MEDICAL SURVEILLANCE OF ASBESTOS WORKERS AND THE REQUIREMENTS OF THE NEW OSHA STANDARD

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Introduction

The Occupational Safety and Health Administration published Final Rules in the Federal Register on Friday, June 20, 1986, entitled "Occupational Exposure to Asbestos, Tremolite, Anthophyllite and Actinolite," setting standards amending the existing regulations (29CFR 1910.1001). The new standard reduces employee exposure to asbestos to 0.2 fibers per cubic centimeter of air (f/cc) averaged over an 8-hour day. A departure from the previous standard is the establishment of an action level of 0.1 f/cc TWA-8. If this level is exceeded employers must begin compliance activities such as air monitoring, employee training and medical surveillance. Separate standards have been developed to apply to general industry (including maritime industries) and to the construction industry.

This paper will discuss medical surveillance as required by the two standards and attempt to determine the rationale for the various techniques which are incorporated in this regulation. The historical facts which need to be considered will be reviewed and used to develop the ~~critique~~ review of the medical surveillance requirements of the amended standards.

Historical Background

It is stated in the literature that the asbestiform minerals have long been used by man and that their use can be traced back to the ancient Greeks. The Greek geographer Pausanias discussed lamps of gold with incombustible wicks of "Carpathian flax" made in approximately 430 BC. The Romans used woven asbestos cremation cloths and the French Emperor, Charlemagne, is reputed to have impressed his enemies by passing an asbestos tablecloth through fire to cleanse it.

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"The fact that asbestos is a hazard to man's health was recognized quite early. In the first century, both Pliny the Elder, the Roman naturalist, and Strabo, the Greek geographer, wrote of a sickness of the lungs in slaves whose occupation was the weaving of asbestos into cloth."

The modern asbestos industry dates from the discovery in the 1870s of large deposits of chrysotile fibre in Canada and Russia. Chrysotile is the fibrous form of serpentine. This fibre is white in colour and is long and flexible, making it ideally suited for textile uses. Shorter varieties of chrysotile are used in cement products, brake-lining and friction materials manufacture and in composite products which have a multitude of uses. Shortly after the introduction of chrysotile into industrial use three other fibres derived from amphibole minerals were commercially exploited, namely crocidolite, amosite and anthophyllite. Perhaps the most important application of the asbestiform minerals was fireproofing of buildings and ships.

The first recorded health effects from exposure to asbestos dust in the English medical literature appeared in 1900. Asbestosis is a twentieth century disease. It was Dr. Montague Murray who drew attention to the death of an asbestos-exposed worker from pulmonary fibrosis in the absence of any evidence of tuberculosis and who subsequently recorded that a number of the deceased's colleagues had died from a similar condition. Cases continued to be reported in the medical literature, the first comprehensive report on the pulmonary fibrotic effects of asbestos being published in Great Britain by Merewether and Price in 1930. As a result of their recommendations the Asbestos Industry Regulations, 1931, were promulgated and the medical surveillance of asbestos workers was required to be carried out by the Silicosis Medical Boards, now known as the Pneumoconiosis Medical Panels. Asbestosis became a compensable illness in the United Kingdom in 1931. The first report of asbestosis cases in the United States of America was made by Pancoast et al in 1917. ~~Further~~ Additional cases were reported into the 1930s. North Carolina industries figure prominently in the U.S. ~~epidemiologic~~ literature on the subject with reports by Shull in 1936 and Dressen et al in 1938. These investigators reported cases of respiratory impairment and various degrees of "asbestosis" among the groups of workers investigated. Dressen's study was conducted on behalf of the U.S. Public Health Service and the permissible exposure limit of 5 million particles per cubic foot of air was based on their conclusions. This limit remained in effect until 1971 when the newly established Occupational Safety and Health Administration promulgated a 12 fibers/cc temporary Standard. A permissible exposure limit of 5 fibres per cubic centimetre of air averaged over an 8-hour period was permanently established in 1972. This standard reduced to 2 fibres per cubic centimetre of air 8-hour TWA in 1976. Medical surveillance was required by this standard for persons exposed to airborne concentrations of asbestos dust. An action level to trigger the medical surveillance requirements was not incorporated in the standard but was unofficially regarded by OSHA as 0.1 f/cc TWA8.

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About fifty years after the commercial exploitation of asbestos started, the first suspicions arose that lung cancer deaths in asbestos workers might be attributable to their occupation. Case reports in the USA and UK in 1935 sparked the interest in this subject. It was the same Dr. Merewether in the UK who found that lung cancer deaths occurred in 13% of certified cases of asbestosis diagnosed by the Silicosis Medical Boards, whereas none were associated with silicosis cases . In 1955 the landmark study of asbestos textile factory workers in Rochdale, Lancashire, England, was published by Doll and demonstrated a definite excess of lung cancer deaths in male workers exposed for more than ten years prior to the introduction of the Asbestos Industry Regulations, an excess in men employed for twenty years or more since that time and no excess in others with briefer lengths of employment. The findings of Merewether in 1945 were confirmed by Buchanan in the UK in 1964 when he found that approximately 60% of certified asbestosis cases died of lung cancer. It is unfortunate that smoking histories were not obtained for these cases and that no attempt was made to compare the cases with a control population.

Dr. Selikoff's studies of various exposed worker populations in the United States drew the world's attention to a number of previously neglected facts. Until the publication of his findings in asbestos insulation workers in New Jersey, the risk to other than factory workers had not been demonstrated and the disastrous effects of cigarette smoking combined with exposure to airborne asbestos fibres had not been realized to its full extent. Table 1 illustrates the overwhelming effect of smoking in combination with asbestos exposure in the etiology of lung cancer.

In 1960 Wagner et al published their findings which associated diffuse malignant mesothelioma of the pleura with exposure to asbestos. Although the first case of malignant mesothelioma in an asbestos worker is said to have been observed by Leicher in 1954, Wagner was the first to conclusively demonstrate an association between exposure to asbestos and this invariably fatal tumour. Wagner also introduced a new concept into the developing understanding of the asbestos-related diseases, namely that of indirect exposure. He found cases of mesothelioma in young persons whose exposure was to fibers which they inhaled when strapped to their mothers' backs while they hand-cobbed pieces of asbestos bearing rock. Cases also developed in the nearby mining town where people were exposed to fibers from the roads which were made from asbestos mine tailings or from the school playing area which was made from the same material. Wagner's work also led to the worldwide development of an interest in determining whether different fibre types produced differences in the observed effects of exposure to them. There is a considerable body of evidence that in the crocidolite mining areas which Wagner studied, in shipyards in the UK which used substantial quantities of crocidolite from World War I until the early 1960s, and in people who manufactured crocodolite containing gas masks during World War II in Britain, this tumour appears more commonly than in mining areas or manufacturing operations involving the use of chrysotile asbestos.

Malignant mesothelioma of the peritoneum was associated ~~some years~~ in the early 1960's with exposure to asbestos. A controversy still exists as to whether some cases of ovarian cancer in women with a history of asbestos exposure were in fact peritoneal mesotheliomata or true cases of ovarian cancer.

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Amosite asbestos (the fibrous variety of the cummingtonite-grunerite mineral series) may be largely responsible for the cases of malignant mesothelioma which have developed in shipyard workers and factory workers in the United States.

Other forms of cancer have been said to be associated with asbestos exposure. Selikoff has demonstrated a slight excess incidence of gastro-intestinal cancers in his study population. Elmes in Belfast also reported an excess of deaths from G-I cancers in insulation workers and so did Newhouse in London. The original cohorts of asbestos textile workers studied in Britain by Merewether and others, exposed mainly to chrysotile fibers, have not shown a similar excess. As Elmes' follow-up of his population progressed, the excess of deaths from G-I cancers declined over time, ~~possibly~~ indicating the effect of improving controls.

Because the 2f/cc standard was designed to protect workers from developing the "earliest signs" of asbestosis and did not take into account the development of lung cancer, mesothelioma and other asbestos-related conditions, it came under progressively increasing criticism from many quarters. In 1975 OSHA published a notice of proposed rule-making to revise the standard because the agency believed that "sufficient medical and scientific evidence has been accumulated to warrant the designation of asbestos as a human carcinogen." The proposal would have reduced the 8-hour time-weighted average to 0.5f/cc and imposed a ceiling limit of 5f/cc for 15 minutes. OSHA based its arguments very heavily on the theory that there was no safe threshold level demonstrable for a carcinogen and that the OSHAct therefore required that the PEL be set at a level as low as technologically and economically feasible. This philosophy was rejected by the Supreme Court in the benzene decision in 1980 and resulted in a further delay in the revision of the 1972 Asbestos Standard.

In 1983 OSHA issued an Emergency Temporary Standard for Asbestos but it was invalidated by the US Circuit Court of Appeals for the Fifth Circuit in 1984.

Before concluding these remarks about the historical background of the asbestos-related diseases, it is necessary to define and briefly discuss two findings which are frequently referred to in reports about asbestos exposed subjects. Lung tissue from asbestos workers contains unusual beaded, dumb-bell shaped or rod-like particles which were originally mistakenly called asbestosis bodies because they were thought to occur only in asbestosis cases. They can be found in sputum and in lung tissue of the general population, larger numbers being present in persons with known occupational exposure and decreasing in number as asbestos exposure possibilities decline. The name asbestosis bodies gave way to asbestos bodies when this was realized. These particles owe their golden colour to the fact that they contain iron and, for this reason, and because some authorities believe that the fibre core may be made up of materials other

than asbestos, in some instances they prefer to call them "ferruginous bodies." There is considerable evidence that most ferruginous bodies are in fact asbestos bodies. Asbestos bodies are the "tombstones" of asbestos fibers and are also found in the absence of disease, thus providing an index to exposure but not having any other diagnostic significance.

The second finding commonly discussed in the literature and probably reported as the most frequent radiographic abnormality in asbestos-exposed workers in the United States today is involvement of the pleura, manifesting itself as localized pleural plaques or as diffuse thickening (pleural fibrosis). Cases of transient pleural effusions also occur in asbestos-exposed people. Pleural plaques have been associated with non-occupational exposure as well as occupational exposure to asbestos. When pleural plaques are seen on both sides of the chest the clinician's suspicion should be aroused to the possibility that they are asbestos-related. Plaques are another indicator of past exposure. They used to be regarded merely as an index to past exposure and not of significance in producing impairment of lung function, but it has become clear from studies conducted over many years on workers in British Naval Dockyards that persons with these radiographic findings may be more prone to subsequently developing asbestosis or may even be at increased risk of developing malignant mesothelioma.

MEDICAL SURVEILLANCE

In the UK, the Silicosis and Asbestosis (Medical Arrangements) Scheme, 1931, made initial and periodic medical examinations obligatory for workers employed in the process of breaking, crushing, disintegrating, opening or grinding of asbestos, and the mixing or sieving of asbestos or any admixture of asbestos, and in the making of asbestos textiles and insulating mattresses. It was believed that these examinations "may be of great value as a means of preventing or arresting the development of asbestosis."

Asbestosis was made a compensable disease under the Asbestos Industry (Asbestosis) Scheme, 1931.

In their 1930 Report Merewether and Price recommended "the control of the disease by periodical medical examinations of the workers by which those unfitted by health reasons are prevented from entering the industry, and cases of fibrosis and pulmonary tuberculosis are detected at the earliest possible moment. The ultimate and only reliable test of the effectiveness of the preventive measures adopted in the industry will be found in the statistics derived from the records of periodical medical examinations of workers....." These words of Merewether and Price remain true today but unfortunately their hopes for the establishment of a statistical data base were never realized and nowhere in the world is it possible to find information of a standardized nature on the incidence of asbestos-related disease in an entire national population.

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In the examination of asbestos-exposed workers, the following has been the traditional approach:

- o Medical history with emphasis on the respiratory system;
- o Chest x-ray pre-placement and periodically thereafter;
- o Physical examination with emphasis on the respiratory system.

The introduction of pulmonary function tests did not occur routinely until the late 1950s and proved most valuable in assessing fitness for work and also in determining disability for compensation purposes.

Assuming that lung function is normal at the time of pre-employment evaluation, the continued follow-up of a worker by means of pulmonary function testing should allow any deterioration to be measured and steps to be taken to remove the worker from any further exposure.

There is no adequately documented evidence to indicate that impairment of lung function can regress on removal from exposure to asbestos although some such claims have been made. Lung function impairment may take one of two forms in the early stages of asbestosis, either reduction in breathing ability, that is to say ventilatory capacity, or impairment of the process of gas exchange in the lungs. In either case the condition may progress to include the other at a later stage or it may become complicated by the changes in function which are associated with bronchitis or emphysema. The impaired ventilatory capacity may be due to diminished lung expansion (restrictive syndrome) related to the fibrotic scarring of the lungs, which is the basic pathological process occurring in asbestosis or it may be due to obstruction or narrowing of the airways (obstructive syndrome). The latter is not usually the presenting lung function abnormality associated with pulmonary fibrosis but may be present if it is complicated by bronchitis or emphysema.

Impairment of gas exchange when it occurs in asbestos workers may be the result of the thickening of the walls of the air sacs (alveolae) in the lungs and destruction of the pulmonary blood vessels associated with diffuse interstitial fibrotic changes. The ability of oxygen to diffuse into the venous blood and thus re-oxygenate it is impaired by the alveolar wall thickening, and may be further impaired in the presence of tissue destruction and abnormal distribution of pulmonary ventilation. The capacity to transfer gas across the alveolar-capillary membrane is known as the diffusing capacity or gas transfer factor. This can be measured by means of a test known as the carbon monoxide diffusing capacity obtained by the single-breath method. Measurement of the carbon monoxide diffusing capacity (T_L or D_L) requires sophisticated equipment and is not suitable for use in the routine medical surveillance of asbestos workers.