CHAPTER 3

Screening for toxic effects of workplace exposure

Screening primarily benefits the individuals who submit to testing. Criteria applicable to the evaluation of medical screening tests performed in community settings (Wilson & Jungner, 1968; Preventive Services Task Force, 1989; Braveman & Tarimo, 1994) and in the workplace (Halperin et al., 1986; Matte et al., 1990; Weeks et al., 1991) have been described in detail. In general, the following conditions should be considered before a screening test is adopted.

The disease:

— causes significant morbidity and mortality;
— can be identified at a pre-symptomatic stage before the individual would ordinarily seek medical care;
— responds to acceptable, available and effective intervention and treatment;
— is prevalent in the population undergoing screening.

Moreover, the test:

— is acceptable to those at risk for disease;
— has adequate sensitivity, specificity and predictive value in the target population;
— is available at a reasonable cost;
— is sufficiently standardized to be performed with consistency, accuracy and reproducibility.

If these conditions are satisfied, the screening test can be adopted. Note that there are a number of qualitative terms in this list. What is considered "reasonable cost" in the workplace may differ from what is considered "reasonable cost" in a community setting (Halperin et al., 1986). "Acceptable" interventions and treatments may also vary according to regional or cultural practice. An "adequate" level of sensitivity and specificity may depend on the severity of the condition and the
benefit achieved through intervention. In general, sensitivity should be
given priority over specificity in screening programmes.

In addition to focusing on appropriate diseases and identifying rea-
sonable tests to use in screening, the administrator or institution re-
sponsible for establishing a screening programme needs to address
other issues, including the availability of suitable personnel to adminis-
ter screening tests and interpret results and the presence of the appro-
priate equipment and facilities to perform, interpret and follow-up on
tests. The desired frequency of screening examinations should also be
determined.

The relationship between the natural evolution, in the absence of
intervention, of the disease for which screening is proposed and the
duration of previous and current levels of the exposure that causes
the disease should influence the timing of screening. Screening should
be conducted when there is an opportunity to detect the disease or
disease-related conditions. For example, if detectable disease indicators
do not occur until at least 10 years after first exposure, intensive screen-
ing could be delayed until that time, once baseline examinations
have been conducted. Knowledge of exposure levels would also influ-
ence the timing of screening, since some diseases progress more quickly
with high exposures. In such a case, however, knowledge of high
exposures should trigger action to reduce exposure levels. The course
of disease progression following the cessation of exposure should
also affect decisions about screening (e.g. after a change of job or
retirement).

It is also necessary to consider the appropriate dividing point be-
tween normal and abnormal test results for the screened population,
since often the difference is not clear-cut. This issue is considered more
fully below in the discussion of tests of pulmonary function.

It is also important to assess the level of risk, if any, from the testing
programme. Naturally, the risks and benefits of the tests that are per-
formed must be considered and weighed in light of the seriousness of
the disease being screened for.

Moreover, the perceived value of the programme to workers must be
taken into account. Screening programmes that are perceived to have
no value or benefit to the worker are likely to be poorly supported by
the target population. Numerous examples have been reported of medi-
cal information being used to deny employment or dismiss employees
without sound justification (Rothstein, 1984). Provisions should be
adopted to protect the privacy of workers and to ensure that the screen-
ing programme does not result in the inappropriate use of medical
data. Likewise, when screening is accompanied by educational pro-
grammes and concerted efforts to control harmful exposure, workers
are more likely to trust its intent. Lastly, screening programmes should be undertaken in light of the available resources.

The actions to be taken following abnormal test results should also be defined before the establishment of a screening programme. Some actions should always be taken:

- Confirmation of the test result. A screening test is the first level of evaluation; further evaluation may be required to confirm a positive screening test.
- Notification of the workers (in writing) of their test results and their medical significance. Workers should be informed of any legal implications of their test results, recommendations for changes in their work practices or exposure conditions, the predicted risk from continued exposure, the nature of any disclosure of test results to the employer and any sources of additional information about their medical condition. When removal from the work environment is medically recommended, notification of the workers should include personal counselling on options for alternative employment.
- Notification of the employer and workers of the aggregate results with personal information removed.

Additional actions and interventions may vary according to the judgement of the administrator of the screening programme as well as the social, economic, legal and political environment in which the programme is offered. Additional actions and interventions include the following:

- Workplace modification (i.e. redesign of the work process or changes in work practice) when a toxic effect is established or strongly suspected.
- Reduction of exposure for affected workers. This may be effected by changes in the work itself or by administrative controls such as job rotation. Occasionally the use of personal protective equipment may be warranted as a temporary measure.
- Worker and employer education.
- Medical treatment and counselling; periodic follow-up evaluations if the employee has disease without clinical manifestations.
- Notification of other workers in similar industries who are at risk for disease.
In addition to the selection and implementation of appropriate medical tests, an occupational medical service engaged in screening or surveillance activities should make provision for the programme components described below.

Qualified occupational health professionals

Programmes should be managed by health professionals with specific knowledge, training and experience in the disciplines of occupational health (toxicology, biostatistics, epidemiology, industrial hygiene and medicine) and who are knowledgeable about the relevant laws, regulations and rules. Moreover, the staff responsible for the medical testing, interpretation and communication of results, the analysis and reporting of data and quality assurance should have the skills and training appropriate for their roles. Training programmes that focus on a programme’s specific features are useful for orienting staff and updating their skills. Local laws and regulations may dictate the licensing and certification requirements for programme staff.

Record-keeping

The security and confidentiality of medical records should be maintained. They should therefore be separated from general personnel and employment records. The medical records should include comprehensive information on employee exposure, and should be kept current by means of periodic examinations. Because of the long latency between exposure and onset for most diseases caused by mineral dust, records should be retained for an extended period (perhaps 30 years) beyond the date of last employment. Records may be kept by the employer, government agencies, the worker or employee representatives.
Information from the record should be available to the worker, to his or
her health care provider and to public health authorities in a manner
consistent with local law.

Surveillance programmes should maintain records of occupational
exposure and medical screening so that data can be retrieved as needed
and comparisons over time and between categories of workers can be
analysed in a continual fashion.

Quality assurance

The efficient performance of medical testing requires ongoing and
systematic quality assurance in order to increase the accuracy and valid-
ity of test results and to promote the comparability of results over time.
The latter is critical for programmes of surveillance where group trends
are of particular interest. Records of all quality assurance activities
should be retained.

Confidentiality

Concerns about confidentiality, and the potential adverse consequences
that can result if personal medical information is inappropriately dis-
seminated, may discourage workers from accepting valuable testing
services. The *International code of ethics for occupational health profes-
sionals*, adopted by the International Commission on Occupational Health
(ICOH), provides the following guidance:

The obligations of occupational health professionals include pro-
tecting the life and the health of the worker, respecting human
dignity and promoting the highest ethical principles in occupa-
tional health policies and programmes. Integrity in professional
conduct, impartiality and the protection of the confidentiality of
health data and of the privacy of workers are part of these obliga-
tions ( ICOH, 1992).

The *Code of ethical conduct for physicians providing occupational medical
services*, adopted by the American College of Occupational and Environ-
mental Medicine (ACOEM), is similar:

Physicians should ... keep confidential all individual medical in-
formation, releasing such information only when required by law
or overriding public health considerations, or to other physicians
according to accepted medical practice, or to others at the re-
quest of the individual; recognize that employers may be entitled
to counsel about an individual's medical work fitness, but not to
diagnoses or specific details, except in compliance with laws and
regulations; communicate to individuals and/or groups any sig-
nificant observations or recommendations concerning their
health and safety... (ACOEM, 1994).

Those initiating or maintaining a programme of medical screening
or surveillance should be able to answer the following questions:

- What is the purpose of the programme: screening, surveillance or
  both?
- Who is responsible for the design, conduct and evaluation of the
  programme?
- What exposures are creating a health risk?
- What disease or condition is the target of the programme?
- Which workers are eligible for participation?
- Is the programme legally mandated or voluntary? If mandated, is
  legal enforcement tied to programme performance?
- Is worker participation in testing voluntary or mandatory?
- Which tests are performed?
- What is the frequency of each test? How soon after initial exposure
does testing begin? How long after cessation of exposure does testing
end?
- Who performs the test, and under what conditions?
- What constitutes an abnormal test result?
- What actions are taken as a result of an abnormal test result?
- Are the actions mandatory or voluntary?
- How (and when) will programme effectiveness be assessed?

It may be possible to answer only some of these questions during
programme planning; some it may be possible to resolve only after
initial programme evaluation. The use of pilot programmes may help
determine the most effective and efficient means to establish regular
and ongoing programmes.
CHAPTER 5

Diseases associated with exposure to selected mineral dusts

The principles discussed in the preceding chapters should be used to evaluate proposals for the screening or surveillance of workers at risk for disease due to mineral dust exposure. This chapter considers the effects, for which screening might be appropriate, of respirable crystalline silica, coal mine dust and asbestos dust. In the next chapter, the tests available for the detection of disease are reviewed.

The effects of mineral dust include the pneumoconioses, cancer, chronic bronchitis and chronic airflow limitation. A programme of prevention should ideally encompass all the adverse effects of mineral dust.

The pneumoconioses have long been the focus of health surveillance and preventive efforts because their cause is uniquely occupational. The term "pneumoconiosis" has been defined by the ILO (1976) as "an accumulation of dust in the lung and the tissue reaction to its presence". Most of the other dust-induced conditions described below are less easily distinguished from diseases with etiologies other than dust exposure, for example, exposure to tobacco smoke. Nevertheless, the well documented diverse effects of mineral dust inhalation require that broad preventive efforts be implemented (Becklake, 1985; Oxman et al., 1993; Becklake, 1994).

For the purpose of developing screening and surveillance strategies, the health effects induced by the inhalation of mineral dust will be discussed with reference to the disease characteristics listed in Chapter 3. Full clinical or epidemiological descriptions are beyond the scope of this book and should be sought from other sources, three of which are cited here (Merchant, 1986; Rom, 1992; Rosenstock & Cullen, 1994). Some of the medical conditions caused by the inhalation of mineral dust have also been reviewed in a recent ILO publication (ILO, 1991).
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Diseases associated with exposure to crystalline silica dust

Silicosis is the pneumoconiosis resulting from the fibrotic reaction of lung tissue to the deposition of inhaled crystalline silica dust. The risk of silicosis has been related to the amount of cumulative dust inhalation. The size distribution, surface characteristics and crystalline structure of silica particles may affect their toxicity. The silica nodule, which is a characteristic pathological entity, results from the pulmonary tissue’s response to inhaled and retained silica and is radiographically detectable. These nodules may coalesce with destruction of the intervening tissue and disruption of the normal intrathoracic architecture. This is known as progressive massive fibrosis.

Silicosis of rapid onset, occurring within a few years of the initiation of exposure, is sometimes referred to as accelerated silicosis. Silicosis with a more indolent progression, generally not identified until one or more decades after first exposure, is sometimes referred to as chronic silicosis. Accelerated and chronic silicosis most likely result from the same disease mechanisms, however, and differ only in their period of latency. Another condition, referred to as acute silicosis, may result from a different mechanism. It is a rapidly progressive disease which develops after the inhalation of high concentrations of fine silica particles. Acute silicosis has pathological characteristics similar to alveolar proteinosis.

All forms of silicosis can progress in the absence of continued exposure, and accelerated and chronic silicosis can both appear and progress after exposure ceases. Although silicosis is irreversible, there is some evidence that the likelihood of progressive impairment from either accelerated or chronic silicosis is diminished by the early identification of disease and the cessation of exposure (Westerholm, 1980). On the other hand, if acute silicosis occurs, interventions do not appear to influence favourably the outcome of the disease. Experimental therapies or interventions that are not widely available, such as whole lung lavage and lung transplantation, have been attempted for workers with acute silicosis and for some with total pulmonary incapacity due to progressive massive fibrosis.

The risk of tuberculosis is greater in workers with silicosis than in the general population. Moreover, it is probable that tuberculosis is more common in workers exposed to silica but without silicosis than in the general population. The risk of tuberculosis rises significantly in workforces exposed to silica where the background rates of tuberculosis are high. Tuberculosis can cause significant morbidity and mortality, particularly if unrecognized and untreated. While effective tuberculosis treatments are available, failures and relapses have been reported
more commonly in individuals with silicosis, though this may have changed with the use of modern treatment regimens (Cowie et al., 1989).

Bronchitis and chronic airflow limitation have also been shown to occur more frequently in workers exposed to silica than in the general population, and their risk is thought to depend on the total exposure to airborne contaminants in the workplace (Becklake, 1989). In general, these conditions cause significant morbidity and can cause premature mortality (Cowie & Mabena, 1991). The extent to which they cause significant levels of disease in the absence of silicosis has not been well studied. Early intervention with the control or cessation of adverse exposures (e.g. dust, tobacco smoke) may result in a reversal of bronchitis symptoms and presumably also diminish the rate of progression of airflow limitation.

Lung cancer resulting from silica exposure is currently the subject of intensive scientific investigation. The International Agency for Research on Cancer (IARC) has identified crystalline silica as a potential human lung carcinogen (IARC, 1987), and, in response to this possibility, NIOSH has recommended the greatest possible protection from silica exposure. Some investigators suggest that silicosis rather than silica exposure is the most important risk factor for cancer. In any event, as with lung cancer from asbestos exposure, there is no indication at present that lung cancers associated with silica dust differ from other lung cancers. No effective methods of early diagnosis leading to successful intervention have been identified. Because exposure to silica dust is widespread and lung cancer is a common cancer, even a small difference in the relative risk of lung cancer resulting from exposure to silica dust could imply a significant number of lung cancer cases of occupational origin.

Other conditions such as scleroderma and chronic renal disease have also been associated with silica exposure in some populations, although the overall levels of morbidity and mortality for these diseases are unlikely to be sufficient to justify active medical screening.

Diseases associated with exposure to coal mine dust

Coal workers’ pneumoconiosis (CWP) is one of the lung diseases arising from the inhalation and deposition of coal dust in the lungs and the reaction of lung tissue to the dust. CWP is a chronic, irreversible disease of insidious onset, usually requiring 10 or more years of dust exposure before becoming apparent on routine chest radiographs. It is characterized by macular and nodular pigmented lesions which may be visible
radiographically as small or large opacities in the lungs. When only small opacities are present, the condition is called simple CWP. Coal mine dust may contain varying proportions of crystalline quartz. CWP may be indistinguishable from silicosis on chest X-ray.

Progressive massive fibrosis (PMF), or complicated CWP, is the term used when radiographic opacities greater than 1 cm in diameter attributable to coal mine dust exposure are present. PMF has been associated with pulmonary incapacity and increased morbidity and mortality.

The risk of CWP has been related to cumulative exposure to coal mine dust. CWP, in some instances, progresses after the cessation of exposure, although this seems to be less common and less likely to occur than the progression in pneumoconiosis resulting from retained asbestos or silica dust. Chest X-ray abnormalities may be visible in the absence of symptoms. Although reduction or elimination of exposure to coal mine dust for workers with chest X-rays showing category 1 CWP (see Annex 3) would presumably result in decreased progression of the disease and fewer cases of PMF, statistical modelling based on a large data set from the United Kingdom suggests that the benefit of exposure reduction may be limited once any degree of CWP is present (Hurley & Maclaren, 1987).

Dust control strategies in a number of countries have been directed at preventing miners from developing category 2 CWP because of the increased risk of PMF associated with higher categories of CWP. In the United States, miners with category 1 CWP have been offered work in environments with a maximum dust exposure of 1 mg/m³ (time-weighted working-shift average). It was anticipated that this would result in the virtual elimination of PMF. The strategy has not been completely successful, however, in part because PMF can appear on an X-ray otherwise free of recognizable diffuse fibrosis or with only minimal fibrosis (ILO category 0 or 1) (Hurley et al., 1987; Jacobsen, 1990). The shortcomings of this approach to screening and intervention to prevent PMF are detailed in a recent review (Attfield, 1992).

Chronic bronchitis and chronic airflow limitation including accelerated loss of FEV₁ (Forced Expiratory Volume in 1 second) are more common in workers exposed to coal mine dust than in others with comparable tobacco-use habits. These conditions appear to be dose-related. Chronic bronchitis may be reversible if exposures are controlled early. Moreover, the rate of FEV₁ loss can presumably be diminished through a reduction or elimination of exposure, although the rate of loss that should trigger action and its optimal timing remain subjects of investigation. Chronic bronchitis, airflow limitation, CWP and emphysema all result from exposure to coal mine dust and may
occur in various combinations. The chest X-ray is not a reliable predictor of the likelihood of airflow limitation associated with dust exposure. In general, a diminished FEV₁ is associated with increased morbidity and premature mortality. Thus, dust exposure that does not result in the development of CWP or PMF may still cause or contribute to clinically significant airflow limitation (Marine et al., 1988).

Above-normal risk for the development of stomach cancer has also been associated with coal mine dust exposure (Ames & Gamble, 1983), although the level of increased risk is not great and the underlying population prevalence of this condition is generally low. Stomach cancers can potentially be detected in a preclinical stage through testing for faecal occult blood, and early surgical intervention may have a beneficial impact on mortality.

Diseases associated with exposure to asbestos dust

Asbestosis is a non-malignant lung disease caused by the inhalation and retention of asbestos fibres and the lung tissue’s reaction to these fibres. The occurrence of asbestosis depends on the cumulative level of respirable asbestos fibre exposure. In environments where levels of airborne asbestos are low, several years of exposure are required to produce asbestosis. Although studies of animal exposure reveal the onset of disease within months after first exposure, in humans the signs and symptoms of asbestosis generally do not appear for years, or even decades, following initial exposure. Once recognized, the disease can have an indolent or a gradually or rapidly progressive course, even in the absence of additional asbestos exposure. Asbestosis can cause incapacity or death. Dyspnoea on exertion is usually the most prominent symptom of asbestosis and may precede any abnormalities noted through medical testing. Other symptoms include coughing and chest pain. No treatment or intervention has been shown to diminish or reverse the progression of asbestosis once the disease is established. There is, however, some evidence that cessation of exposure may diminish the rate at which radiographic change progresses and presumably the rate at which pulmonary disability develops (Becklake, 1991; Becklake, 1992).

Pleural fibrosis, in the form of discrete plaques or diffuse thickening of the pleura, can occur concurrently with or independent of pulmonary fibrosis. One or more discrete areas of fibrosis (plaques) may be recognized along the diaphragm or along the lateral chest walls. The occurrence of pleural fibrosis increases with the time from first exposure and with the age of the worker. Diffuse pleural fibrosis, with or without
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blunting or obliteration of the costo-phrenic angle on chest X-ray, is less common than discrete plaques and is often associated with significant abnormalities in pulmonary function. Discrete plaques may also be associated with pulmonary dysfunction, even in the absence of radiologically determinable parenchymal disease (Schwartz, 1991; Ernst & Zejda, 1992). Pleural fibrosis may be present as an incidental finding on chest X-ray when there are no abnormal chest complaints. Pleural plaques that are visible on chest X-ray are quite common in certain occupations. For example, in shipyard workers and construction insulators, the incidence of pleural plaques often exceeds that of parenchymal radiographic change (Becklake, 1991). A common characteristic of these occupations is intermittent exposure to high levels of dust. In areas of high tuberculosis prevalence a determination of the etiology of pleural fibrosis by radiography alone may not be possible. Pleural disease is rarely of sufficient severity to justify intervention, although surgery has been used to treat diffuse pleural thickening in its advanced, disabling stages.

Benign pleural effusions lasting from weeks to months before spontaneous resolution occur in workers exposed to asbestos, often within the first two decades of initial exposure. These effusions are sterile and without malignant cells. Associated symptoms such as non-specific chest pain may be present. There are data that suggest an association between the development of diffuse pleural fibrosis and antecedent pleural effusions. The extent to which other morbidity may result from benign effusions is unknown. Although pleural effusions have been identified without associated symptoms, there is no clear "preclinical" stage to this condition, nor are there any reported studies evaluating the results of interventions.

Chronic bronchitis — defined as a persistent cough with phlegm — occurs more often in people who work in dusty environments than in those who do not when they are compared with others with similar smoking habits. This observation has been confirmed in some cohorts of workers exposed to asbestos (Ernst & Zejda, 1991). The association of chronic bronchitis with cancer or asbestosis in workers exposed to asbestos has not been well studied. If exposure to dust is eliminated in people with chronic bronchitis, it may, in some cases, lead to the resolution of symptoms.

Chronic airflow limitation due to the narrowing of small airways as a result of the inflammation and fibrosis of the respiratory bronchioles (demonstrable on spirometry as a reduction in mid-expiratory flow rates) is widely accepted as a manifestation of asbestos exposure, even in the absence of visible asbestosis on chest X-ray (Ernst & Zejda, 1991). Pure airflow obstruction, demonstrable as a reduction in FEV1, probably
also occurs (Becklake, 1989; Bakke et al., 1991). Some studies report FEV₁ deficits in asbestos workers with pleural fibrosis (Schwartz, 1991). Others have demonstrated an association between airflow limitation and increasing parenchymal fibrosis visible on chest X-ray. Controlled intervention studies capable of providing guidance on the benefit of intervening at various stages during the loss of FEV₁ are unlikely to be carried out, and there is a dearth of observational reports. Nevertheless, control of adverse pulmonary exposures is likely to reduce the rate of loss in the FEV₁ and thus diminish morbidity over time.

Respiratory tract cancers are the most common cancers associated with occupational asbestos exposure. These are pathologically indistinguishable from other respiratory tract cancers. Cancers of the lung are seen more frequently in workers exposed to asbestos than in others with comparable smoking habits; the same is true with respect to laryngeal and pharyngeal cancer. When cancers occur, their period of latency averages about 25 years from first asbestos exposure. Exposure to both asbestos and cigarette smoke puts workers at considerably higher risk for the development of cancers of the respiratory tract (Rothwell, 1992). There is evidence that the cessation of smoking diminishes lung cancer risk within a decade in individuals with previous asbestos exposure, as compared with those who continue to smoke. In some cohorts of workers exposed to asbestos, lung cancer appears to be confined to those who have already developed pulmonary fibrosis. In other cohorts, lung cancer has been documented in subjects without radiographically visible pulmonary fibrosis (Wilkinson et al., 1995). The fact that the development of both fibrosis and lung cancer is related to cumulative exposure to asbestos has precluded clear resolution of the controversy whether fibrosis is a necessary step in the development of asbestos-related lung cancer. It is possible that more than one pathological process can result in lung cancer.

Lung cancer is the most common malignant cause of death for men in many countries. Intensive efforts to detect its early indicators through frequent sputum cytology and chest X-ray analysis have failed to have a favourable impact on the morbidity or mortality of high-risk groups (Marfin & Schenker, 1991).

Malignant mesotheliomas of the pleura, peritoneum or mediastinum, which may all occur spontaneously, are presumed to result from exposure to asbestos fibres if there is a credible history of asbestos exposure and a reasonable latency period. Malignant mesothelioma has been reported in association with apparently minimal exposure to asbestos after many years of latency. The expected exposure-response relationship may not always be clearly demonstrable, perhaps because of the competing risk of other malignant and non-malignant diseases,
with shorter latency periods, that are evoked by higher exposures to asbestos. The period of latency between first exposure to asbestos and the development of mesothelioma is, on average, longer than that for bronchogenic cancer, averaging 30–35 years. Such tumours grow rapidly and are invariably fatal, at least with current therapeutic options (i.e. surgery and chemotherapy).

Cancers of the gastrointestinal tract, including cancer of the oesophagus, stomach, pancreas, colon and rectum, have been observed to occur more frequently in some, but not all, populations with heavy exposure to asbestos. The risk of cancers of the gastrointestinal tract from asbestos exposure appears to be less than the risk of lung cancer. In populations without asbestos exposure, gastrointestinal tumours (both benign and malignant) have been identified in preclinical stages through testing for occult faecal blood and sigmoidoscopy; early detection and surgical treatment appear to have a favourable effect on morbidity and mortality (DeMers & Parsons, 1994).

Other cancers, including certain lymphomas and kidney cancer, have been observed more frequently in a few populations exposed to asbestos. The interrelationships between multiple pathogenic exposures may account for the diversity of malignancies associated with asbestos exposure. The morbidity and mortality resulting from lymphomas and kidney cancer are far less than those of the other cancers linked to asbestos exposure. There have been no reported studies of efforts for the early detection and treatment of these conditions in workers exposed to asbestos.

The risk of developing any particular disease varies in working populations according to the type of work performed, even when all workers are exposed to the same types of asbestos fibres, and exposure levels are taken into account. Patterns of disease also vary in relation to the particular type of asbestos fibre present in the work environment. The specific toxicity of fibre types is an active area of scientific investigation. Regardless of the additional information that may thus be yielded, it is clear that exposure to respirable asbestos fibres of all types must be considered hazardous. In many work settings (such as construction, repair and demolition), materials often contain a mixture of fibre types, and useful information about the specific composition is generally lacking. In such circumstances, prudent practice dictates that exposed workers should be afforded the highest level of health protection. Furthermore, various synthetic fibres are currently used in some work processes as substitutes for asbestos. The more closely these fibres resemble asbestos (in size, shape, durability and surface characteristics) the more likely they are to cause disease and thus the more intense control and surveillance efforts should be.
CHAPTER 6
Tests for detecting diseases induced by exposure to mineral dust

The tests commonly used to detect diseases related to mineral dust exposure include questionnaires, chest radiographs, spirometry and physical examination. Other tests include sputum examination, other imaging techniques, other measures of lung function, bronchoscopy, skin testing for tuberculosis and stool examination for occult blood (Balmes, 1990). A number of biological markers are under investigation with respect to their relationship to the occurrence of diseases caused by mineral dust.

Conventional chest radiography (X-rays)

Chest radiographs are the most important means for the detection of pneumoconioses (asbestosis, silicosis and coal workers’ pneumoconiosis). As described above, responses to dust inhalation vary with the mineral inhaled. These differences are also reflected in the patterns of abnormality that are characteristically present on radiographs.

A standardized method of X-ray interpretation (see Annex 3) published by the International Labour Office (ILO) is often used to recognize and classify the pneumoconioses (ILO, 1980). With this method, opacities resulting from inflammation, dust deposition or scarring are classified according to their shape, size, location and profusion. Profusion is determined by comparing the worker’s film with standard films distributed by ILO. Some exposures, such as to coal mine and crystalline silica dust, can result in the development of opacities greater than 1 cm in diameter. These are classified according to size. There are also conventions for classifying pleural abnormalities as well as for noting the appearance of changes suggestive of certain other diseases.

The ILO system was originally established to improve disease detection and achieve consistency in film interpretation during health sur-