsel often is necessary in establishing eligibility (see Chapter 57.1).

References

1. US Department of Labor, Mine Safety and Health Administration. Mine Industry Accident, Injuries, Employment, and Production Statistics. http://www.msha.gov/ACCINJ/accinj.htm [cited October 1, 2001]
2. Love RG, Miller BG, Groat SK, et al. Respiratory health effects of opencast coalmining: a cross sectional study of current workers. Occup Environ Med 1997; 54:416–23.
3. Attfield MD, Wagner GR. Chronic occupational respiratory disease. Occup Med 1996; 11:451–65.
4. Fernie JM, Ruckley VA. Coalworkers' pneumoconiosis: correlation between opacity profusion and number and type of dust lesions with special reference to opacity type. Br J Ind Med 1987; 44:273–7.
5. Vallyathan V, Brower PS, Green FH, et al. Radiographic and pathologic correlation of coal workers' pneumoconiosis. Am J Respir Crit Care Med 1996; 154:741–8.
6. Anonymous. Pathology standards for coal workers' pneumoconiosis. Report of the pneumoconiosis committee of the College of American Pathologists to the National Institute for Occupational Safety and Health. Arch Path Lab Med 1979; 103:375–432.
7. Ruckley VA, Gauld SJ, Chapman JS, et al. Emphysema and dust exposure in a group of coal workers. Am Rev Respir Dis 1984; 129:528–32.
8. Ruckley VA, Fernie JM, Chapman JS, et al. Comparison of radiographic appearances with associated pathology and lung dust content in a group of coal workers. Br J Ind Med 1984; 41:459–67.
9. Maclaren WM, Hurley JF, Collins HP, et al. Factors associated with the development of progressive massive fibrosis in British coal miners: a case-control study. Br J Ind Med 1989; 46:597–607.
10. Green FYH, Althouse R, Weber K. Prevalence of silicosis at death in underground coal miners. Am J Ind Med 1989; 16:605–15.
11. Leigh J, Driscoll TR, Cole BD, et al. Quantitative relation between emphysema and lung mineral content in coal workers. Occup Environ Med 1994; 51:400–7.
12. Coggon D, Inskip H, Winter P, et al. Contrasting geographical distribution of mortality from pneumoconiosis and chronic bronchitis and emphysema in British coal miners. Occup Environ Med 1995; 52:554–5.
13. Yi Q, Zhang Z. The survival analyses of 2738 patients with simple pneumoconiosis. Occup Environ Med 1996; 53:129–35.
14. IARC Working Group on the evaluation of carcinogenic risks to humans. Silica, some silicates, coal dust and para-aramid fibrils. Lyon, October 15–22, 1996. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. IARC: Lyon, 1997.
15. Swaen GM, Meijer JM, Slangen JJ. Risk of gastric cancer in pneumoconiotic coal miners and the effect of respiratory impairment. Occup Environ Med 1995; 52:606–10.
16. Oberdorster G. Lung particle overload: implications for occupational exposures to particles. Regul Tox Pharm 1995; 21:123–35.
17. Schins RP, Borm PJ. Mechanisms and mediators in coal dust induced toxicity: a review. Ann Occup Hyg 1999; 43:7–33.
18. Rom WN. Relationship of inflammatory cell cytokines to disease severity in individuals with occupational inorganic dust exposure. Am J Ind Med 1991; 19:15–27.
19. Miller BG, Hagen S, Love RG, et al. Risks of silicosis in coal workers exposed to unusual concentrations of respirable quartz. Occup Environ Med 1998; 55:52–8.
20. Gautrin D, Auburtin G, Alluin F, et al. Recognition and progression of coal workers' pneumoconiosis in the collieries of northern France. Exp Lung Res 1994; 20:395–410.
21. Goodwin S, Attfield M. Temporal trends in coal workers' pneumoconiosis prevalence. Validating the National Coal Study results. J Occup Environ Med 1998; 40:1065–71.
22. Henneberger PK, Attfield MD. Respiratory symptoms and spirometry in experienced coal miners: effects of both distant and recent coal mine dust exposures. Am J Ind Med 1997; 32:268–74.
23. Kuempel ED, Stayner LT, Attfield MD, et al. Exposure–response analysis of mortality among coal miners in the United States. Am J Ind Med 1995; 28:167–84.

24. Bang KM, Althouse RB, Kim JH, et al. Recent trends of age-specific pneumoconiosis mortality rates in the United States, 1985–1996: coal workers' pneumoconiosis, asbestosis, and silicosis. Int J Occup Environ Hlth 1999; 5:251–5.
25. National Institute for Occupational Safety and Health. Criteria for a recommended standard: occupational exposure to respirable coal mine dust. 1-9-95. DHHS (NIOSH) Publication No. 95-106. Cincinnati: US Department of Health and Human Services, Public Health Service, Centers for Disease Control.
26. Lamers RJ, Schins RP, Wouters EF, et al. High-resolution computed tomography of the lungs in coal miners with a normal chest radiograph. Exp Lung Res 1994; 20:411–9.
27. Seixas NS, Robins TG, Attfield MD, et al. Longitudinal and cross sectional analyses of exposure to coal mine dust and pulmonary function in new miners. Br J Ind Med 1993; 50:929–37.
28. Henneberger PK, Attfield MD. Coal mine dust exposure and spirometry in experienced miners. Am J Respir Crit Care Med 1996; 153:1560–6.
29. Love RG, Miller BG. Longitudinal study of lung function in coal miners. Thorax 1982; 37:193–7.
30. Attfield MD. Longitudinal decline in FEV1 in United States coal miners. Thorax 1985; 40:132–7.
31. Hodgins P, Henneberger PK, Wang ML, et al. Bronchial responsiveness and five-year FEV1 decline: a study in miners and non-miners. Am J Respir Crit Care Med 1998; 157:1390–6.
32. Marine WM, Gurr D, Jacobsen M. Clinically important respiratory effects of dust exposure and smoking in British coal miners. Am Rev Respir Dis 1988; 137:106–12.
33. Hurley JF, Soutar CA. Can exposure to coal mine dust cause a severe impairment of lung function? Br J Ind Med 1986; 43:150–7.
34. Wang ML, Petsonk EL, Beeckman LA, et al. Clinically important FEV1 declines among coal miners: an exploration of previously unrecognized determinants. Occup Environ Med 1999; 56:837–44.
35. Bourgkard E, Bernadac P, Chau N, et al. Can the evolution to pneumoconiosis be suspected in coal miners? A longitudinal study. Am J Respir Crit Care Med 1998; 158:504–9.
36. Gevenois PA, Sergent G, De Maertelaer V, et al. Micronodules and emphysema in coal mine dust or silica exposure: relation with lung function. Eur Respir J 1998; 12:1020–4.
37. Nemery B, Veriter C, Brasseur L, et al. Impairment of ventilatory function and pulmonary gas exchange in non-smoking coal miners. Lancet 1987; 2:1427–30.
38. Wang X, Yu IT, Wong TW, et al. Respiratory symptoms and pulmonary function in coal miners: looking into the effects of simple pneumoconiosis. Am J Ind Med 1999; 35:124–31.
39. Wang X, Yano E, Nonaka K, et al. Respiratory impairments due to dust exposure: a comparative study among workers exposed to silica, asbestos, and coal mine dust. Am J Ind Med 1997; 31:495–502.
40. Miller BG, Jacobsen M. Dust exposure, pneumoconiosis, and mortality of coal miners. Br J Ind Med 1985; 42:723–33.
41. Meijers JM, Swaen GM, Slangen JJ. Mortality of Dutch coal miners in relation to pneumoconiosis, chronic obstructive pulmonary disease, and lung function. Occup Environ Med 1997; 54:708–13.
42. Beeckman LA, Wang ML, Petsonk EL, et al. Accelerated declines in FEV1 and subsequent increased respiratory symptoms, illnesses, and mortality in US coal miners. Am J Respir Crit Care Med 2001; 163:633–9.
43. Marek K, Lebecki K. Occurrence and prevention of coal miners' pneumoconiosis in Poland. Am J Ind Med 1999; 36:610–7.
44. Wilt JL, Banks DE, Weissman DN, et al. Reduction of lung dust burden in pneumoconiosis by whole-lung lavage. J Occup Environ Med 1996; 38:619–24.
45. Attfield MD. British data on coal miners' pneumoconiosis and relevance to US conditions. Am J Pub Hlth 1992; 82:978–83.
46. Attfield MD, Castellan RM. Epidemiological data on US coal miners' pneumoconiosis, 1960 to 1988. Am J Pub Hlth 1992; 82:964–70.
47. Pon MRL, Roper RA, Petsonk EL, et al. Pneumoconiosis prevalence among working US coal miners participating in federal chest x-ray surveillance programs: 1996 to 2002. MMWR 2003; 52:336–400.