The Pneumoconioses

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CHAPTER 2

Asbestosis

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Historians have recorded the fact that the ancient world was well aware of the peculiar properties of the asbestos fibre, chiefly its resistance to fire and the ability of the fibres to be spun into cloth. In Plutarch we read about the vestal virgins who had lamps with "perpetual" wicks; elsewhere we find reference to lamps with incombustible wicks made from "Carpathian flax," derived from a mineral found on islands near Cyprus. Pliny refers to the "funeral dress of kings" which apparently were shrouds of woven asbestos used in funeral ceremonies of the nobility. When Marco Polo travelled through Siberia he found a fossil substance which "When woven into cloth and thrown into the fire, remains uncombustible."

The term "asbestos" is generally used to describe several fibrous magnesium silicates which are different in their chemical composition and physical properties. The most important types of fibres are chrysotile, amosite, crocidolite, anthophyllite, actinolite and tremolite. Deposits of various types of this mineral are found in many countries, but the largest mines are located in Canada, Africa and Russia. About 90 per cent of the fibres produced today are of the chrysotile variety.

Open pit or underground mining methods are employed to obtain the asbestos fibre. The fibre-containing rock is transported to the mill where it is dried, crushed and the rock separated from the fibre by screening methods. The fibres then are opened, cleaned, graded and bagged for shipment. It is estimated that there are between 12 to 15 thousand people in North America employed in the production and primary manufacturing of asbestos. No estimate has been made of the number of people who handle finished asbestos products.

Individual chrysotile fibres are white, but when seen together as they occur in the veins of serpentine rock their colors vary from green, to yellow-green, to amber. Amosite fibres usually are yellow-

brown and are mined only in South Africa. Crocidolite is known as the "blue asbestos" and occurs chiefly in South Africa, although some is found in Australia and South America.

As reported by Badollet[1-2] chrysotile, amosite and crocidolite are most commonly used by industry today. Figures 1, 2, and 3 are enlarged electron micrographs of these three types of fibre. Chrysotile fibre is long, soft and silky while amosite and crocidolite fibres are shorter, more stiff and more brittle than chrysotile.

Asbestos fibres are highly resistant to heat and acids. They have great tensile strength and large surface areas. Because of these properties as well as their filamented structure, industrial use of these fibres throughout the world is increasing. The textile industry has used them for many years to produce blankets, clothing, threads, ropes, tapes, braided tubing, and filters. In recent years, however, there has been an increasing use of asbestos in the insulation, building and friction-material trades. In addition, the fibres can be found

Fig. 1. Electron micrograph, chrysotile asbestos; x 4000.

Fig. 2. Electron micrograph, amosite asbestos; x 4000.

in paper, wallboard, shingles, pipe covering, floor tiles, brake linings and brake blocks, cements, putties and plastics.

In the Canadian and American asbestos industries the various mining, milling, and manufacturing operations create some dust containing asbestos fibres. If the fibres up to 50 μ in length are inhaled continually and in sufficient quantities over a period of several years, a typical pulmonary fibrosis will develop. It has been stated that this fibrosis is due not to the chemical but rather to the mechanical action of the fibres.[3] The asbestos fibres are deposited in the terminal bronchiles, initiating a tissue response which coats the fibre and eventually produces what is known as the asbestos body. This appears to be a defense mechanism of the lung. Numerous asbestos bodies can be found in the sputa of individuals who have had only short and sporadic exposure to the dust. These persons are healthy and have no demonstrable signs or symptoms of asbestosis.

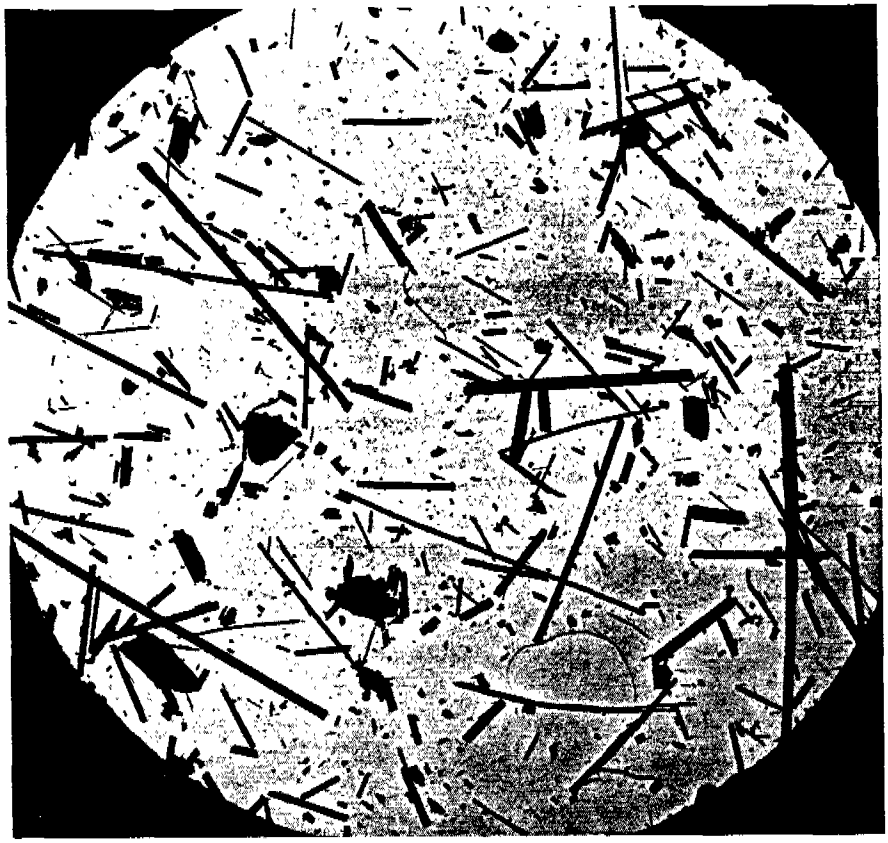

Fig. 3. Electron micrograph, crocidolite asbestos; x 4000.

Therefore, it seems more appropriate to use the term "asbestos" bodies rather than "asbestosis" bodies, signifying exposure to the fibres but not necessarily indicating disease. In addition, the finding of occasional asbestos bodies, singly or in clumps in pulmonary tissue does not definitely indicate generalized parenchymal asbestosis.

If increasing quantities of the fibres are continually inhaled, the tissue reaction progresses, and a generalized, diffuse fibrosis gradually appears throughout the lower lobes of the lungs. With additional exposure, this fibrosis will spread to the other lobes, eventually causing respiratory embarrassment and finally cardiac failure.

There is no allergic response of the skin or respiratory tract membranes to contact with the asbestos fibre. Thus poisoning or similar toxicological effects of the fibre should not be considered.

The theory of mechanical irritation is interesting. Will the asbes-

tos fibre, implanted in muscle tissue which is motile, produce the same response as that in the lung? If it is implanted in tissue which is immobile, such as the brain, what will be the response? It is known that brucite, a fibrous mineral but not a silicate, can cause a pulmonary fibrosis in experimental animals similar to asbestosis.[3] But, because brucite is not a silicate, it would seem that some reaction, other than chemical, is responsible for the fibrogenic response of the asbestos fibre.

The pulmonary fibrosis resulting from prolonged inhalation of asbestos fibre will produce a typical X-ray pattern. In early, or first-stage asbestosis, the X-ray shows a fine diffuse homogeneous infiltration throughout both lower lung fields (Fig. 4). It should be noted

Fig. 4. Early asbestosis.

Fig. 5. Moderately advanced asbestosis.

that this infiltration is bilateral, that it is generalized at both bases, and that the nodular or conglomerate patterns of other pneumoconioses, such as silicosis, are not seen in asbestosis. There is a considerable amount of pleural reaction associated with this disease, which may account for the "ground glass" pattern which has been used to describe the typical X-ray picture. We do not know why this pleural thickening is so marked in advanced stages of asbestosis and is not seen similarly in other pneumoconioses. Asbestos bodies and asbestos fibres have not been identified within this thickened pleura.

In moderately advanced, or second-stage, asbestosis, the infiltration has increased but still is confined to the lower lung fields (Fig. 5). The "ground glass" pattern is more apparent, and the heart borders are becoming indistinct or shaggy. There is some irregularity of the diaphragmatic outlines and beginning obliteration of both the cardiophrenic and the costophrenic angles.

In far-advanced, or third-stage, asbestosis, the infiltration still is homogeneous and bilateral, has spread to the middle and possibly the upper portion of the lung fields, but the apices remain clear (Fig. 6). The cardiac outline is almost completely obliterated, as are the

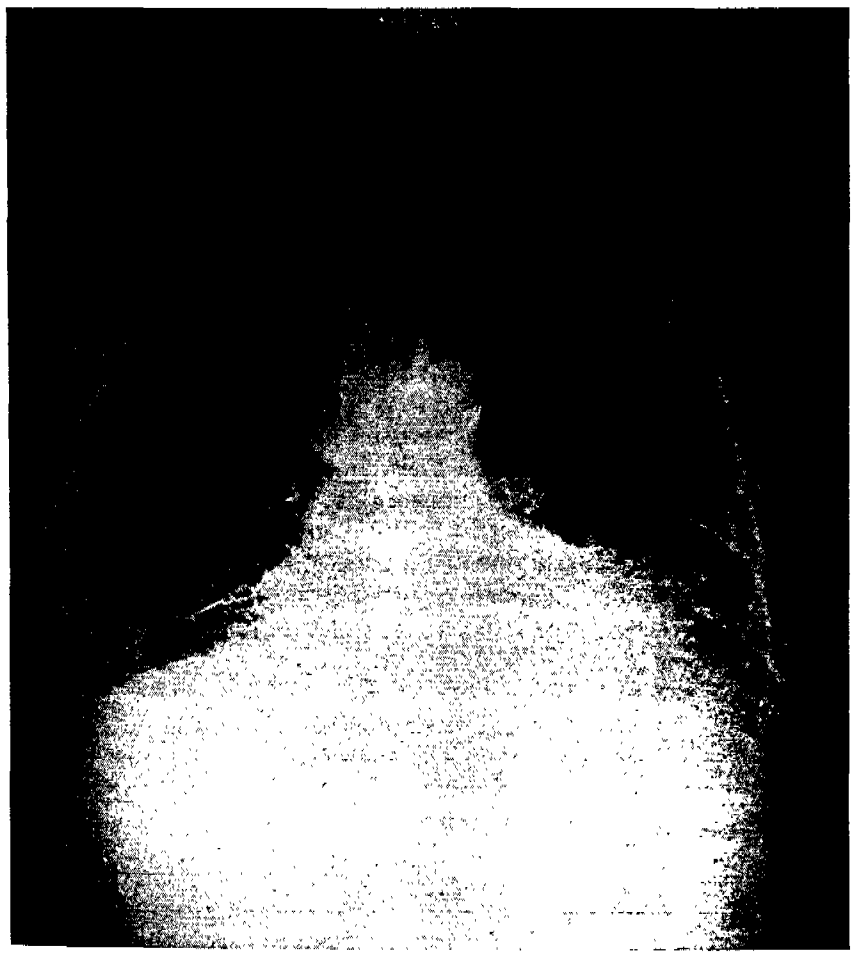

Fig. 6. Far advanced asbestosis.

domes of the diaphragm and the costophrenic sulci. With this picture in mind, it is advisable to reiterate the observations of many physicians, namely, that the X-ray picture should never be used to estimate the presence or the extent of impaired pulmonary function or disability. Many cases with X-ray evidence of third-stage asbestosis have been known to carry on their usual work and live fairly comfortable lives for several years. On the other hand, no case of definite disability has been seen unless there was the typical X-ray pattern of asbestosis. X-ray changes typical of asbestosis seldom are seen unless there has been a period of at least ten years of continuous exposure.

Schepers[4] has reported X-ray readings of massive fibrosis with coalescent lesions mainly along the upper mediastinum and in the apices. These X-ray patterns are not seen in the American and Canadian asbestos workers who have been exposed only to the fibre and no other potentially toxic dusts. The cases reported by Schepers may have occurred in people who were exposed to asbestos fibres of a different type, with much greater concentrations and lengths of exposure than seen in North American workers.

In some cases it has been suggested that the appearance of calcified pleural plaques on the X-ray, coupled with a potential exposure to asbestos fibre are pathognomonic of asbestosis. Reviewing a quarter century of serial X-ray films, the author noted calcified pleural plaques appeared rarely in several thousands of workers in an asbestos mine in Canada. The fibre from this mine has been used for a great many years in numerous American plants. Cases of asbestosis have occurred in this mine and in these plants, but in only one of the plants is there any evidence of pleural plaque formation. If inhalation of the asbestos fibre alone could cause these plaques, one would expect to find plaques wherever the fibre is used. In view of the above-mentioned evidence the casual relationship of pleural plaques to other pulmonary and other cardiac conditions in addition to asbestosis is worthy of investigation.

As previously indicated, industry today is finding many new uses for the fibres when they are mixed with other substances. It is an established fact that when asbestos fibres are mixed with silica, diatomaceous earth, or other potentially toxic dusts, the pulmonary changes resulting from the inhalation of these mixtures are not typical of asbestosis. The X-ray pattern may be different, the clinical course changed, or the susceptibility to intercurrent infection increased or decreased. Thus, in making a diagnosis of occupational pulmonary disease, it is highly important to obtain a detailed occu-

pational history, so that asbestosis, silicosis, or mixed pneumoconioses can be differentiated (Figs. 7 and 8).

There is no typical clinical picture for asbestosis. The disease is insidious in its onset and slowly progressive with continued inhalation of the fibre. There is a gradual increase in cough and expectoration, some anorexia and weight loss, then slowly increasing dyspnea. Cyanosis and clubbing of the fingers are rare findings.

Asbestos warts or corns occasionally are seen on the hands of those who handle the pure asbestos fibre, but are not found in those who finish asbestos products. No cases of malignancy associated with

Fig. 7. Mixed dust pneumoconiosis due to asbestos and silica.

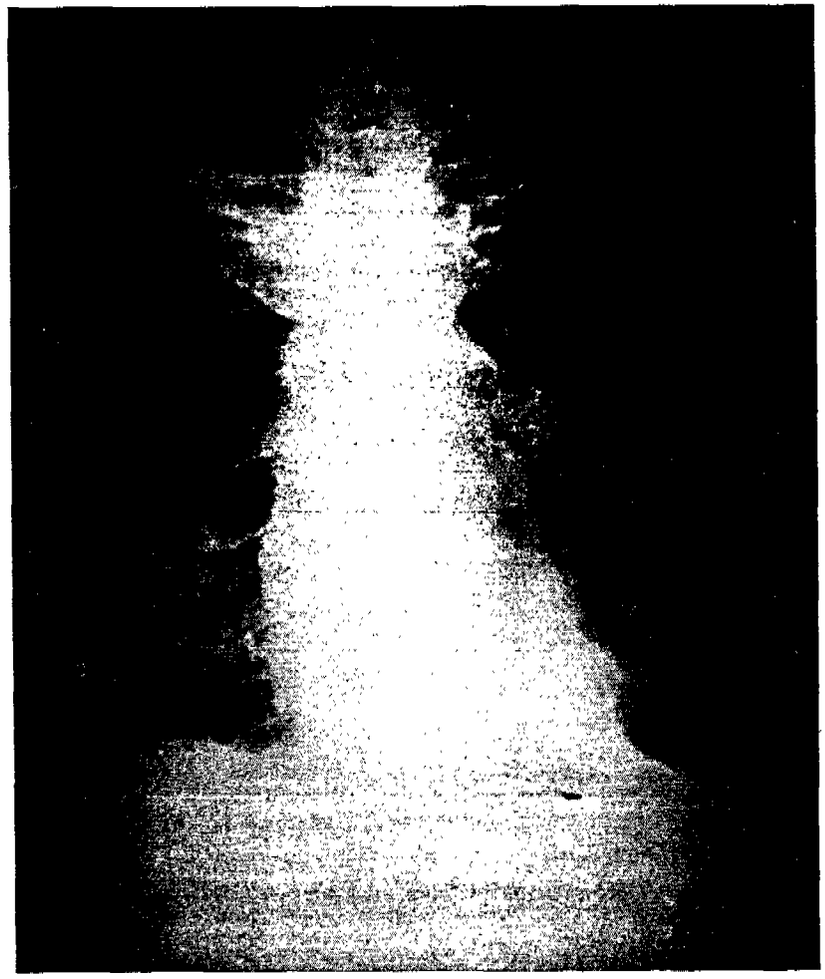

Fig. 8. Mixed dust pneumoconiosis due to asbestos, silica, and coal.

asbestos corns among North American asbestos workers have been recorded in the author's experience.

For industrial hygiene purposes a Maximum Allowable Concentration, or Threshold Limit Value, of 5 million particles per cubic foot has been established by the American Conference of Governmental Industrial Hygienists. This M.A.C. has been widely accepted for many years by industry and most state organizations. However, in every sample of air-borne asbestos dust there will be traces of

inert particulate matter, such as serpentine rock, in addition to the fibre. No standard definition has been made to distinguish the particulate matter from the fibre.

Most industrial hygiene procedures specify impinger sampling and light-field counting techniques. However, these procedures do not distinguish between the standard Greenburg-Smith Impinger and the Midget Impinger. There are some who believe that the impingers fracture the asbestos fibres and so do not reflect the true picture of the work environment. Other instruments such as the electrostatic and thermal precipitators, the konimeter and membrane filters have been used. Unfortunately, no comparison studies have been made to evaluate the relative efficiency of these instruments in the sampling of asbestos fibre. Consequently, no data is available to compare one industrial exposure with another with any degree of accuracy.

There is evidence that asbestosis will not progress after exposure ceases, but this seems to be true only if the worker does not develop an intercurrent pulmonary infection. Asbestos workers are not predisposed to develop more intercurrent pulmonary infections than are found in other workers.[5] However, when an acute pneumonitis develops in the presence of an established asbestotic fibrosis, the infection is slow to heal, relapses are frequent, and the patient may be more susceptible to subsequent pulmonary infections. While it is true that the disease is slow and insidious in its onset and that people with advanced asbestosis may lead relatively quiescent normal lives, eventually the heart begins to fail, and death from cor pulmonale rapidly follows. It has been thoroughly established by animal experiments and clinical experience that asbestosis does not predispose an individual to the development of pulmonary tuberculosis, nor does it aggravate an apparently healed tuberculous lesion.[5]

Gregoire[6] has reported that pulmonary function studies on asbestos workers have shown that the chief physiological problem is that of a "tight" lung. The vital and maximum breathing capacities are lowered, expansion of the lung is difficult, and arterial oxygen saturation of the blood is diminished in some cases, indicating an impairment of gas transfer through the lung. Diffuse obstructive emphysema, frequently seen in silicosis, is not apparent in asbestosis. In addition, bronchiectasis is not a common finding in North American asbestos workers.

It is imperative when discussing any disease that clear and concise terms must be used. This is especially true in the case of occu-

pational diseases because of the serious Workmen's Compensation implications. The term "asbestos worker" has been used too freely in recent years by many physicians. When alluding to an asbestos worker one would naturally assume that the individual used asbestos fibre in his occupation. Any physical disability in this person then might be attributed to his inhalation of the asbestos fibre.

Unfortunately, the term "asbestos worker" very frequently is used to denote a member of the asbestos workers union, a group of people whose chief occupation is the application of insulating materials. Many years ago, the asbestos fibre was the only insulating material commonly used. Today, mineral wool, glass fibre, magnesia, and a host of other products are used in the insulation industry, but the applicators still are called "asbestos workers" because of their union affiliation. Hence, when the literature presently available is reviewed, it would be advisable to ascertain the true occupational job title.

Some observers in the past few years have associated various conditions such as lung cancer, bronchiectasis, emphysema and bronchitis with the asbestos worker. No attempt was made to associate the disease with the clinical, radiological and pathological entity, known as asbestosis. The occupational title of the individual was sufficient to associate a certain disease with the asbestos fibre. Clear-thinking and diligent authors have associated other pulmonary diseases with asbestosis. It is unfortunate that less accurate authors have generalized their comments and thereby confused intelligent readers. Perhaps with more accurate reporting the alleged association of inhalation of asbestos fibre and other intercurrent disease conditions would be clearer today. It is entirely possible that the asbestos fibre may be a carcinogen, or a co-carcinogen. However, the true relationship will remain obscure until responsible reporters can evaluate the relationship between agent and disease and not just job title and disease.

This author's experience has extended over a period of eighteen years, during which time over 50,000 physical examinations and chest X-ray films have been observed among workers in the asbestos mining, milling and fabricating operations. Among these workers, who were exposed only to chrysotile fibre, there were no more cases of pulmonary malignancy than among the general population.

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