CHAPTER 5

Occupational Lung Disease

This chapter will assist readers to understand and manage occupational lung disease risks in the South African mining industry. The most important diseases are described, with an emphasis on clinical features and on the association between exposure and occurrence of disease. Where possible this is based on research conducted in South Africa. The relevant legal framework in terms of the Mine Health and Safety Act (MHSA) and the Occupational Diseases in Mines and Works Act (ODMWA) is reviewed. Medical surveillance is discussed in detail. The use of questionnaires, chest x-rays, spirometry and other investigations in screening for occupational disease is detailed, as well as how to manage the resulting information.

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Glossary

Asbestosis: Fibrosis of the lungs due to inhalation of asbestos dust

COPD: Chronic obstructive pulmonary disease. (Alternatively, COAD: Chronic obstructive airways disease). Condition characterised by persistent obstruction of airflow through the lungs

CWP: Coal worker's pneumoconiosis. Fibrosis of the lungs due to inhalation of coal and silica dust in coalmining work

Dose response relationship: Association between increasing amount of exposure and likelihood of disease

Fibrosis: Formation of scar tissue

ILO Classification: International Labour Organisation Classification of Radiographs of the Pneumoconioses. Standardised system for describing pneumoconiosis radiologically

Inflammation: Localised or widespread reaction of the body's defences to injury, infection, foreign substances or sometimes unknown triggers in the body

Mesothelioma: Cancer of the lining (pleura) of the lung or abdomen

Occupational asthma: Asthma caused by substances primarily encountered at work

PMF: Progressive massive fibrosis. Complication of silicosis characterised by appearance of large fibrotic masses in lung

Pneumoconiosis: Fibrosis of the lungs due to inhalation of mineral dust

Progressive system sclerosis: Disease characterised by thickening of the tissues under the skin and fibrosis of the lungs

SAMODD: South African Mining Occupational Diseases Database (maintained by Department of Minerals and Energy)

Silicosis: Fibrosis of the lungs due to inhalation of silica dust

Silicotuberculosis: Combination of silicosis and tuberculosis of the lungs

Spirometry: Test of lung function that measures amount and force of air breathed in and out

5.1 Introduction

Occupational lung diseases are a major preventable cause of premature retirement and death among people working in the South African mining industry. The Mine Health and Safety Act (MHSA) requires employers to take measures to assess and reduce the risk of these diseases.

Anyone involved in risk assessment needs to have an understanding of the epidemiology of the common occupational lung diseases suffered by miners. Medical and nursing practitioners in the industry need to be aware of the clinical features of occupational lung disease for purposes of setting up medical surveillance programmes, diagnosis, treatment and appropriate referral for compensation.

5.1.1 Definition of occupational lung disease

The MHSA defines an occupational disease as any condition listed in either the Occupational Diseases in Mines and Works Act (ODMWA) or the Compensation for Occupational Injuries and Diseases Act (COIDA).

Table 5.1 Mining related lung diseases listed for compensation purposes in South Africa

Occupational Diseases in Mines and Works Act as Amended, Act 208 of 1993
• Silicosis in miners and surface workers exposed to silica dust • Silicotuberculosis in miners and surface workers • Coal workers' pneumoconiosis in coal miners • Obstructive airways disease in miners • Tuberculosis, compensable only if cardio-pulmonary organs involved, in miners or those exposed to dust on surface • Progressive systemic sclerosis or scleroderma, in miners exposed to silica dust
Section 1 (e) of the Act includes any other disease of the cardio-pulmonary organs which experts consider attributable to risk work. In practice, this section covers:
• Occupational asthma in platinum salt workers ("platinosis") • Asbestosis (interstitial lung disease) in asbestos miners • Malignant mesothelioma in asbestos miners • Pleural plaques in asbestos miners • Asbestos-related lung cancer in asbestos miners • Bronchiolitis obliterans due to nitrous fumes in mine workers • Hard metal pneumoconiosis, usually in drill shop workers. Stannosis in tin miners
Compensation for Occupational Injuries and Diseases Act, Act 130 of 1993 (Third Schedule)
Of particular relevance to mining, but not included in the ODMWA list are:
• Asphyxiation due to carbon monoxide, hydrogen cyanide fumes or its derivatives, and hydrogen sulphide fumes • Occupational asthma due to nickel, cobalt, vanadium, chromium salts, soldering or welding fumes, isocyanates, formaldehyde, anhydrides, amines and diamines, hardening agents including epoxy resins

5.1.2 Importance of occupational lung disease in the South African mining industry

Dust-related lung diseases overshadow mine accidents in numbers of workers affected. Among the 429 000 persons at work in all mines in 1998 there were 371 reported fatalities (0.85 per 1 000) and 6 064 reported injuries (14.1 per 1 000). By comparison there were 5 603 new or upgraded certifications for pneumoconiosis with or without tuberculosis and over 5 000 new cases of tuberculosis (> 20 per 1 000).

Occupational lung disease may result in illness, premature retirement because of disability, or death. There are significant costs involved, experienced by individuals in loss of income and medical or related expenses, and by mining companies through the loss of experienced employees and the expense of recruiting and training new employees, direct medical expenses and compensation levies.

The total direct costs of occupational lung disease in the gold mining industry were estimated in 1996 as R343 million. These costs included those for compensation, medical surveillance and treatment, and losses of output, the single largest direct cost. Included also are employers' contributions to the compensation fund under ODMWA, which amounted to R38 million. The total direct and indirect costs of occupational lung disease to the economy is cumulative and difficult to estimate, but could be about 3 percent of the gold mining industry's annual contribution to the gross domestic product, or approximately R558 million.

Indications are that rates of occupational lung disease have risen to the same high levels that were experienced during the early part of the 20th century. There appear to have been only insignificant reductions in dust exposures, particularly in gold mining, in the second half of the 20th century.

In addition, the mine labour force has contracted and stabilised over the last twenty years. This change in employment patterns has aggravated the problem of occupational disease by increasing the average period of dust exposure of underground miners.

It is vital that the mining industry come to grips with the problem of mining related lung diseases by seeking to prevent them and not merely to compensate established disease.

5.2 The occupational lung diseases

In this section, the characteristics of the more important mining related occupational lung diseases are described. The diseases are grouped into diseases due to mineral dusts, chronic obstructive pulmonary disease (COPD), occupational cancers, occupational asthma, and inhalation injuries including those due to nitrogen dioxide and fire.

5.2.1 Mineral dust disease (pneumoconiosis)

The mineral dust diseases share the common feature that they are caused by dust particles that are small enough to reach the alveoli or gas exchanging part of the lung. Such dust particles are termed respirable dust.

The relationship between particle size ("aerodynamic diameter") and the ability of particles to penetrate the lungs is illustrated in Figure 5.1.

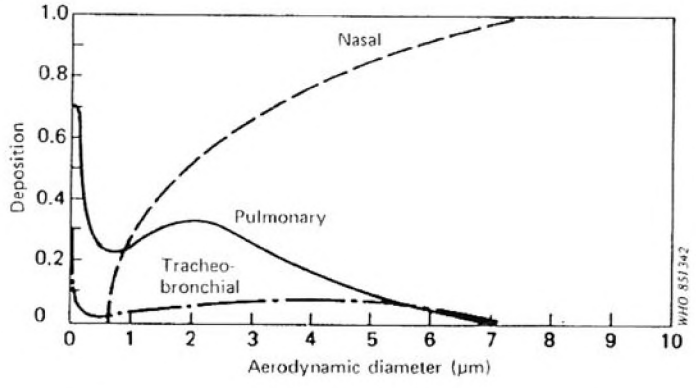

Figure 5.1 Deposition in the respiratory system as a function of particle size
(Reprinted from World Health Organisation. Technical Report 743, 1986, p 17)

The nasal passages are the first line of defence and will trap all particles with an aerodynamic diameter > 7 microns. The nose is less effective at preventing particles < 7 microns entering the lungs

and below this limit particles are defined as respirable. The lung's defences can still remove particles reaching the airway walls in the tracheobronchial tree. However, approximately 30 percent of inspired particles in the range 1-3 microns will be deposited in the lung tissue (Figure 5.1).

Dust particles in the respirable range that are deposited in the tiny airways deep in the lung will be engulfed by alveolar macrophages (defensive cells in the lung) and carried across the respiratory epithelium into the lung tissue itself. Crystalline silica (usually quartz) particles, especially when newly generated, are toxic to macrophages and result in cell death. A similar process occurs with coal and other mineral dusts. Death of the macrophage results in release of the dust particles and attracts other macrophages that engulf the particles again. This recurring cycle of defensive cell death results in a low-grade inflammatory process in the lung. Gradually the dust particles are sealed off in areas of fibrotic (scar) tissue that replaces normal lung tissue. This scar tissue is in the form of rounded nodules in the cases of silicosis and coal workers' pneumoconiosis, whereas in asbestosis or hard metal pneumoconiosis the scars are elongated or linear.

The chest x-ray appearance of the pneumoconioses is by convention described using the International Labour Organisation's (ILO) classification system. This system is based on comparison with a set of standard radiographs. The abnormalities in the lung fields of a chest x-ray are classified according to the type of abnormality seen (nodular or linear) and the optical density or profusion of the abnormalities (as an index of severity). The profusion is graded from 0 (no abnormality) to 3 (high profusion) with a series of intermediate grades.

5.2.2 Silicosis

Silicosis is the most important occupational lung disease in gold mining where almost all of the 183 000 people employed underground in 1998 were exposed to silica dust. The features of silicosis are well reviewed in the medical literature. (See Guide to Resources).

The dangers of silicosis from the inhalation of silica dust produced by drilling, blasting, scraping and other mining operations, have been recognised since the earliest days of gold mining operations in South Africa. The risk of silicosis occurs in all types of hard rock mining, tunnelling, quarrying and crushing where crystalline silica particles are liberated. Quartz is a pure form of silica crystal. Newly fractured quartz is the most toxic variety of crystalline silica.

Silica contained in an amorphous (i.e. non-crystalline) form appears to be less toxic than the crystalline variety, although amorphous silicas can cause silicosis. Amorphous silicas include diatomaceous earth, colloidal silica, fused silica, fumed silica and thermally generated silica fume. Although there is a tendency to treat all amorphous silicas as a group, there is evidence that their toxicities do vary, depending on the particle size and content of crystalline silica. In risk assessment where there is exposure to amorphous silica the health hazard associated with that type of amorphous silica should be considered.

Clinical features of silicosis

The clinical features of simple and complicated silicosis are described in Table 5.2.

Table 5.2 Usual clinical features of simple silicosis and complicated silicosis

Simple silicosis
• Simple (chronic) silicosis is the most common manifestation of silica dust exposure • Simple silicosis develops after 10-15 years of dust exposure • In simple silicosis nodules 1 – 3.5 mm in size become evident on the chest x-ray, usually in the upper zones of the lung • As the condition progresses the nodules become more numerous and may become larger, 3.5 — 10 mm. Initially this process causes relatively minor impairment of lung function • Silicosis is often slowly progressive over time, even in the absence of further dust exposure • With increasing lung dust burden the degree of functional impairment increases. Either obstructive or restrictive abnormality of lung function may result • With increasing lung dust burden there is an increase in risk of pulmonary tuberculosis (TB). The risk of TB is highest with established silicosis

Complicated silicosis (progressive massive fibrosis)
• In approximately 5% of cases of simple silicosis the nodules in the lung coalesce into larger nodules > 1 cm in size. This is known as complicated silicosis or progressive massive fibrosis (PMF) • The replacement of normal lung tissue by scar tissue with resulting distortion of lung tissue in complicated silicosis / PMF can produce significant lung function impairment and reduced life expectancy • The known determinants of progression of simple silicosis to PMF are: high cumulative dust exposure; high ILO profusion category of simple pneumoconiosis; young age of onset of pneumoconiosis; continued dust exposure in the presence of simple pneumoconiosis

Epidemiology of silicosis

The relationship between silica dust exposure and the occurrence of silicosis in South African gold mines was first investigated by Beadle in the 1960s, and later by Hnizdo and Sluis-Cremer on a cohort of white miners. As yet there are no published studies of dose-response relationships of silica exposure among black mineworkers in the South African gold mining industry.

Beadle believed that there was little evidence of any improvement in dust exposure levels in the years between 1938 and 1969. This finding still has relevance. In 1995 the Leon Commission concluded that there had been little significant change in dust exposure in South African gold mines for fifty years. In 1999 the National Centre for Occupational Health published data based on 26 000 underground dust measurements in 48 South African gold mines from 1995 to 1997. Only 8 (17%) of these mines had all of their estimated time weighted average (TWA) measurements for respirable quartz below the DME's standard of 0.1 mg/cubic metre. Of the remainder 21 mines (43%) had a high proportion of dust measurements in the range 0.1 — 0.4 mg/cubic metre, whilst 19 (40%) had most of their measurements above 0.4 mg/cubic metre.

Risk assessment requires an understanding of cumulative exposure response curves. These curves illustrate what proportion of mineworkers exposed to a given cumulative dose of silica (i.e. the sum of all past exposures) will develop silicosis at some stage in their lives.

The exposure-response relationships from four studies of silicosis among miners are shown in Figure 5.2 (derived from Chen 2000). Hnizdo and Sluis-Cremer's study of white mineworkers is indicated by squares. For example, if this working population is exposed to 0.1 mg/cubic meter respirable crystalline silica for 20 years, their cumulative exposure is 2 mg/cubic metre-years (0.1 times 20). It can be inferred from Figure 5.2 that less than 10 percent of such a population will ultimately develop radiological silicosis. If this group of workers spent the same time (20 years) working at 0.2 mg/cubic metre (cumulative exposure of 4.0 mg/cubic metre-years) it can be expected that over 50% will ultimately develop silicosis.

Black workers comprise the vast majority of the mine labour force and do the jobs such as drilling or ore removal in the dustiest areas of gold mines such as stoping and development. Available information from recent studies indicates that black mineworkers have a working lifetime risk of between 220/1 000 and 360/1 000 of developing radiological silicosis from gold mining. These estimates, based on retired or retrenched men, are much higher than estimates of the prevalence of silicosis in active black gold mineworkers.

Silicosis is a condition that develops slowly over time. As a group, active miners include members with a mix of various lengths of service. The men with shorter length of service (e.g. < 10 years) are unlikely to have detectable silicosis, even if they are working in dust levels that are high enough to ultimately cause the condition. Silicosis continues to progress slowly with continued mining exposure. Cowie found that silicosis among Free State mineworkers, once established, progressed by approximately one ILO classification subcategory (e.g. 2/1 to 2/2) over a 4-5 year period. Even once exposure ceases, dust particles retained within the lung continue to be biologically active and the condition continues to develop.

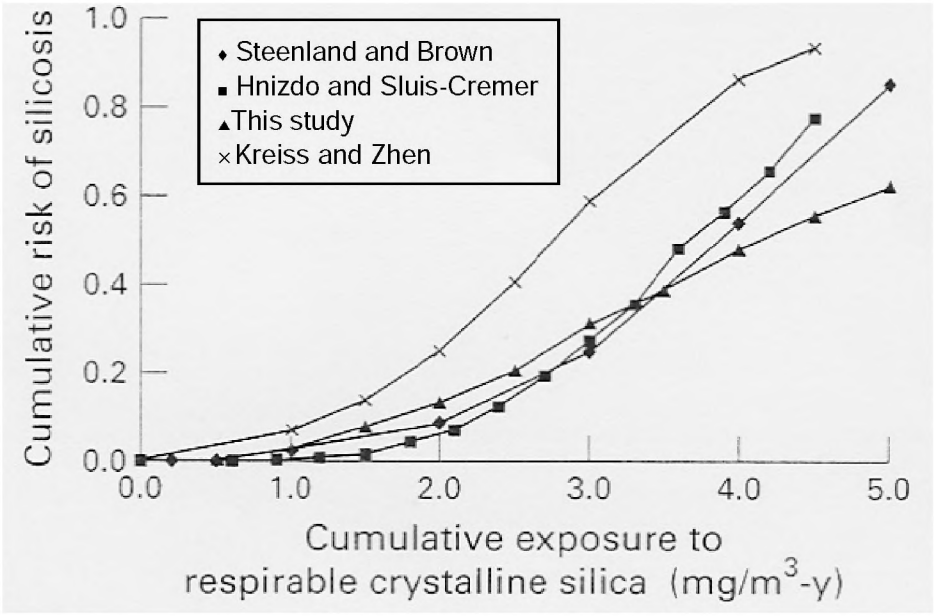

Figure 5.2 Cumulative risk of silicosis versus cumulative exposure to respirable crystalline silica

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The different estimates of the prevalence of silicosis in the active and retired workforce can probably all be reconciled on this basis. This natural history of silicosis has two major implications. First, the risk of silicosis can only be accurately measured by long-term follow up of cohorts of mineworker, including ex-mineworkers. Second, estimates of Occupational Exposure Limits (OEL) for silica dust exposure, as with other exposures, are usually based on studies of active workers because this is the easiest group to study. Where studies of older and retired workers have been conducted, such as those included in Figure 5.2, they indicate that there is still a risk of pneumoconiosis from long working lifetime exposures below the DME OEL of 0.1 mg/cubic metre for respirable quartz.

Certification rates of compensable pneumoconiosis are also substantially lower than the actual rates of occurrence of pneumoconiosis (>220/1 000) quoted for black workers above. There are a number of reasons for this. Firstly, epidemiological studies use sensitive radiological definitions of the presence of abnormalities consistent with pneumoconiosis. By contrast, certification of compensable pneumoconiosis, as detailed in Chapter 14, requires a more serious degree of abnormality consistent with at least a 10 percent impairment of cardio-respiratory function. The findings of two recent studies of retired black mineworkers confirm that compensation statistics substantially underestimate the occurrence of pneumoconiosis. These studies both showed that there are substantial numbers of men whose disease has either progressed since leaving the industry, or who were not referred to the Medical Bureau for Occupational Diseases (MBOD) for a benefit examination, as was their right, at the time of their exit from the industry.

Prognosis of silicosis

Mine medical practitioners typically diagnose simple silicosis early in the natural history of the condition in active miners. Unless the condition is already complicated by tuberculosis, there is seldom any significant observable impairment of lung function at that stage since simple silicosis is only a slowly progressive condition. This observation could give rise to an erroneous view that simple silicosis does not cause much abnormality and does not influence life expectancy.

A progressive reduction of vital capacity of the lungs with progression of x-ray changes of silicosis has been shown by a number of studies. Even ILO category 1 pneumoconiosis is associated with some loss of lung function. A recent estimate of the influence of silicosis on annual loss of FEV1 showed the following annual average losses:

- No silicosis: 37 ml/year
- ILO category 1: 57 ml/year
- ILO category 2: 100 ml/year
- ILO category 3: 128 ml/year

Although there is evidence that simple silicosis without measurable lung function abnormality does not affect life expectancy in North America, there are no comparable data for South Africa. Complicated silicosis can be expected to reduce life expectancy. The commonest complication of silicosis in South Africa is pulmonary tuberculosis, which has a significant adverse effect on life expectancy.

5.2.3 Silicotuberculosis

Both silica exposure and silicosis are risk factors for tuberculosis (TB). Tuberculosis in a person with established silicosis is termed silicotuberculosis. The risk of developing TB increases with duration of exposure to silica dust even in the absence of silicosis. The presence of radiological silicosis increases the risk of pulmonary TB approximately four fold, with the risk rising as radiological silicosis becomes more severe. This increased risk of TB associated with silicosis is lifelong, continuing after silica exposure ceases. This relationship is discussed further in Chapter 6.

The presence of silicosis in the lungs can modify the natural history of TB and may alter its radiological appearances. The interaction of TB and silicosis is very damaging to the lung, unless the TB is diagnosed and treated early. With each episode of TB lung function abnormality increases, as it does with each category of ILO category of profusion of silicosis.

Diagnosis of TB in the presence of silicosis should be according to standard guidelines for TB (see Chapter 6). However, it is well recognized that among the clinical variants of silicotuberculosis are fibrotic and initially slowly progressive cases where sputum examination is persistently negative. Silicotuberculosis may also be difficult to distinguish from PMF. Radiological suspicion of pulmonary TB in the presence of silicosis is greatly aided by comparisons with previous films, and should be prompted by the:

- Rapid appearance of new infiltrates, especially in the upper third of the lung fields
- Coalescence of nodules, especially in the upper third of the lung fields
- Appearance of a cavity, especially with an irregularly shaped inner wall, within a conglomeration of nodules
- Appearance of any > 1 cm, non-retractile opacity limited by a fissure
- Development of pericardial or pleural effusion
- Development of segmental or lobar collapse secondary to bronchial stenosis or occlusion

Treatment of silicotuberculosis is discussed in Chapter 6.

5.2.4 Coal workers' pneumoconiosis (CWP)

Coal mining in South Africa employed in excess of 57 000 people in 1998. Most coal mining is either surface mining or occurs at comparatively shallow depths, not exceeding about 300 m. The output is predominantly bituminous coal although a small amount of anthracite coal is also mined. The silica content of South African coal is generally fairly low. Mpumalanga, Gauteng and Free State coal has a quartz content of about 2 percent, whereas that of Natal contains 3 percent quartz.

Coal miners are at risk of coal workers' pneumoconiosis (CWP). Given the size and economic importance of coal mining in South Africa, there is surprisingly little information available on CWP.

Clinical features of CWP

The clinical features of CWP are described in table 5.3.

Table 5.3: Usual clinical features of Coal Workers' Pneumoconiosis (CWP)

- CWP develops after 10-15 years of coal mine dust exposure
- In CWP nodules < 1 mm in size become evident on the chest x-ray, usually in the upper zones of the lung
- As the condition progresses the nodules become more numerous
- CWP is frequently complicated by chronic obstructive pulmonary disease (COPD)
- CWP is usually slowly progressive over time
- The features of CWP resemble silicosis if there is appreciable silica content in the coalmine dust
- CWP differs from other pneumoconioses in that even with large dust burdens, provided that the silica content is low, fibrosis or scarring in the lungs is less severe
- With higher silica content of coal mine dust, high lung dust burden and/or infection with tuberculosis, CWP may evolve into a progressive massive fibrosis (PMF) where smaller nodules have coalesced into large nodules > 1 cm

Epidemiology of CWP

Some information relating to certification of CWP among coal miners is available from MBOD reports. Prior to 1980, certifications of pneumoconiosis (when expressed as a proxy rate of living cases certified per 1 000 miners currently employed) tended to be higher in coalmines than in gold mines (approx. 6 per 1 000 in 1980 vs 2 per 1 000 in gold mining). CWP certification rates declined in the years 1980 to 1989 although in 1989 the rate was still over 4 per 1 000, a similar rate to the silicosis rate in gold mines. The MBOD Director's Report 1998/99 suggests a continuing decline in certifications of CWP. In that report 0.6% of all first degree pneumoconiosis certifications among miners (25 cases or < 0.5 per 1 000 currently employed miners) were for CWP among coal miners. There were no second-degree certifications apart from 6 cases of CWP and TB combined.

A recent unpublished study documented radiological abnormalities among KwaZulu-Natal anthracite miners. A total of 187 employees with more than five years exposure at a mine were included. 15.8% were thought to have CWP ILO grade 1/0 or higher, including 7.6% of grade 1/1 or greater and 1.1% (2 cases) grade 2/1 or greater. There was a positive association between presence of pneumoconiosis and length of service in a relatively young workforce (mean age 40.3 years). Findings of past or present TB were more common in those workers with CWP than those without.

A relatively low prevalence of CWP was found in another unpublished study of active and ex-coalminers from three South African mines (SIMHEALTH 607 — see SIMRAC website). In the latter study, the prevalence of pneumoconiosis was 1.8 to 4.2% depending on the chest x-ray reader. The prevalence of CWP increased with cumulative exposure to coal dust. As part of the same study autopsy findings among former miners who had had only coal exposure were examined. CWP (prevalence: 7.3%) and silicosis (prevalence: 10.8%) were detectable at autopsy, with both conditions showing strong associations with increasing years of coal exposure.

As with silicosis, it appears that MBOD certification rates underestimate the occurrence of CWP. The limited available information makes it difficult to be certain about the actual degree of underestimation, but it does seem plausible that rates of CWP have declined in the last 20 years.

The cause of this decline is speculative in the absence of trend data on coal dust exposures in South Africa. Unlike gold mining, which remains a largely labour intensive process, coal mining has become increasingly mechanised in the second half of the 20th century. On the one hand, mechanised mining greatly increases the potential for dust exposure in coal mining. On the other hand, mechanised mining usually also means that fewer miners are exposed to these higher dust levels.

Exposure-response relationships for CWP and PMF have been well documented in other coal mining regions of the world. The ability of coal dust to cause disease is generally highest for anthracite and

higher rank coals, i.e. those that have been formed under conditions of higher temperature and pressure and have higher carbon content. These dose response relationships may or may not be applicable to coal mining in South Africa, given that most South African coal is bituminous or low rank.

The DME's current OEL for coalmine dust is 2 mg/cubic metre. As may be seen from the illustrative dose response curves based on US coal miners in Figure 5.3, after 40 years of work in coal mining at a respirable coal mine dust concentration of 2 mg/cubic metre, miners have about a 12% probability of developing ILO category 1 pneumoconiosis or greater. About 2% will develop PMF working at this concentration.

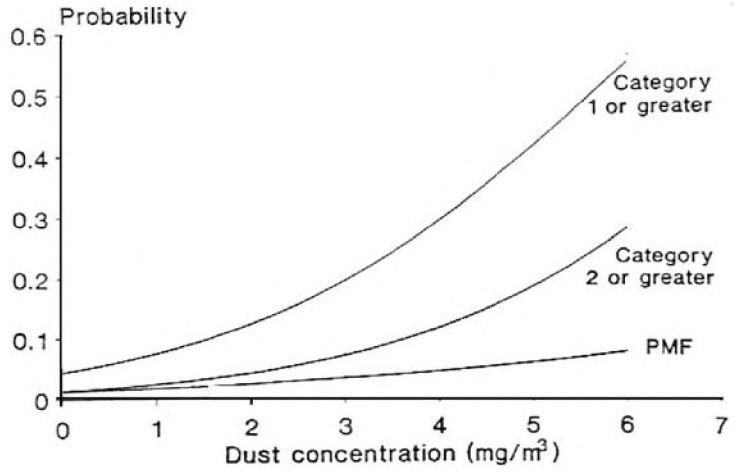

Figure 5.3 U.S. coal miners' predicted risk of contracting irreversible pulmonary disease
Reprinted from W Rom (ed.), Environmental and Occupational Medicine, 2nd edn. Little Brown & Co. Warner, 1992, p. 329.

5.2.5 Asbestos related diseases

Asbestos is a family of crystalline hydrated silicates forming fibres, i.e. with a ratio of length to diameter, or aspect ratio, of more than 3:1. Asbestos fibres thus differ from silica or coal dust particles, which are roughly spherical. This difference in the geometry of the particles is important for the causation of disease. The asbestos fibres that are retained in the lung can be far larger and tend to be deposited in the lower zones of the lung. Macrophages are unable to engulf such large fibres and many remain in the alveoli. Chemical reactions take place on the surface of the asbestos particles that are toxic to lung tissue, resulting in cell death and scar formation.

Asbestos mining in South Africa commenced around the turn of the century and this country became the major global supplier of asbestos of the amphibole family, crocidolite and amosite. In addition, some chrysotile was mined. The asbestos minerals share the properties of heat resistance and tensile strength. They found many uses in heat insulation, construction, textiles and engineering. Today effective and acceptable substitutes are available for all of these applications. Felix et al. (1994) have traced the history of the mining of the three major commercial forms of asbestos in South Africa and the epidemics of asbestos-related diseases that followed.

Although asbestos is no longer mined on any significant scale, there are two ways that mine occupational health services are still likely to encounter asbestos related diseases. The first occurs when individuals are recruited into mining operations with a past history of asbestos mining or residence in an area where there has been environmental contamination with asbestos. The second instance occurs with secondary uses of asbestos in the mining industry such as in boiler rooms for insulation purposes. People who apply or remove asbestos lagging have a significant risk of asbestos related diseases. The Asbestos Regulations in terms of the Occupational Health and Safety Act, while not mandatory for mining operations, contain provisions appropriate to the prevention of asbestos related diseases in this context.

Clinical features of asbestos related diseases

The general features of the asbestos related diseases are summarised in Table 5.4. The asbestos related diseases are well described in the medical literature and diagnostic criteria for these diseases have been published (see Guide to Resources).

Table 5.4: General clinical features of the asbestos related diseases. (Note: two or more of these conditions may coexist)

Pleural plaques	• The most common manifestation of exposure to asbestos. They occur as hard, discrete and often calcified, flat lesions on the surface lining of the inner chest wall • Plaques have been dubbed the "visiting cards of asbestos", a term implying a minimal health effect but a history of some exposure to asbestos. Plaques may appear where there has been only environmental exposure to asbestos • Plaques may signify an increased risk of mesothelioma because they indicate significant asbestos exposure, but are not themselves premalignant
Diffuse pleural thickening and pleural effusion	• Asbestos related diffuse pleural thickening is often the consequence of a pleural effusion that has become organised with fibrosis • When widespread there may be significant functional impairment. Lung function testing is required to evaluate possible impairment
Asbestosis	• The term refers only to the pneumoconiosis, i.e. fibrosis of the lung tissue • It varies in severity. When severe the features include shortness of breath, cough, finger clubbing with cyanosis, basal crackles heard on auscultation of the chest, and ultimately right-sided heart failure and respiratory failure • Radiologically, the features are those of fine-to-coarse irregular and linear opacities that are initially basal. In severe cases the whole lung may be involved • Lung function testing is required to evaluate possible impairment
Mesothelioma	• Mesothelioma is a malignant cancer arising from the pleura or peritoneum (the membrane lining the organs and walls of the chest and abdominal cavities) • Untreated, the disease has a poor prognosis, with less than 10% of sufferers surviving two years. Surgery, radiotherapy or chemotherapy may reduce pain and discomfort
Lung cancer	• Lung cancer is a malignancy that arises either in the bronchial tubes or in the lung tissue • Asbestos related lung cancers usually occur in people who already have asbestosis • Asbestos exposure and tobacco smoking together multiply the risk of lung cancer • Thoracic surgery may be curative, but most lung cancers are inoperable at the time of diagnosis. Radiotherapy and chemotherapy may reduce pain and discomfort

Epidemiology of asbestos related diseases

At its peak in the 1970s asbestos mining directly employed about 25 000 people. Employment in this sector has fallen a negligible number, largely because of a collapse in demand for the mineral consequent on its replacement by safer materials.

South Africa continues to pay the price of having mined this injurious mineral. As shown from MBOD reports the numbers of workers certified as having asbestos related diseases slowly climbed during the 1990s. In 1998/99 asbestosis accounted for 54.2% of all certifications for first-degree pneumoconiosis and 19.6% of all second-degree notifications.

All types of asbestos are known to cause asbestosis, as well as pleural disorders. The consequences of asbestosis for life expectancy have not been fully evaluated in South Africa, but survival in Australian crocidolite mine and mill workers with asbestosis has been studied. Median survival from claim for compensation was 17 years in subjects with asbestosis ILO category 1, 12 years in those with category 2, and 3 years in those with category 3 disease.

In 1960 the association between asbestos exposure and mesothelioma was established in a report of 33 asbestos workers with this rare disease from the Northern Cape crocidolite fields. There is a dose response relationship between asbestos exposure and mesothelioma, i.e. the greater the exposure the greater the risk. However, the threshold is low, i.e. even modest exposures are capable of causing this disease. Younger age of first exposure to asbestos is an additional risk factor because there is a long time lag between exposure and the appearance of the cancer. The potential of the various types of asbestos fibre to cause mesothelioma appears to be crocidolite > amosite > chrysotile.

An association between asbestos exposure, asbestosis and lung cancer (as distinct from mesothelioma) has been recognised for many years. Evidence suggests that asbestos related lung cancers occur more readily in lungs where there is existing fibrosis. However, this fibrosis may only be detectable at autopsy and may not be evident on the chest x-ray. All types of asbestos have been associated with lung cancer.

Environmental contamination has resulted in many cases of fatal asbestos related disease, primarily mesothelioma, an impact that continues in areas of the Northern Cape and Northern Province. Through a special programme the SA government, through the DME, has spent more than R40 million in the rehabilitation of abandoned and hazardous asbestos mines and works. The UK based companies that mined in these areas disinvested in the 1970s and have not made co-payments for this process, nor do they pay levies to the ODMWA Compensation Fund.

Historically, the cost of compensating such occupational diseases has had to be met from South African government revenues. In 2000 the British House of Lords upheld the right of South African former employees of Cape Plc to sue the UK based company for compensation. Cape Plc operated large crocidolite and amosite mines and mills in SA, mainly in the Northern Cape and Northern Province, for more than 70 years. More than 4 000 former employees could ultimately be party to the case.

5.2.6 Chronic obstructive pulmonary disease (COPD)

COPD is closely associated with cigarette smoking. However, COPD has been a compensable disease in the South African mining industry for nearly 40 years. Subsequent research in South Africa has confirmed the effect of underground exposures (i.e. dust, gases and other particulates) on the risk of COPD.

The usual clinical features of COPD are given in Table 5.5.

A 1989 study of black goldminers showed that the risk of chronic airflow limitation increases with duration of underground exposure and is an effect that is independent of the presence of silicosis. The magnitude of this effect has been estimated as an additional decline of 8 ml per year in lung function. In the same study the estimated direct effect of smoking 20 cigarettes a day was an excess decline of 7 ml per year. Differently expressed, the effect of gold mine air exposure on lung function is roughly equivalent to smoking twenty cigarettes a day.

Considerable attention has been paid elsewhere in the world to the relationship between exposure to the coal-mining environment and COPD. Dust related increases in prevalence of chronic bronchitis and chronic airflow limitation have been found in coal miners that are similar for smokers and non-smokers. Although measured lung function abnormalities are greater in smokers, the separate contributions of smoking and coalmine dust to COPD appear to add to each other rather than to multiply each other. An adverse effect of coal mine dust exposure on lung function has been observed even in the absence of radiographically detected CWP.

Table 5.5: Usual clinical features of Chronic Obstructive Pulmonary Disease (COPD)

- COPD is the result of a combination of environmental exposures and genetic susceptibility
- The term COPD is used to describe three inter-related disorders that are often present together to a varying degree:
  - Chronic bronchitis, which is the consequence of airway inflammation and resultant mucus gland hyperplasia. Chronic bronchitis is defined by the presence of cough and sputum production on most days for three or more months of the year for two or more consecutive years
  - Emphysema, which is the destruction of the gas exchanging tissue of the lung consequent on a chronic inflammatory response to environmental exposures
  - Chronic airflow limitation which results from narrowing of the airways as a consequence of both the inflammation occurring in chronic bronchitis and the loss of lung elastic recoil that occurs in emphysema
- Lung function tests are required both to confirm the diagnosis of COPD and to assess its severity

In an unpublished study of coalminers (HEALTH 607 — see www.simrac.co.za), the average decline in FEV1 attributable to coal dust exposure was 17 ml per mg/ml per year of coal dust exposure among active miners. This decline was of the same order as the adverse effect of smoking in this group.

Further information concerning the natural history of COPD in the mining industry can be found in past MBOD reports. These indicate the long duration of exposure required for the disease. In 1989/90 the average period of risk work prior to certification for COPD in living miners was between 25 years (first degree) and 30 years (second degree) in gold mining and somewhat shorter in coal mining. Less than a third of these certifications were in black mineworkers.

With the increased use of lung function tests in periodic and benefit examinations, the number of certifications appears to have risen. In 1989/90 a total of 363 new, upgraded or post mortem certifications for COPD were made by the MBOD. In 1998/9 there were 650 certifications (excluding post-mortem certifications which were not given). It therefore appears that certifications for COPD have approximately doubled over a ten-year period.

Although not usually included under the heading COPD, obstructive lung function loss as a complication of chronic tuberculosis is now well established. In a recent unpublished study of South African goldminers, TB was associated with accelerated loss of FEV1 and FVC (HEALTH 617 — see www.simrac.co.za). Although chronic TB adversely affects both FEV1 and FVC, the predominant effect appears to be obstructive. The implication is that any clinical evaluation of chronic TB, whether at the end of treatment or at any subsequent evaluation, e.g. for a medical benefit examination, must include lung function testing.

5.2.7 Lung cancer

Lung cancer usually arises in the airway tissues where exposure to noxious environmental agents is highest. Growth of the cancer may cause local effects such as obstruction of the airways but death is usually caused by spread of the cancer to other sites in the body. Most lung cancers are diagnosed too late for curative surgery to be undertaken. For illustration, among patients treated at Groote Schuur Hospital in one year only 11% underwent possibly curative surgery. The overall 1- and 2-year survival rates of this group were 18% and 8% respectively.

Lung cancer is the commonest fatal cancer among men in South Africa. Tobacco smoking is globally the most important single cause of lung cancer, but in addition there are a number of possible links between lung cancer and mining.

- The association between asbestos exposure, asbestosis and lung cancer is strong
- There is a link between silica dust, silicosis and lung cancer although the relationship is not as strong as for asbestos

- Certain nickel compounds encountered in the smelting process are considered to be carcinogenic.
- Exposure to radon gas underground and diesel engine emissions are other possible causes of lung cancer in the underground environment

The occurrence of lung cancer in the South African mining industry has received less attention than it warrants, given the seriousness of this condition. One of the barriers to proper assessment of risk is that lung cancer is uncommon among active miners because most cases arise in the fifth or subsequent decades of life, after most miners have retired. For example, lung cancer among asbestos miners comprised only 0.15% of all second-degree certifications by the MBOD in 1998/9.

In addition to controlling exposures of miners to carcinogenic agents, an essential part of health promotion among miners is to discourage smoking and to assist with smoking cessation.

5.2.8 Scleroderma/progressive systemic sclerosis (PSS) and rheumatoid arthritis

Progressive systemic sclerosis is a relatively rare and progressive disorder of the body's connective tissues that is strongly associated with silica exposure. The usual clinical features are described in Table 5.6.

Table 5.6: Usual clinical features of Progressive Systemic Sclerosis (PSS)

• Normal connective tissues are replaced by collagen, a dense fibrous tissue that affects normal functioning. The process usually begins in the skin, which becomes hardened, depigmented and contracts
• If this process remains localised to the skin it is called morphea — this is considered to be a different condition and is not compensable
• Once internal organs are involved the disease is considered to be systemic
• Over time organs such as the lung, oesophagus and kidneys become affected by the disease process. This results in shortness of breath, difficulty in swallowing and kidney failure
• PSS generally has a poor prognosis (< 40% 10 year survival)
• D-penicillamine, colchicine and other agents may alleviate or slow progression of this disorder

An association between PSS and exposure of Witwatersrand gold miners to dust containing a high fraction of silica was first suggested by Erasmus in 1957. Sluis-Cremer later showed an association between PSS and lifetime silica exposure rather than with silicosis. Cases appeared to have had higher intensity of exposure to silica during mining service, rather than longer duration of service.

It has been estimated that the annual incidence of PSS is 81.8 per million amongst black mineworkers in the group aged 33-57 years, compared with approximately 3.4 per million in a general population of similar age. A high incidence of tuberculosis was noted among workers suffering from PSS. Both the 1989/90 and 1998/9 MBOD reports show six certifications for PSS among living black miners. It is likely that PSS is under recognised and underreported among miners.

Rheumatoid arthritis is a disease that is sometimes grouped with scleroderma. The radiological and histological features of silicosis and CWP are sometimes modified in a characteristic manner in miners with rheumatoid arthritis — a phenomenon known as Caplan's syndrome. In a case control study of white gold miners attending the MBOD, Sluis-Cremer showed that miners with rheumatoid arthritis were more likely to develop silicosis, that silicosis was more likely to start with larger radiological nodules (r) and that the rate of progression of silicosis in patients with rheumatoid arthritis appeared to be more rapid than for miners without rheumatoid arthritis.

5.2.9 Miscellaneous mineral dust diseases

Tin (stannosis) and iron oxides (siderosis)

Stannosis is the name given to the condition that results from the inhalation of tin in the form of tin oxide (SnO_2) as fume or dust. Tin oxide does not cause a fibrotic tissue reaction and it is arguable whether stannosis should be called a pneumoconiosis. Iron oxides, which cause siderosis, appear to

be similar in this respect. Since both tin and iron have high atomic weight and consequently absorb x-rays, chest radiographs depict these elements very clearly when they are deposited in lung tissue after inhalation.

Occurrence of stannosis has been described in South Africa. Stannosis in tin mines may be caused by the process of bagging where a highly concentrated (70-80%) tin oxide is packaged prior to transport for smelting. This can be a very dusty occupation. In the smelter itself there is exposure to tin oxide fumes, and most of the cases of stannosis seen at the MBOD have been from tin smelters. Silicaceous rock occurs where tin is mined and silicosis may occur.

There are no reports on the respiratory health of iron ore miners in South Africa. Siderosis may, however, occur in jobs involving welding or metal cutting with high temperature torches.

Hard metal

Hard metal is an alloy of tungsten carbide and a matrix of cobalt to which small amounts of titanium, nickel, chromium and other metals may be added. The unique properties of this compound are its extreme hardness, 90 to 95 percent that of diamond. Because of this hardness and temperature resistance, hard metal is used in numerous mining applications. Hard metal is extensively used in the mining industry in South Africa for the tips of drills. The cutting edges on drills have to be reshaped at intervals by grinding. Workers who grind drill tips will be exposed to hard metal dust unless this operation is carried out under water to prevent any respirable dust being produced.

Pneumoconiosis due to hard metal was first described in the 1940s. This pneumoconiosis takes the form of interstitial lung fibrosis. Cobalt has been identified as the most toxic component of the alloy. The prognosis of hard metal pneumoconiosis with pulmonary fibrosis is generally poor since the disease is slowly progressive despite removal from exposure. A typical end stage with respiratory failure may occur within a few years. The clinical features of this end stage disease closely resemble cryptogenic fibrosing alveolitis. In 1987 Sluis-Cremer published the details of four cases. Since that time there have been no further reports of the condition. The number of people at risk in the mining industry is unknown.

5.2.10 Occupational asthma and Reactive Airways Dysfunction Syndrome (RADS)

Asthma is a condition characterised by reversible narrowing of the airways of the lung that is accompanied by airway inflammation. This inflammation is often caused by an allergic reaction, i.e. an adverse reaction of the body's immune system to a substance in the environment. Occupational asthma is asthma acquired as a result of sensitisation or reaction to a substance present in the workplace environment.

The usual clinical features of occupational asthma are described in Table 5.7.

Table 5.7 Usual clinical features of occupational asthma

• Asthmatic narrowing of the bronchial airways results in coughing, wheezing or breathing difficulty
• This airway narrowing is episodic and can be reversed or prevented by the use of medication
• Sensitisation to a substance is a permanent condition and re-exposure to the causative substance, even in relatively low dose, will predictably result in recurrence of asthma
• Exposure to the causative substance usually results in an asthmatic reaction within 30 minutes (the early response) but this reaction may even occur 18 to 24 hours following exposure (the late response)
• Symptoms of allergic rhinitis (hay fever) and conjunctivitis and skin rashes may accompany occupational asthma, or may precede it
• Smokers and atopic individuals (i.e. people with a predisposition to allergic diseases) are generally at increased risk of developing occupational asthma
• Permanent removal of sensitised individuals from further exposure to the causative agent may result in the asthmatic condition improving, although this is not always the case
• Continuing exposure despite symptoms usually results in progression of the disease and reduces the chances of a complete recovery following removal from exposure

The smelting and processing of certain minerals can give rise to airborne exposures to metallic compounds that are known to result in sensitisation and occupational asthma. This is certainly the case for platinum and nickel compounds. It is probably the case for vanadium as well.

Platinum refining results in exposures to various salts of platinum that are capable of causing sensitisation, allergic rhinitis (hayfever) and asthma. The most potent sensitising agent is ammonium hexachloroplatinate (ACP). Platinum is a very important mineral in South Africa and platinum sensitisation has been studied in some detail in the industry.

Chest tightness and wheezing are the most common symptoms that bring a platinum refinery worker to his doctor. Most sensitised workers have these symptoms immediately following exposure to an atmosphere with soluble platinum salts. They may also experience delayed symptoms, which occur when they are at home in the evening or asleep, commonly at about 2 a.m. The existence of an allergy to platinum salts can be confirmed by skin prick tests in which a dilute solution of the offending salt results in a skin reaction. These tests often become positive a few months before symptoms develop, but are occasionally negative even in the presence of work-related symptoms.

A study carried out at a local platinum refinery showed that most cases of occupational asthma developed early, usually in the first or second year following employment. Conversion to a positive skin prick test was most common in workers who were atopic, as defined by positive skin prick tests to common aero-allergens such as pollens or house dust, and was also more common in cigarette smokers.

Diagnosis does not usually present much difficulty. Although medical treatment may suppress symptoms for a while, the only way to effectively deal with this problem is to withdraw symptomatic individuals from exposure. This must be permanent.

Advances in platinum refining are capable of greatly reducing, but not altogether eliminating opportunities for exposure to platinum salts. Great care must be taken in dealing with chemical spills or other possible sources of accidental exposure. Even under optimal conditions it has been shown that the incidence of sensitisation can range from 0.73 to 6.8 cases per 100 person-months worked, with symptomatic platinum salt sensitivity ranging from 0.59 to 2.4 cases per 100 person-months worked. The DME's OEL for soluble platinum salts is 0.002 mg/cubic meter. Even at exposures below this level it is possible for susceptible individuals to become sensitised. Individuals who have become sensitised to platinum salts will experience symptoms even at exposure levels well below the OEL.

Nickel exposure or exposure to its compounds has been linked to occupational asthma but this appears to be a rare phenomenon and most cases have been in nickel-plating processes, rather than in refining. Vanadium in the form of vanadium pentoxide is a known skin and respiratory irritant. There is some evidence that vanadium pentoxide can result in asthma-like symptoms.

Irritant induced asthma, also known as Reactive Airways Dysfunction Syndrome (RADS), can occur following single high dose exposures to a variety of irritant airborne exposures such as sulphur dioxide, ammonia and chlorine as well as the substances causing inhalation injuries as detailed below. RADS should be suspected when a previously well person has persistent symptoms of airflow obstruction three months after an accidental high dose exposure incident. Lung function tests should show airflow obstruction, or a histamine or metacholine challenge test should be positive, to make the diagnosis.

RADS differs from occupational asthma caused by sensitisation to a substance in the workplace in that it represents a response to direct injury to the airways and the symptoms of airflow obstruction do not necessarily have any relationship to ongoing workplace exposure. However, like asthma, it may become a long-lasting condition.

5.2.11 Inhalation injuries and asphyxia

Inhalation injuries in the mining industry may occur after underground fires with smoke inhalation or from exposure to nitrous fumes from gelignite used in blasting operations. Many other agents are

capable to causing inhalation injuries but only these two sources will be dealt with in any detail. The usual clinical features of inhalation injuries are detailed in Table 5.8.

Table 5.8 Usual clinical features of inhalation injuries

• The inhaled agent is usually directly toxic to the epithelial or subepithelial tissues of the respiratory tract
• Acute effects < 48 hours include: laryngeal oedema, lower airway obstruction, pneumonitis or adult respiratory distress syndrome
• Persistent effects (after weeks or months) include: irritant induced asthma and constrictive bronchiolitis (a condition characterized by fixed airflow obstruction in small airways)
• The site and nature of injury to the respiratory tract depends not only on the dose/amount of the offending agent, but also on particle size and solubility
• Highly soluble agents (e.g. sulphur dioxide, ammonia, aldehydes) have an effect very early (within minutes of exposure) and cause damage and related symptoms to the conjunctivae, upper respiratory tract and proximal bronchi
• Low solubility agents (e.g. nitrogen dioxide, nitrous oxide, ozone and phosgene) have a much later effect (hours to days) and predominantly affect the distal airways and alveoli. Because of the delayed effects and symptoms, exposure is often not noted early by the subject and exposure can thus be prolonged and more damaging
• Smoke inhalation injuries vary from upper airway thermal injury to lower respiratory tract injury, depending on the nature of the exposure and accompanying injuries, especially burns
• Following an exposure incident where there is a possibility of an inhalation injury affected persons must be referred to an inpatient facility for 24-48 hours of observation for possible delayed effects
• In all cases where there is respiratory difficulty supplemental oxygen must be given. IV steroids are indicated for severe nitrous inhalation injuries
• All persons with inhalation injuries should be re-evaluated after three months. Lung function tests should be carried out to detect persistent effects (RADS or constrictive bronchiolitis)

Asphyxia among miners may result because of the development of a non-respirable atmosphere in the mine secondary to an explosion or a fire. Carbon monoxide and other products of combustion may contribute to the tissue deprivation of oxygen that results in asphyxia. Self-contained self-rescue apparatus and safety bays underground where oxygen is available are essential to prevent deaths from asphyxia. A second important cause of asphyxia occurs when miners trapped under rock falls experience chest compression. Relief of the compression using hydraulic jacks within the first ten minutes may be life saving.

Nitrogen dioxide

Nitrogen dioxide is a heavy red/brown pungent gas produced by explosives. It is denser than air and therefore accumulates close to the ground in the workings following blasting. Adequate time is required for the gas to be cleared from the area after blasting is done. This requirement has set the pattern of work in the gold mines for more than half a century, in which all blasting is done at the end of the shift and no one is allowed to enter the workings until the next morning when the dust has settled and the nitrous fumes have dispersed. Very high concentrations of nitrogen dioxide may result if explosives burn during underground fires.

The DME OEL for nitrogen dioxide is 3 ppm. Because of the gas's low solubility, the conjunctivae and upper respiratory tract are not irritated at this or higher levels and 50 ppm can be breathed for some time before discomfort results. This lack of an irritative response at toxic levels of the gas can result in prolonged exposure and delayed presentation of the lung injury. Because of the gas's low solubility, damage is deep in the respiratory tract at the level of the small conducting airways and adjacent respiratory bronchioles.

Following high dose or prolonged exposure, the onset of shortness of breath and cough is delayed for about three hours, but ranging from half an hour to 30 hours after exposure. Cases should be observed for at least 48 hours following exposure incidents, even if asymptomatic and with a normal chest

x-ray. Because of the marked variation in individual response to inhalation injury, follow-up lung function tests should be performed after 3 months to ensure a return to normality or to document presence of persistent airflow obstruction that could be a consequence of irritant induced asthma or constrictive bronchiolitis.

Smoke inhalation injuries

Underground fires are an extremely serious occurrence with risks of asphyxia and smoke inhalation injuries. Fires underground occur usually in coal and gold mines where methane gas deposits initiate explosions that are then propagated as fires. Propagation of fires in coalmines is a special hazard since the coal dust particles suspended by the original explosion are combustible. Underground fires burn any combustible material in the mine, making the resultant fire smoke a complex mixture of different ingredients, which vary not only from fire to fire, but also within the time course of a single fire.

Fires deplete atmospheric oxygen underground creating a danger of asphyxia but also increasing the rate of incomplete combustion with production of carbon monoxide and other toxic gases. Incomplete combustion of plastics and polyurethanes will emit both carbon monoxide and hydrogen cyanide and is therefore especially hazardous.

Inhalation injury causes 50 to 70 percent of the mortality of all burns patients. Simple fires usually result in pure thermal burns affecting only the upper respiratory tract because of the low heat-carrying capacity of dry smoke. However, in patients with severe burns and upper airway injury, most also have involvement of the lower respiratory tract. Inhalation injury can occur in the absence of surface burns.

Patients judged to be at risk of inhalation injury from fire and/or smoke exposure should be observed in hospital for at least 24-48 hours, as symptoms are often delayed. Inspired air should be humidified and supplemental oxygen given. The possibility of carbon monoxide or cyanide poisoning should be considered.

Again, because of the marked variation in individual response, follow-up lung function tests should be performed after 3 months to ensure a return to normality or to document presence of persistent airflow obstruction that could be a consequence of irritant induced asthma or constrictive bronchiolitis.

5.3 Legal framework for prevention and compensation of occupational lung disease

The legal framework relevant to prevention of occupational lung diseases in the mining industry is the Mine Health and Safety Act and certain regulations, guidelines or guidance notes that have been published or will be published in terms of that Act. This legal framework is described in Chapter 1 and only aspects of direct relevance to the medical prevention of occupational lung disease will be considered here, specifically medical surveillance.

The starting point for prevention is hazard identification and risk assessment, considered in Chapter 3. Measurement of airborne pollutants is dealt with in Chapter 4. Section 11 of the MHSA requires the employer to assess and respond to risk. Once a significant risk has been identified, Section 13 of the Act requires the employer to establish and maintain a system of medical surveillance of employees exposed to health hazards if necessary to do so in terms of a risk assessment, or if required to do so by regulation or notice. The definition of employee in this instance includes the employees of contractors who perform work in a mine.

Benefit examinations, compensation and autopsies in terms of the Occupational Diseases in Mines and Works Act are dealt with in Chapter 14. Similarly, procedures for reporting injuries of the respiratory system not covered by the ODMWA but falling under the COID Act (see Table 5.1) are discussed in Chapter 14.

5.4 Medical surveillance

Medical surveillance is a programme of regular examinations designed to detect disease for early treatment, referral or appropriate placement of employees, as well to collect information for risk assessment and prevention purposes. The legal duties and rights of parties with respect to medical surveillance are summarised in Appendix 5.1.

A medical surveillance programme for occupational lung disease should be initiated if risk assessment indicates that there is significant risk, usually defined in occupational hygiene terms. The occupational medicine practitioner will be required to make decisions or recommendations about employees requiring inclusion in the surveillance programme. In such instances an informed assessment of the accuracy and generalisability of the available hygiene data to different groups of employees must be made. The past history of exposures in these groups and the occurrence of occupational lung diseases should be considered. If there is uncertainty about the presence of significant risk, it is better to conduct additional occupational hygiene measurements and to carry out medical surveillance until a clearer picture of risk is obtained. This policy can then be reviewed at suitable intervals.

In the case of airborne pollutants significant risk is defined as follows:

- If the prevailing hazard exposure concentration as evaluated by the air quality programme is > 50% of the occupational exposure limit (OEL) for that hazard, then a full programme of medical surveillance must be instituted for all employees exposed to the hazard. This programme must include initial, periodic and exit medical examinations according to a programme that is appropriate to the level of risk
- If employees have previous exposure to mineral dust carrying a significant risk of disease, they must also be included in a medical surveillance programme to monitor possible deterioration or development of disease
- If the prevailing hazard for mineral dust exposure is > 10% but < 50% of the OEL, a full programme of medical surveillance may not be required. Initial and exit medical examinations are appropriate to this level of risk

OELs for important airborne pollutants mentioned in this chapter are given in Table 5.9.

Table 5.9: Proposed occupational exposure limits for important airborne pollutants causing occupational lung diseases (DME, 2001)

Crystalline silica (all forms)	0.1 mg/cubic meter
Silica, amorphous inhalable Silica, amorphous respirable	6 mg/cubic meter 3 mg/cubic meter
Coal dust (respirable particulates): < 5% crystalline silica (quartz) > 5% crystalline silica (quartz)	 2 mg/cubic meter As for crystalline silica
Soluble platinum salts	0.002 mg/cubic meter

5.4.1 Purpose of medical surveillance as mandated by MHSA

The MHSA gives clear guidance on the purpose of a medical surveillance programme, summarised in Table 5.10. If there is a requirement for a medical surveillance programme the employer must draft a Code of Practice for medical surveillance in accordance with the appropriate DME guidelines or regulations, as required by section 9.2 of the Act.

It is clear from the MHS Act that there is an intention that there be a clear communication between the occupational medical practitioner responsible for the medical surveillance programme and the health and safety committee or committees on the mine. A copy of the annual medical report, detailing occupational lung disease statistics for the most recent year must be given to the health and safety committee. This is the minimum requirement, but clearly the occupational medical practitioner can play an active role in health promotion, in conjunction with the health and safety committee.

Table 5.10: Elements and aims of a medical surveillance programme in terms of proposed DME Guidelines.

A medical surveillance programme of employees should:
• Be appropriate to the health hazard
• Provide information that the employer can use in determining measures to –
• Eliminate, control or minimise the health risk and hazards
• Prevent, or detect and treat occupational diseases at an early stage
• Consist of an initial medical examination and other medical examinations at appropriate intervals
• Establish a baseline against which subsequent changes in the health status of employees can be evaluated over time
• Be designed to identify medical conditions that may render employees temporarily or permanently unable to perform their occupations
• As far as reasonably practicable ensure that –
• Employees are fully informed of the health risk and hazards associated with their occupations and of the measures to eliminate, control and minimise the health risk and hazards
• The health status of employees does not place their health at increased risk in a particular working environment nor place other employees or the public at increased risk

5.4.2 Employee education and medical surveillance

The training and education implied by the requirement that employees be informed about health risks needs to be linked to medical surveillance wherever possible. Medical surveillance is itself an opportunity for education. Further, such surveillance is more likely to be acceptable to employees if they understand its purpose.

There are a variety of media that can be used for employee education. Such media are likely to have the most impact if they carry a simple message and are locally developed with professional assistance. Involving the health and safety committee representatives in the design of the media and programmes to disseminate the message will help to ensure relevance and impact. The various media include formal training sessions, peer education methods, videos, posters and warning signs in the mine. As far as is practicable all vernacular or mother tongue languages in use by miners at a mine should be used.

Educational interventions need to be provided in an appropriate form for both newly engaged miners and with more experienced miners. Such activities could be timed to coincide with medical surveillance, i.e. at 3-year periods. Topics that are of particular relevance to lung disease include: explanations of relevant airborne pollutants, their health effects and preventive measures in place at the mine; symptoms of pulmonary TB to encourage appropriate self-presentation to health services; the hazards of smoking, and HIV/AIDS education.

The interaction of smoking and occupational risk factors for lung disease needs to be emphasised. The combined effect of occupational airborne pollutants and smoking may be more than the sum of the adverse effects of each risk factor alone. Investment in dust control and in smoking cessation programmes to assist workers to avoid or give up smoking will thus both contribute to the reduction of the risk of occupational lung disease.

5.4.3 Medical surveillance for pneumoconiosis and COPD

Recommended current practice for tests and their frequency in workers exposed to > 50% of the OEL of mineral dusts known to cause pneumoconiosis or COPD is summarised in Table 5.11. Each of the tests to be performed will be discussed separately below.

Table 5.11: Proposed regulations for medical surveillance in the case of exposure to mineral dust (silica, coal, or other mineral dust known to cause pneumoconiosis or COPD)

Examination	Tests to be performed
Initial — carried out on starting or before undertaking work.	Respiratory questionnaire Cardiorespiratory examination Chest x-ray (large plate) Lung function test (spirometry)
Periodic — every three years	Cardiorespiratory examination Chest x-ray (large plate) Lung function test (spirometry)
Exit — when employment terminated for any reason	Cardiorespiratory examination Chest x-ray (large plate) (not necessary if done < 3 months before) Lung function test (spirometry) (not necessary if done < 1 year before)

5.4.4 Medical surveillance for occupational asthma

The problems encountered in platinum refineries or other environments, where sensitising agents are encountered, require specific medical surveillance programmes. Only those for platinum salt exposure will be detailed. At an initial medical examination it is justifiable to exclude persons from such an environment if they already suffer from asthma on the basis that the risk to their health from additional sensitisation to platinum salts is unacceptable. There are arguable grounds for exclusion of current smokers since they are also at increased risk for developing occupational asthma. There is general acceptance that there are no grounds for exclusion of individuals who are atopic (as defined by positive skin prick tests to common non-occupational aeroallergens).

Initial medical examination should include a respiratory symptoms questionnaire, a lung function test and skin prick tests for the occupational sensitising agent that will be encountered in the workplace, if such a test is available. These three examinations should be repeated every six months during the first two years of employment and thereafter annually. They should also be repeated if an exposed individual presents to the occupational medical practitioner with appropriate symptoms. Methods of conducting skin prick tests should follow standard procedures published in the literature (see Merget 1991). If an individual develops appropriate symptoms but has negative specific skin prick tests, a specific bronchial provocation test with platinum salts is justified if it can be carried out under close supervision of a medical practitioner and standard procedures are followed.

5.4.5 Medical surveillance for mesothelioma, lung cancer and Progressive Systemic Sclerosis (PSS)

At the present time there are no screening methods of proven value in the early detection of mesothelioma or lung cancer. There is also no method of proven usefulness for the early detection of individuals at special risk of developing PSS. Early recognition of PSS and removal from further silica exposure could possibly reduce the severity of the PSS. Early diagnosis of this condition is not easy, but mine medical officers should be constantly reminded about the possibility of a diagnosis of PSS.

5.4.6 Data management and reporting responsibilities as part of medical surveillance

Record maintenance

The MHSA requires that employee records be kept for 40 years. Preservation of hard copy and software for such a long period requires special expertise. Within the company there must be the allocation of responsibility to ensure that records are kept in the proper manner so as to remain both legible and accessible.

It is not a legal requirement that all records are kept as hard copy/paper, but there should always be at least partial (summary) paper records. Chest x-rays from medical surveillance must be kept and not

sent for recycling for silver. If computerised records are kept it is essential that provision is made for all records to be backed up, and for transfer of old records onto new software systems when systems are changed.

The medical surveillance record system also has to meet the responsibility towards the employees of contractors. A standard operating procedure should be in place on the mine with respect to employees of contractors, to identify those working in risk areas and avoid unnecessary repetition of medical surveillance of short-term employees.

Reporting

Section 13 (2)(b) of the MHSA is explicit that one of the most important purposes of a medical surveillance programme is to provide information to assist with the prevention of occupational diseases. The main regular summary of medical surveillance data is the annual medical report. The data relevant to occupational lung disease required for such a report, as mandated by regulation, are summarised in Appendix 5.2.

Occupational medical practitioners have additional statutory reporting duties in terms of the ODMWA, and are required to produce exit medical certificates and to report diagnosed occupational diseases to the DME's SA Mining Occupational Diseases Database (SAMODD). As is evident from Appendix 5.2 there is a great deal of overlap in the data required for these various responsibilities. It is combinations of these data that will give the most informative reports.

Computerised records will help to meet these various responsibilities, particularly if they are stored in relational databases. Several relational database software programmes are available commercially. An advantage of a relational database in managing a medical surveillance programme is that all data concerning an individual can be interrelated through particular identifiers, such as name or company number. Using a relational database, new data captured in each year's surveillance can be linked to the results in previous years using these identifiers. Using the same or compatible software it is possible to import or use other data, for example, employee identifiers, past employment record and compensation history from the human resources department.

Most database software enables checks that data are not incorrect by pre-programming plausible values of limits for data being entered. It allows for deviations from baseline measurements to be flagged and drawn to the attention of the occupational health practitioner (such as a 10% or greater decline in lung function). Database programmes can also produce outputs or reports according to any required format for the data contained in them.

An important function of a medical surveillance records system is to link occupational hygiene and medical surveillance information, as required by Section 12 (3) of MHSA. The occupational hygiene database should allocate every person working in the mine to a particular Sample Area for purposes of assigning them to an Activity Area and within that to a Homogeneous Exposure Group (HEG). Within each HEG the hygienist will be sampling the exposure of a 5% proportion of exposed individuals, according to a schedule that is more frequent if there is higher risk. Linkage of exposure and outcome information will enable a more accurate risk assessment of the effects of dust and other exposures in the medium and long term.

Performance indicators.

The data captured in Appendices 5.2 to 5.4 can be used to derive a variety of useful measures of the occurrence of occupational lung disease on a mine. Cases should always be expressed per number of employees. These measures can be used to track trends over time, or to compare different parts of a mine characterised by exposure information. Such comparative rates will better serve the functions of risk assessment and prevention.

Incidence: annual new cases of pneumoconiosis or new certifications

The minimum requirement for the annual medical report and SAMODD is the crude incidence (number per 1 000 employees) of new ODMWA certifications in the whole mine. A more specific indicator of occurrence would be new certifications per 1 000 employees in a sample area or new

certifications per 1 000 employees in an HEG. The most sensitive available indicator would be new cases of pneumoconiosis diagnosed in the radiological surveillance programme for exposed employees, as detailed below. Sample areas or HEGs can be ranked according to the measures of exposure made there and an approximate dose-response relationship can be derived by examining the incidence of certifications at each level. In large mines, where there may be more than one Health and Safety Committee, certifications could be reported according to activity area for which the Committee is responsible.

Pneumoconiosis, COPD and the occupational cancers mentioned are diseases of long latency. In the instance of silicosis, it may take 15 years of underground gold mining before the radiological changes become evident. This means that occurrence rates of these diseases are not particularly useful in indicating areas where engineering control of dust is required. Current environmental measurements are thus a better indicator of risk. In diseases of short latency, such as occupational asthma, the occurrence of cases in a particular area, for example in a section of the platinum refinery, would draw attention to areas in need of control.

Cumulative incidence: all certifications over a period of years

Compensation history, or cumulative incidence of previously certified cases of occupational lung diseases over a given period such as five years, is also a potentially useful statistic on disease occurrence. Cumulative incidence, like simple incidence can use as its employee denominator the whole mine, sample or activity area, HEG, or occupation.

5.4.7 Respiratory questionnaires

Respiratory questionnaires are required for initial examinations of persons who will be exposed to significant risk from mineral dust.

Although questionnaires are useful for some aspects of a medical surveillance programme, they are not required for periodic or exit examinations of persons exposed to mineral dusts. Use of respiratory questionnaires in periodic or exit examinations is only advocated for circumstances where there is a risk of occupational asthma. Specific questions should then be added to the questionnaire to assist in eliciting appropriate symptoms of asthma.

An example of an abbreviated questionnaire is attached as Appendix 5.3. In using such a questionnaire as a screening tool, any positive response to a question about symptoms should be followed up by additional questions.

Questionnaires should be administered by people who have been specifically trained for the task. The multilingual environment of mines makes use of such questionnaires challenging since some Southern African languages do not have direct translations for terms such as wheezing. One approach is to prepare mother tongue language translations for all languages in use at the mine. This is often feasible since there may be only three or four languages in common usage. Usual practice in preparing the questionnaire is to have the questions translated by a person proficient in the target language, followed by independent back-translation of the questions into English by another person proficient in both languages. Alternatively where the interviewers have multilingual abilities, it is acceptable for the questionnaire to be available only in English and the questions translated appropriately as the interview is conducted.

There can be considerable savings of time and effort if questionnaire administration is computer assisted. Entry of responses to computer prompted questions results in immediate data capture, as well as guiding the interviewer through the process. The interviewer can be prompted if no response is entered or if a response cannot be correct. Epi-Info is an example of free software, available through the Centers for Disease Control (CDC), USA., that can be used for data capture from a questionnaire. (See CDC website under Guide to Resources).

5.4.8 Chest Radiology

All of the pneumoconioses (silicosis, asbestosis, coal workers' pneumoconiosis and others) are best detected by full size postero-anterior (PA) (35 x 43 cm, or 35 x 35 cm) plain chest x-rays of acceptable

technical quality. A large PA film alone is acceptable, although lateral films should be taken if pathology other than pneumoconiosis is suspected. Oblique views of the chest are required for a more sensitive evaluation of asbestos related pleural disease.

Chest x-rays are usually able to detect signs of pneumoconiosis well in advance of the onset of symptoms and abnormal lung function tests. The most important limitations in the use of chest x-rays in medical surveillance are the technical quality of the x-rays and the capabilities of the interpreter of the films.

It is the radiographer's responsibility to produce a film quality acceptable for detection of pneumoconiosis. It is the film reader's first decision when looking at the film to judge whether it is acceptable. A close working relationship between radiographer and reader will improve quality and reduce the cost and inconvenience of having to repeat films.

Table 5.12: Features of a technically adequate chest x-ray for detecting pneumoconioses

- Labelled with name, date and the location
- Well centered, with apices and costophrenic angles visible
- Full inspiration (such that the sixth rib intersects the shadow of the diaphragm near its dome). (Underinspired films can result in over-reading of pneumoconiotic opacities)
- Scapulae clear of the inner aspect of the chest wall where they may obscure other lesions
- Reasonable contrast, uniform across the film and allowing easy distinction between blood vessels and air-filled parenchyma. Film fogging or poor film-screen contact during developing can result in poor contrast
- Acceptable exposure of the film such that the major pulmonary vessels can be clearly seen behind the heart. Overexposure (film too dark) results in underreading of pneumoconiotic opacities, and conversely under exposure (film too light) can result in overreading
- Avoidance of artefacts such as mottle, which can result in small opacities visible throughout the film including the soft tissues
- For screening radiology, high kV films (125 kV) are recommended

The ILO has established a universally recognised system for grading of the degree of changes on the chest x-ray by comparison of an individual's chest x-ray with standard films. Although the ILO classification is primarily an epidemiological tool it has also widely used as an aid to medical surveillance programmes and has even been adopted as a grading system by many compensation authorities. Sets of the ILO radiographs are available through the ILO Office in Pretoria or Geneva (cost: approx. US$600).

Appendix 5.4 shows a reading form for use in radiological occupational lung disease surveillance programmes and it incorporates the ILO Short Classification as used at the MBOD. The primary reading form lists seven categories of abnormality that will be encountered by the reader of x-rays in a radiological surveillance programme. The significance of each finding is detailed, together with the recommended plan of action. Information generated by collecting information from use of such a form will provide important information for outcome indicators, e.g. annual incidence of new cases of pneumoconiosis diagnosed.

SIMRAC is currently developing a programme that will make available training materials for distance learning in these skills. Accredited ability to interpret films taken for occupational lung disease surveillance can be expected to become the standard in the mining industry.

Miniature chest x-rays are considered to be less sensitive than large ones in the detection of pneumoconiosis and are not recommended for this purpose.

There is no system analogous to the ILO system to categorise miniature chest x-rays. If previously undiagnosed pneumoconiosis is present on a miniature x-ray, a large film should be obtained to confirm the diagnosis. Use of miniature radiography in active case finding of tuberculosis is dealt with in Chapter 6. There is currently no formal training and accreditation process for skills in the reading of mass miniature radiography for tuberculosis.

The radiation hazard of even repeated full size chest x-rays is very low. Miniature radiography carries a significantly higher radiation dose.

High resolution computerised tomography (CT) scans of the chest offer significant advantages in imaging of the lungs and are useful in diagnostic evaluations of complicated occupational lung disease cases.

5.4.9 Lung function testing

The measurement of lung function by spirometry is now a required part of medical surveillance for workers exposed to mineral dusts or other airborne pollutants. Spirometry consists of a forced expiratory manoeuvre for measurement of Forced Expiratory Volume in one Second (FEV1) and Forced Vital Capacity (FVC). There are a number of spirometers commercially available for use in the occupational setting.

Quality control of spirometry is essential to produce meaningful results. There are many factors such as subject understanding, tester competence and equipment calibration, which affect the reliability of the test.

The DME has recently published a guidance note for occupational medical practitioners to assist in the performance and interpretation of lung function tests that accord with best current practice for this test (See Guide to Resources). This guidance note is to be widely distributed by the DME and mine occupational health services will be expected to use it as a best practice standard.

In pneumoconiosis, lung function abnormalities occur some years after radiological changes have become obvious. It is increasingly being realised that lung changes caused by past, even successfully treated, pulmonary tuberculosis cause significant lung function abnormalities. In these conditions lung function tests are not used for the screening or diagnosis, but rather for the documentation of the degree of abnormality related to the underlying abnormality.

In COPD and asthma, lung function tests serve both as a screening test and as the diagnostic test that, particularly in COPD, give an indication of the degree of severity.

Relational databases are well suited to storage and subsequent analysis of lung function data. A variety of commercial software programmes, sometimes made available with equipment, are available for converting information depicting the full flow-volume curve into a database for storage. The minimum information that must be retained is age, height, FEV1, FVC and the ratio of FEV1/FVC expressed as a percentage.

Reference or predicted values for lung function testing are used to decide whether a single test result falls outside of the normal range or to categorise the degree of abnormality. In response to consistent findings of ethnic variation in FVC and FEV1, international guidelines have recommended that reference equations be derived from healthy non-smoking populations similar in origin to that of the persons tested. In a recent SIMRAC report on reference values for FEV1 and FVC for use in entry medical and medical surveillance examinations in the mining industry, the use of prediction equations derived from a study by Louw (1996) for black male employees in the mining industry, and from a study by Mokoetle (1995) for black female employees, was recommended. European Community for Coal and Steel (ECCS) prediction equations were recommended for white men and women employees in the mining industry These equations, which can be programmed into spirometers, are contained in the report. A detailed discussion of how these recommendations were arrived at, together with advice on the monitoring of lung function over time, are included in SIMRAC Health 610 report (See Guide to Resources).

Staff properly trained in the performance of lung function tests are essential if reliable results are to be obtained. It has been recognised for some time that provisions in South Africa for training to achieve competency in performing screening spirometry in industry are inadequate. While spirometry

is part of the curriculum of diplomas in occupational health for doctors and nurses, graduation with this qualification does not necessarily carry with it the competence to perform screening spirometry. Medical technologists who specialise in pulmonary technology acquire competence in spirometry in addition to many other skills, but are in fact over-skilled for the performance of screening spirometry. A more limited form of training is required to ensure competency for the performance of screening spirometry in industry. There are currently moves to put in place a curriculum and system of accreditation in screening spirometry, similar to the system in place for training and certification of competency in the performance of screening audiometry.

5.5 Guide to information resources

5.5.1 Official reports

Department of Minerals and Energy. Commission of Inquiry into Safety and Health in the Mining Industry. Leon RN, chairperson. Pretoria, Department of Minerals and Energy, 1995. (Leon Commission).

Department of Health (and previously Department of Minerals and Energy). Reports of the Director, Medical Bureau for Occupational Diseases. 1981-1999. Government Printer, Pretoria.

National Centre for Occupational Health. A report on occupational health indicators for South Africa. Report No. 1/99. NCOH, Johannesburg. 1999.

World Health Organization. Recommended health-based limits in occupational exposure to selected mineral dusts (silica, coal). Technical Report Series 734. World Health Organisation, Geneva. 1986.

5.5.2 Official regulations and guidelines

Department of Minerals and Energy. Regulations: Annual Medical Report. Occupational Health and Safety Inspectorate, DME, December 1999.

Department of Minerals and Energy. Minimum standards of fitness to work in a mine. Guideline for the compilation of a mandatory code of practice. Occupational Health and Safety Inspectorate, DME, February 2001.

Department of Minerals and Energy. Guidance note for occupational medical practitioners: Lung function testing. Occupational Health and Safety Inspectorate, DME, August 1999.

Department of Minerals and Energy. Airborne pollutants. Guideline for a mandatory code of practice for airborne pollutants. Occupational Health and Safety Inspectorate, DME (in draft)

Department of Minerals and Energy. Regulations for medical surveillance for exposure to asbestos, silica dust and coal dust. Occupational Health and Safety Inspectorate, DME (in draft).

5.5.3 Further reading

Silicosis

Cowie RL, van Schalkwyk MG. The prevalence of silicosis in Orange Free State gold miners. *Journal of Occupational Medicine* 1987; 29: 44-46.

Hnizdo E, Sluis-Cremer GK. Risk of silicosis in a cohort of white South African gold miners. *American Journal of Industrial Medicine* 1993; 24: 447-57.

Leger JP. Occupational diseases in South African mines — a neglected epidemic? *South African Medical Journal* 1989; 76: 557-561.

Steen TW, Gyi KM, White NW, et al. Prevalence of occupational lung disease among Botswana men formerly employed in the South African mining industry. *Occupational and Environmental Medicine* 1997; 54: 19-26.

Trapido AS, Mqoqi NP, Williams BG. Prevalence of occupational lung disease in a random sample of former mineworkers, Libode District, Eastern Cape Province, South Africa. *American Journal of Industrial Medicine* 1998; 34: 305-313.

Asbestos

Felix M, Leger JP, Ehrlich RI. Three minerals, three epidemics — mining and asbestos related disease in South Africa. In : Mehlman MA, Upton A, eds. *The identification and control of Occupational and Environmental Diseases*. Princeton: Princeton Scientific Publishing, 1994.

SORDSA

Diagnosis and management of asbestos related diseases in South Africa — Rees D, Cantrell I, (eds.). *National Centre for Occupational Health*, Johannesburg 2000.

COPD

Cowie RL, Mabena SK. Silicosis, chronic airflow limitation and chronic bronchitis in South African gold miners. *American Review of Respiratory Diseases* 1991; 142: 80-84.

Progressive Systemic Sclerosis

Cowie RL, Dhansay RD. Features of systemic sclerosis (scleroderma) in South African gold miners. *South African Medical Journal* 1990: 77:400-402.

Medical surveillance

Chest radiography

International Labour Office. Guidelines for the use of ILO international classification of radiographs of pneumoconiosis (revised 1980). *Occupational Health and Safety Series*, No. 22. Geneva: ILO, 1980.

Lung functions

Department of Mineral and Energy. Safety in Mines Research Advisory Committee (SIMRAC). Development of lung function reference tables suitable for use in the South African mining industry. Ehrlich R, White N, Myers J et al. (HEALTH 610A). SIMRAC, Johannesburg, 2000.

Louw SJ, Goldin JG, Joubert G. Spirometry of healthy adult South African men. Part I. Normative values. *South African Medical Journal* 1996; 86:814-819.

Allergy testing/platinum

Merget R, Schultze-Werninghaus G, Bode F, Bergmann E-M, Zachgo W, Meier-Sydow J. Quantitative skin prick and bronchial provocation tests with platinum salts. *British Journal of Industrial Medicine* 1991; 48; 830-837.

Table 5.13. Websites covering occupational lung disease and medical surveillance.

Address	Content
www.simrac.co.za	Research reports on occupational lung disease and medical surveillance commissioned by SIMRAC.
www.asosh.org	South African occupational health and safety information, including laws, resources and links to other sites.
www.compensation.gov.za	Compensation Commissioner (COIDA). General information, procedures, statistics and forms.
www.ohscinfo.tripod.com	DME site for occupational hygiene standards, including those in development.
www.cdc.gov/niosh/homepage.htm/	National Institute for Occupational Health and Safety, USA. Research and advisory agency. Large database of reports and information on occupational health and safety.

Appendix 5.1: Legal duties and rights with respect to occupational diseases and medical surveillance programmes in terms of the MHSA

Employers must	• 13 (3): engage the services of a full-time or part-time occupational medical practitioner, together with, insofar as is necessary, other appropriately qualified occupational health practitioners • 13 (3)(b): provide occupational medical practitioners with the means necessary to perform their functions • 18: pay the costs of all clinical examinations and medical tests • 13 (3)(c) & 8 (a)&(b) & 15 (2) (a)&(b): keep a record of medical surveillance for each employee exposed to a health hazard, store it safely and not dispose of it for 40 years or until the mine closes. When the mine closes the records of medical surveillance to be delivered to the Medical Inspector • 12 (3): keep occupational hygiene records in a manner that can be linked as far as practical to each employee's record of medical surveillance • 17 (1): if the services of an employee who was subject to medical surveillance are terminated for any reason, arrange for an exit medical examination of the employee • 13 (6): conduct an investigation in terms of section 11 (5) if any employee is declared unfit to perform work as a result of an occupational disease
Occupational medical practitioner must	• 13 (5): take every measure that is reasonably practicable to promote the health and safety of employees at the mine and assist employees in matters relating to occupational medicine • 16 (1 & 2): compile an annual medical report covering employees at that mine, giving an analysis of the employees' health based on employees' records of medical surveillance, without disclosing the names of the employees. Copies of the report to be provided to the employer, the health and safety committee and the DME Medical Inspector • (15) & 13 (2)(a): maintain confidentiality of medical surveillance records, to be stored in a safe place and made available only in accordance with the ethics of medical practice, or if required by law or court order, or if the employee has in writing consented to the release of the information • 13 (7): inform the employer and employee in the event of the employee being temporarily unfit to perform work as a result of an occupational disease • 14 (4)(a & b): when conducting an exit medical examination ensure that an exit certificate is produced, indicating the results of all medical surveillance and the presence or absence of any occupational disease. A copy of the exit certificate to be entered into the employees' record of medical surveillance • 19 (2): provide the employee with a copy of the exit medical certificate • 20 (4): in the instance of an appeal against a finding that an employee is unfit to perform any particular category of work, or a finding contained in an exit medical certificate, provide a report to the Medical Inspector
Employee	• 19 (1) (a)&(b) may request and be provided with a copy of any medical surveillance record or part of it that relates to him/herself • 17 (1)(3): must attend the arranged exit medical examination • 20 (1) & (2): may appeal against a decision that he/she is unfit to perform any category of work or any finding contained in an exit medical certificate. The appeal must be lodged with the Medical Inspector within 30 days of the relevant decision

Appendix 5.2: Minimum data requirements for various reporting responsibilities of an occupational medicine practitioner in the SA mining industry

	Annual Medical Report	SAMODD Input Form	MBOD Benefit Application	Exit Medical Examination
Name and Surname of individual	No	Yes	Yes	Yes
Individual's signature	No	No	Yes	Yes
Gender	No	Yes	No	No
National ID Number / Passport Number	No	Yes (1)	Yes (1)	Yes (1)
Industry Employee Number (1)	No	Yes	No	Yes (1)
Employee Number (1)	No	Yes	Yes (2)	Yes (2)
Date of birth	No	Yes	Yes	Yes
Date of death	No	Yes	Yes	No
Individual's address	No	No	Yes	No
Name of mine / employer	Yes	Yes	Yes	Yes
Employee of a contractor	Yes	Yes	No	No
Type of mine or commodity mined	Yes	No	Yes (2)	Yes
Total number of employees in the mine	Yes (3)	Yes (4)	No	No
Employee job title	No	No	Yes (2)	Yes (2)
Years of exposure in current job	No	Yes	Yes (2)	Yes (2)
Hazards exposed to in current job	No	Yes	Yes (2)	Yes (2)
Previous job titles, years of exposure with current and former employers	No	No	Yes (2)	Yes
Name, address, telephone number of OMP or other person completing form	Yes	Yes	Yes (5)	Yes (5)
Submitting party's case reference number	No	Yes	No	No
Date of initial medical examination	No (6)	No	No	Yes
Date of most recent medical examination	No	Yes	Yes	Yes
Clinical findings at most recent medical examination	No	No	Yes (7)	No
Results of lung function tests	Yes (8)	Yes	Yes	Yes
Results of chest radiograph	Yes (8)	Yes (9)	Yes (10)	Yes (9)
Results of other tests (11)	Yes (8)	Yes	Yes	Yes
Diagnosis	Yes (8)	Yes (12)	Yes	Yes (13)
ODMWA benefit application made.	No	Yes	—	Yes
ODMWA compensable disease	Yes	Yes	—	Yes
Previous compensation history	No	No	Yes	Yes
MBOD Reference number	No	No	Yes	No

Footnotes:
(1) National ID number, Passport number, DME ID number, industry number, company number, TEBA number and MBOD number are optional. (2) The MBOD and exit medical report requires a full occupational exposure history, as well as employee ID numbers with previous employers in the mining industry. (3) The annual report requires that number of employees (including contract workers) subject to medical surveillance in terms of Section 13 be reported, together with the total number of hours worked in the reporting period. (4) SAMODD requires reporting of the employer's workforce for the previous calendar year, classified into surface, underground, open cast or at sea. (5) Signature also required. (6) The annual medical report requires a summary of the number of initial, periodical and exit examinations performed. (7) MBOD requires past and present medical history, findings of height, weight, blood pressure, general and chest examination. (8) Required to conduct an analysis of employees' health based on employees' records of medical surveillance. (9) Use of the ILO Radiological Classification of the Pneumoconioses mentioned. (10) The MBOD radiologists will interpret the films using the ILO Radiological Classification of the Pneumoconioses. (11) Tests include audiometry and other biological monitoring. (12) Diagnoses are entered by disease group (pneumoconiosis, cardiorespiratory tuberculosis, COPD, NIHL, heat disorders and other). (13) Occupational diseases, past or present, including severity.

Appendix 5.3: An abbreviated respiratory symptoms questionnaire

(Also consider which of the data in Appendix 5.2 you require to collect directly from employees)

I am going to ask you some questions, mainly about your chest. I should like you to answer YES or NO whenever possible. Please think carefully before answering.						
No.	Question				Yes	No	Don't know
1	**COUGH**: Do you usually have a cough on most days? (Count a cough with first smoke or on first going out doors)(Exclude clearing of throat)						
2	**PHLEGM**: Do you usually bring up phlegm from your chest? (Count phlegm with the first smoke or on first going out doors. Exclude phlegm from the nose. Count swallowed phlegm)						
3	**WHEEZING:** Does your chest ever sound wheezing or whistling?						
4	**CHEST ILLNESS**: During the past three years have you had any chest illness that has kept you away from work for as much as a week?						
	Has a doctor ever told you that you have? 4.1 Heart trouble 4.2 Bronchitis 4.3 Asthma 4.4 Pneumonia 4.5 Silicosis 4.6 Other chest trouble						
	4.7 If yes to 4.6, please specify other chest trouble (was it confirmed by a doctor & age at start?)						
5	**TUBERCULOSIS**: Have you ever had tuberculosis? If yes complete the following						
Episode no.	**Year**	**Doctor confirmed?**	**In lungs?**	**Affect heart?**	**Affect any other part of body?**		
					Yes	No	Don't know
6	**MEDICATION**: Do you take any medication? 6.1 If yes to above, specify condition/disease and medication.						
7	**TOBACCO SMOKING**: Have you ever smoked? (Yes = more than 20 packs of tobacco in your life or more than 1 cigarette a day for a year) 7.1 If yes, do you smoke now? (Yes = present smoker; No = Ex smoker)						
8. **Present smoker**					(Number)		
8.1 What was your age in years when you started smoking?							
8.2 How much do you smoke per day? Commercial cigarettes Hand rolled cigarettes Pipes							
9. **Ex-smoker**					Answer (Number)		
9.1 What was your age in years when you started smoking							
9.2 In the past, on average, how much do you smoke per day? (No. of) Commercial cigarettes Hand rolled cigarettes Pipes							
9.3 How old were you when you stopped smoking?							

Appendix 5.4. Reading forms for use in occupational lungs disease surveillance, including a short form for the ILO Radiological Classification of the Pneumoconioses

Symbol	Significance
0.	Technically inadequate film. Repeat film.
1 NAD	No abnormality detected. No specific action required
2a Pn new case 2b Pn as before 2c Pn with progression	Significant pneumoconiosis seen. Review past films for significant interval change. Classify film according to ILO short classification. (see below). Arrange for counseling of employees with new abnormalities. Refer for benefit examination if appropriate and not already done
3 TB as before	Signs consistent with tuberculosis seen Past films show no interval change No specific action required
4 TB Current	Signs consistent with tuberculosis seen Either no past films available or there is significant interval change Refer for diagnostic evaluation by TB/medical service. Refer for benefit examination if appropriate and not already done
5 Pn + TB as before	Signs consistent with TB and significant pneumoconiosis. Classify film according to ILO short classification. (see below) Manage as for (2) above
6 Pn + TB Current	Signs consistent with TB and significant pneumoconiosis. Classify film according to ILO short classification. (see below) Manage as for (4) above Refer for benefit examination if appropriate and not already done
7 Other	Other significant abnormality seen (e.g. pneumonia, pleural effusion or cardiac enlargement). Review past films for significant interval change. Refer for radiological diagnostic evaluation and/or referral to an appropriate medical service.

Date of X-ray plate:																
0/0	0/1	p	q	r	s	t	u	A	B	C	tba	tbu	cv	hi	es	hv
		1/0		1	2		3				ef	pla	plw	plc	em	oth

Symbols in the short ILO Classification

0/0	Pn not present.
0/1	Possibility that Pn present considered but rejected.
p	Small, rounded opacities < 1.5 mm in diameter.
q	Small, rounded opacities 1.5 to 3 mm in diameter.
r	Small, rounded opacities 3 to 10 mm in diameter.
s	Small, irregular opacities < 1.5 mm in width.
t	Small, irregular opacities 1.5 to 3 mm in width.
u	Small, irregular opacities 3 to 10 mm in width.
1/0	Pn present — insufficient small opacities to classify as category 1. Earliest sign of Pn.
1, 2 or 3	Small opacities present (refer to standard films).
A	Large opacity present with greatest diameter between 1 cm and 5 cm.
B	Large opacity or opacities present with combined area < right upper lobe.
C	One or more large opacities with a combined area > right upper lobe.

Abbreviations in the short ILO classification

tba	Active tuberculosis, probably
tbu	Tuberculosis — activity uncertain
cv	Cavitation
hi	Enlargement of hilar or mediastinal glands
es	Egg-shell calcification of glands
hv	Heart and vessels
ef	Effusion
pla	Costophrenic angle obliteration
plw	Pleural wall abnormality — diffuse thickening
plc	Pleural calcification (plaque)
em	Emphysema
oth	Any other pathology or abnormality not represented in the table, which should be described in words.