RECENT ADVANCES IN OCCUPATIONAL HEALTH

Edited by J.C. McDonald

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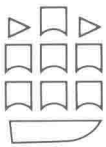

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Recent Advances in
OCCUPATIONAL HEALTH

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Preface

The first volume on any subject in an established series is a special challenge to the editor. The field of occupational health is so wide, there is no previous edition against which to distinguish recent advances from current practices and, as always, topicality is no guide to importance or progress. To ease the choice, many more, shorter, reviews are presented here than is usual, imposing on the contributors perhaps too great a brevity.

Live to work or work to live, productive labour is close to the heart of things in all societies. The implications and consequences for health cannot be avoided; deliberately, casually and even unknowingly, each community makes its cost-benefit judgements. The main sections which follow reflect the prime importance of the job that has to be done, the increasing refinement of methods for scientific study, improvements in equipment and procedures for worker protection and the changing emphasis of social concern.

Evidence will be found of several current anxieties — energy and food production, occupational 'asthma', unexpected dangers in our largest industry (health care), environmentally induced cancer, and the vexed question of mineral fibres. Asbestos and, by extension, other natural and man made materials of similar structure have been a preoccupation of occupational health in the 1970s. This problem, important in its own right, is a recurrent theme in many chapters, not by accident or editorial bias, but simply because it has provided a challenge for most aspects of occupational health practice. It has led to improved methods of epidemiological and laboratory study, to more critical evaluation of personal and group methods of protection and, above all, to sharply focused attention on social, scientific and political responsibilities, as related to safety at work.

A first priority is to make the workplace safe and free from risk of disease; most chapters outline advances being made towards this goal. Less urgent but as important is the need to increase work satisfaction as a prerequisite for a full and healthy life. That only two chapters are concerned with this more positive approach is some reflection of present values.

The world today is an anxious place, and for good reason; moreover, the news media and communication systems see to it that no cause for alarm need pass unnoticed. This, more than social idealism, may help to explain why occupational health issues are often in the headlines, particularly in countries where high standards of hygiene and safety prevail. Fortunately, the advances which are being made in workers' health are better balanced than the newspaper accounts. Even so, the relationship between extent and severity of problems and the research and control effort devoted to them is less close than might be.

The gross occupational diseases of the past are no longer obvious in western

societies but the same cannot be said of the poorer countries, comprising three quarters of the globe. In the developing world the impact of industrialization can only be guessed; although lessons may have been learned from others' experience, the pace, pressures and magnitude of this second far larger industrial revolution are surely exacting a high price. But, without increased productivity to break the vicious cycles of poverty, neither health nor any other human right can be achieved. It ill behoves wealthy nations to complain of unfair competition from developing countries which cannot yet afford the highest environmental health and safety standards. By far the largest problems in occupational health today are in the developing countries. It also reflects on our social and scientific priorities that only one chapter in this book is concerned with these problems and this does more to underline their gravity than to report advances in control.

No other area of health activity depends so much on the contribution of many disciplines. Virtually every science, art and human skill is involved. Even among the 33 authors of this book, not every important speciality is represented. With such variety and so few limits, can occupational health claim any real identity? Hopefully, the structure and content of this book may help convince the reader that this discipline is indeed more than the sum of its parts; if not, the fault is mine.

London, 1981 J.C.McD.

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1. The mineral fibre problem

J. C. Wagner P. C. Elmes

For many years interest in the biological effect of mineral fibres was mainly confined to the commercial types of asbestos. Recently many other fibrous minerals have been recognised as potentially dangerous pollutants of the environment. The majority of these materials are naturally occurring, others are synthetic. The natural fibres are either specifically exploited for commercial purposes or else occur as atmospheric contaminants which are released during mining or tunnelling operations. Industry has been developing other mineral fibres as a substitute for asbestos to meet an increasing need for cheap and reliable materials for reinforcement, friction products and insulation. The latter demand has been emphasised by the present fossil fuel crisis. Minerals being exploited for a variety of purposes other than insulation and reinforcement are known to consist of fibres or elongated crystals, for example, some clays and some zeolites. Thus, these minerals can be considered under the following groups:

1. Asbestos minerals
   a. Of commercial value
   b. As potential environmental contaminants
2. Synthetic mineral fibres
3. Other naturally occurring fibrous minerals

ASBESTOS

Asbestos of commercial value

Practically all the knowledge that is available about hazards associated with the inhalation of fibrous mineral dusts has been obtained in studies of asbestos. Asbestos consists of six naturally occurring minerals: chrysotile, crocidolite, amosite, anthophyllite, tremolite and actinolite. Chrysotile is a member of a group of minerals referred to as the serpentines and is composed almost exclusively of magnesium in combination with silica. It has a sheet structure which curls to produce hollow tube-like fibres. The other five are members of one mineralogical group referred to as the amphiboles. They are very similar in crystal structure, being chain silicates, but they vary in chemical composition. Crocidolite and amosite are iron-rich varieties, anthophyllite is a magnesium rich mineral, while tremolite and actinolite contain a large amount of calcium together with magnesium.

The annual world production of asbestos in 1976 was 5×10^9 kg, of which 97 per cent was chrysotile and the remainder crocidolite and amosite. The commercial production of the other three amphiboles has been on a small scale in the past, but they are important as contaminants of other minerals and agricultural soil.

Chrysotile is widely distributed, with the largest production from the Ural

mountains in Russia, Quebec and certain other parts of Canada, Zimbabwe and Swaziland in Southern Africa, the Italian Alps and Cyprus. Crocidolite is now mined almost exclusively in the Cape Province in South Africa, and until recently in Western Australia. Amosite is only exploited in the Transvaal, but deposits have been discovered in Southern India.

Uses of asbestos

Asbestos has over a thousand uses (Hendry, 1965) so that the number of occupations in which exposures may have occurred is large. Crocidolite, because of its resistance to acids and sea water, was extensively used in naval insulation and fire proofing, as insulation for steam locomotive boilers, and later for sound proofing in passenger coaches. A significant amount was used in the insulation of buildings. The use and importation of crocidolite into the United Kingdom has been severely limited since 1969 (Health and Safety Executive, 1970). Amosite has been used for thermal insulation, in floor tiles and in the superstructure and insulation of ships. Chrysotile has been used for all other purposes, but particularly in asbestos cement products, insulation, fire proofing and in the manufacture of friction materials such as brake-linings and clutch plates. Chrysotile is still the main fibre used in textiles but this is no longer a major section of the industry. The destruction, or demolition, of buildings, ships and railway rolling stock are sources of environmental pollution.

Sequelae of exposure to asbestos dust

There have been numerous descriptions of the lesions resulting from exposure to the different types of asbestos fibre, e.g. Wagner (1979). The sequelae are:

1. Asbestos bodies and fibres in the sputum and lung
2. Pleural plaques and diffuse pleural fibrosis
3. Interstitial pulmonary fibrosis (asbestosis)
4. Cancer of the lung
5. Diffuse mesothelioma of pleura and peritoneum
6. Cancer of the gastro-intestinal tract and possibly of the larynx.

1. ASBESTOS BODIES AND FIBRES IN THE SPUTUM AND LUNG

Asbestos bodies consist of an iron-mucoprotein complex which surrounds the asbestos fibres after inhalation and retention in the lungs. These bodies usually have bulbous ends giving the appearance of drumsticks. Initially, these coated fibres were known as asbestosis bodies suggesting that their presence was indicative of the presence of the disease, but it soon became clear that they were at most indicative of occupational exposure to asbestos. Later, as more sensitive means of detection were developed by Thomson et al (1963), and later by Pooley (1975), it was realised that these bodies can be found in the lungs of practically everyone who has lived in an urban environment. Under the scanning electron microscope it can be shown uncoated fibres are far more common in material extracted from macerated lung tissue, the ratio of fibres to asbestos bodies being more than 1000:1 and varying with fibre type. In spite of the fact that Gross et al (1969) and Pooley (1975) demonstrated that these bodies can occasionally form around other fibres besides asbestos, there has been a tendency among research workers studying atmospheric pollution to state that all fibres and bodies are asbestos without carrying out tests for precise identification.

With the development of more sophisticated techniques it is now obvious that a correct estimation of the number of fibres in tissue or environmental samples can only be obtained by examination under a transmission electron microscope, otherwise the large number of fibres of less than 0.5 μm in diameter will not be observed. The crucial question of the amount, size and type of fibre found in tissue which can be related to the diseases which will be described later, cannot be stated with confidence at this stage. In macerated specimens of dried lung 10^6 fibres per gramme can be found without evidence of disease; in cases of asbestosis the count is usually over 10^8. With a light microscope seldom less than 250 000 fibres per gramme lung tissue are found in cases of asbestosis.

2. PLEURAL PLAQUES AND DIFFUSE PLEURAL FIBROSIS

The presence of circumscribed areas of fibrous thickening below the mesothelium on the lower portion of the chest wall, over the diaphragm or on the pericardium are characteristic of exposure to fibrous mineral dusts. These plaques may be extensive, are leaf-shaped, often bilateral and have an irregular embossed surface. They consist of woven collagen fibres and as they mature become acellular and avascular. This avascularity leads to necrosis and sometimes to the gradual deposition of calcium in the lesions (Meurman, 1966). It can take 20 years or more for sufficient calcium to be deposited for the plaques to become radio-opaque and visible on chest radiography. Therefore, the plaques are seen much more frequently by the pathologists at necropsy than by the radiologist. In some cases there is generalised pleural fibrosis, leaving the lungs *en cuirasse* completely sheathed in a thick layer of fibrous tissue. Unlike plaques, generalised pleural thickening can restrict the expansion of the lungs and cause breathlessness.

3. ASBESTOSIS

Asbestosis is a slowly progressive and persistent interstitial fibrosis of the lung associated with the inhalation of asbestos dust and characterised by asbestos bodies and fibres in large numbers in the tissue. If sufficient dust has been retained, the individual lesions in the alveoli join up until the individual acini become linked in a fibrous mesh, the process starting at the base of the lung and gradually spreading upwards. This process is fairly well established before there is recognition on radiological or by physiological examination, the latter often being obscured by the effects of cigarette smoking. If exposure has been sufficient the disease will progress after the worker has left the industry (Becklake et al, 1979).

4. CANCER OF THE LUNG

Carcinoma of the bronchus is a frequent cause of death among workers with radiological evidence of asbestosis (Liddell and McDonald, 1980). The risk of a cigarette smoking worker heavily exposed to asbestos developing lung cancer is 25 to 50 times greater than an age matched non-smoker who has not worked with asbestos (see J. C. McDonald, 1980). Initially the carcinomas reported in the asbestos workers were peripherally situated, with adenocarcinomas being the most common (Buchanan, 1965). With the increase in cigarette smoking, all types of endobronchial tumours are being seen, but the number of adenocarcinomas is still more frequent than in non-exposed cigarette smokers (Kannerstein and Churg, 1972).

5. DIFFUSE MESOTHELIOMA OF PLEURA AND PERITONEUM

Diffuse mesotheliomas of both pleura and peritoneum are now accepted as being frequently associated with exposure to asbestos dust. Occasional case series were recorded in the European and American literature from about 1910 on (see McDonald and McDonald, 1977), but the first series associated with asbestos was reported by Wagner et al (1960). This observation from the crocidolite mining area in the Cape Province in South Africa has now been confirmed in most industrial countries throughout the world. In many cases the exposure is for as little as six weeks and there is no radiological or pathological evidence of pulmonary fibrosis. Recently, Newhouse and Berry (1976) have shown that there is a 'dose' relationship in the development of these tumours. Environmental exposure was a common finding in the cases from the Cape asbestos fields, and has also led to the occurrence of mesotheliomas in the vicinity of the mine in Western Australia (Hobbs et al, 1980), but not in other asbestos mining areas. The tumour has also been observed in family contacts of asbestos workers probably due to fibres brought home on clothing or hair. Cases have resulted from this type of exposure in early childhood. The time between first exposure and diagnosis of the tumour is usually 20 to 40 years, irrespective of the age at first exposure. Elmes and Simpson (1976) described the clinical aspects of mesothelioma in a large series of cases. The possibility that the general public is at risk from asbestos in the ambient air has been raised on many occasions but there is no good evidence that this type of exposure alone has caused a mesothelioma.

6. OTHER CANCERS

A significant increase in cancers of the gastro-intestinal tract has been reported from studies in New York, Quebec, Belfast and London (Hammond et al, 1965; McDonald et al, 1971; Elmes and Simpson, 1971; Newhouse, 1973). Carcinoma of the larynx, first reported by Stell and McGill (1973) has not been generally confirmed (e.g. Newhouse et al, 1980); the association seems to be more with heavy cigarette smoking.

Problems arising from exposure to asbestos dust

BIOLOGICAL EFFECTS OF DIFFERENT TYPES OF ASBESTOS

Experimental studies have shown that all types of asbestos are capable of producing pleural plaques, asbestosis and malignant tumours. Asbestos bodies are formed around amphibole fibres and, as Pooley (1972) illustrated, the concretions that develop on chrysotile are irregular in distribution and are deposited on clumps of fibre. Inhalation studies with rats (Wagner et al, 1974) demonstrated that after exposure to equivalent dust clouds in the respirable range the amount of chrysotile retained in the lungs was far less than the amphiboles, but the resulting fibrosis was the same. As the dusts used in this study were the highly respirable UICC standard samples prepared by Timbrell and Rendall (1971/2), these findings do not necessarily apply to occupational exposure.

The major controversy with regard to variety of asbestos concerns the mesotheliomas. Our original South African evidence indicated a clear association with crocidolite, and in Southern Africa this is still valid (Webster, 1973). In Britain, as in other industrial countries, most asbestos workers have been exposed to more than one type

of fibre; apart from mining areas, pure exposure is rare. South African experience with crocidolite has been repeated on a smaller scale at Wittenoom in Western Australia, where mesotheliomas have occurred, both in those employed in the mines and in the environmentally exposed population (Hobbs et al, 1980). Nothing comparable has been reported for chrysotile, amosite or anthopyllite mining. The gas mask workers investigated by Jones et al (1976) and some of those by McDonald and McDonald (1978) appeared to have had a pure exposure. Pooley's analysis of the lungs of the Nottingham cases also showed significant amounts of chrysotile, but not more than is found in autopsy material generally. The technique developed by Pooley (1975) for the identification of asbestos and other mineral fibres in lung tissue is the most useful method available for identifying individual exposures and the complexity of the situation gives emphasis to the need for the parallel examination of appropriate controls. The comparisons made between the fibres in the lungs of the mesothelioma cases and controls in Britain by Jones et al (1980) when compared with those obtained by A. D. McDonald (1980) in the United States and Canada has shown that chrysotile fibres are found in considerable quantity equally in cases and controls. In Britain, crocidolite and to a lesser extent amosite were associated with mesothelioma, whereas in the USA it was predominantly amosite and less often crocidolite. Selikoff et al (1972) found a considerable excess of mesotheliomas in factory workers exposed to amosite but has not reported on the fibre content of their lungs. The Advisory Committee to the Secretary of State for Employment (Health and Safety Commission, 1979) concluded that in the causation of mesotheliomas, crocidolite was more dangerous than chrysotile but that amosite might be intermediate between the two.

Experimental evidence has complicated the situation by showing that many types of mineral fibre can cause mesothelioma. This evidence has come in the main from intrapleural implantation studies which were initiated by Wagner (1962) and continued in collaboration with Stanton and Wrench (1972); Pott et al (1972) undertook similar intraperitoneal investigations. This work has indicated that the size of the fibres was more important than their nature.

IMPORTANCE OF FIBRE SIZE

The significance of the physical characteristics of fibres in explaining the biological effects of asbestos was first emphasised by Timbrell (1965). He demonstrated that diameter was the most important factor in determining whether a fibre would be inhaled. The finer the fibre the more easily would it reach the lung parenchyma. Later, Timbrell et al (1970) showed that this could be applied to the amphiboles and that the ultimate diameter of crocidolite was less than that of amosite. Although the individual fibres of chrysotile have a diameter less than that of crocidolite, they occur in a woven coil formation, the total diameter of which affects its aerodynamic behaviour. Thus chrysotile behaves as a coarse fibre and finds difficulty in reaching the pleural surfaces through the peripheral airways. However, chrysotile fibres in aqueous solution can divide longitudinally into fibrils which under some circumstances are straight and have similar properties to very fine amphiboles. The typical electron microscopic appearance of asbestos fibres is shown in Figure 1.1.

Calculations and experiments with casts of the lower respiratory tract showed that fibres up to 3.0 μm in diameter would reach the respiratory bronchioles. The length of fibres most likely to cause fibrosis would appear to be greater than 10 μm (Timbrell