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Asbestosis

REPORT OF THE SECTION ON NATURE AND PREVALENCE
COMMITTEE ON OCCUPATIONAL DISEASES OF THE CHEST
AMERICAN COLLEGE OF CHEST PHYSICIANS

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Asbestosis

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DEFINITION

ASBESTOSIS IS A DIFFUSE PULMONARY disease in which fibrosis is the dominant reaction to the prolonged retention in the lung of asbestos fibers.

ASBESTOS

Asbestos is of mineral origin. The name asbestos derives from the Greek word for "unquenchable." The term embraces at least 30 silicate compounds which characteristically have a fibrous structure. Their chemical constituents include: silicon, magnesium, iron, fluorine, manganese, aluminum and chromium in various molecular states and hydrated to different degrees.

Of the 30 or more asbestiform minerals, only six have current economic significance. They are: chrysotile, crocidolite, amosite, anthophyllite, tremolite and actinolite. Chrysotile is a fibrous form of serpentine, the other five are amphiboles.

The major physical properties of asbestos are its incombustibility, its facility to be separated into filaments of fibers, its unusual flexibility and its high tensile strength. Other valuable and characteristic properties include resistance to heat, to moisture and to the corrosive action of acids. These properties vary considerably with the different types of asbestos. The size and flexibility of the fibers and some chemical attributes of asbestos are characteristics with medical implications.

SOURCES

Canada, Russia, South Africa and Rhodesia are the main producers of chrysotile. Crocidolite is found in South Africa, Australia, South America and Italy. Amosite is mined in South Africa while anthophyllite is found in Finland, United States, New Zealand, China, Japan and India. Asbestosis has been reported from all these countries.

INDUSTRIAL USES

The industrial uses of asbestos are increasing steadily and now exceed 3,000 specific conditions in which asbestos serves uniquely as a protectant against excessive heat, cold and corrosion. There are, therefore, a multiplicity of circumstances under which asbestosis can be encountered.

PROCESSING OF ASBESTOS

Because asbestos is found both on or near the earth's surface as well as at considerable depths below it, the ore can be recovered by open pit or underground mining. The fact that 15 to 20 tons of serpentine rock must be mined in order to produce 1 ton of usable asbestos fibers means that in many asbestos mining operations, workers are exposed to serpentine as well as to asbestos dust even to a ratio of 95 per cent to 5 per cent.

Asbestos fibers are recovered by mechanical milling consisting essentially of successive stages of crushing and separation by air screening. The fibers are further separated, cleaned, graded according to length and then bagged for shipment.

The asbestos fibers are used afterwards in the weaving of fiber resistant textiles and in the manufacture of reinforced molded cement products where they are submitted to further mixture with other constituents according to different specifications.

INDUSTRIAL HYGIENE ASPECT

In determining the dust hazard, both the total count of asbestos particles* in the working environment and the prevalence of fibers of different lengths should be considered.

It is generally accepted that the risk of contracting asbestosis is highest where a

*Particles used in this context includes fibre

greater proportion of the dust particles is fibrous and in those operations in which most of the processing is done in the dry state.

In spite of the fact that the total dust exposure may be higher in the asbestos mining industry, the health hazard is more serious in the asbestos textile industry. The explanation for this is that in mining 80 per cent to 95 per cent of the dust may be serpentine rock dust, which is biologically relatively inert.

The wet stage of the asbestos cement industry yields a low risk, but the dry mixing of asbestos with cement or dry cutting of finished asbestos products are sources of asbestos hazard.

There is no agreement on safe limits for asbestos dust and the best evidence for this is that several governmental agencies have recommended 5,000,000 asbestos particles (of any length) per cubic foot of air as the maximal allowable concentration, some industries have adopted as their standard 1,000,000 asbestos fibers of 5 microns and greater length per cubic foot, and a third group of hygienists considers a count of 10,000,000 total dust particles per cubic foot as a safe limit. Techniques of dust counting vary considerably and need to be standardized. The most practical method is the membrane filter device. The most accurate but least practical methods are the thermal and electrostatic precipitators. The Konimeter and Impinger methods are unreliable. Currently the value of the Tyn-dallometer and photoelectric methods are being explored.

MEDICAL ASPECTS

(a) Toxicology:

Asbestos is not currently considered a toxic substance since it does not produce systemic poisoning. Thus far, no distinctive blood or urine deviations have been detected among workers exposed to asbestos. No minimum lethal dose has been established for asbestos.

(b) Allergy:

Asbestos dust has not been shown to be allergenic, either in occupational groups or in experimental animals.

(c) Pathogenesis:

Two theories of causation have gained acceptance in different quarters. These are, respectively, mechanical irritation by asbestos fibers and fibrogenicity of the protein capsule of the asbestos body.

The mechanical irritation concept is supported by the observations that fibrosis occurs most densely in the parts of the lungs where the respiratory movements are greatest, or where the lung impinges against other intrathoracic structures; that grinding the fibers to less than 5 microns in length decreases their capacity to evoke fibrosis in experimental animals; and that a fibrous mineral containing no silicate causes pulmonary fibrosis in experimental animals.

The chemical theory postulates an unknown fibrogenic agent which is released by disintegration of asbestos bodies. This theory takes into account the delayed development and progressive nature of asbestotic fibrosis, and is based on the assumption that there may be a correlation between dosage of exposure and the elapsed time after exposure.

(d) Pathology:

The principal pulmonary lesions include regional atelectasis, interstitial, mucosal and focal fibrosis, and bronchiolar fibrosis. Distortion, distention or stenosis of bronchi and fine diffuse emphysema may be associated with the fibrosis and become in such instances additional factors in the genesis of the respiratory disability. Perivascular fibrosis is also observed in most cases of asbestosis contributing significantly to the development of cor pulmonale. The disease process is dispersed throughout the lungs, but the lesions may be more accentuated subpleurally and at the bases of both lungs where the amplitude of the breathing movements is greater. No asbestotic fibrosis has ever been found in the mediastinal lymph nodes. Many alveolar spaces may contain asbestos bodies which

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consist of asbestos fibers surrounded by a proteinaceous capsule. Their presence, although indicative of asbestos exposure, is not necessarily diagnostic of asbestosis.

Asbestotic fibrosis may concentrate around focal lesions such as inactive tuberculosis or a bronchiectasis. This fact must be kept in mind in assessing the severity of asbestosis through biopsy.

Pleural plaques have been detected by many observers in the United States of America, Canada, Finland, Germany and South Africa. In many individuals and perhaps even the majority of cases, these pleural plaques do not present any histologic lesions of asbestosis and contain no asbestos fibers. Some plaques calcify early and are localized on the parietal pleura leaving the visceral pleura unaffected. After many years, however, irritation by a parietal plaque can evoke a lesion of the opposing visceral pleura. These plaques, though apparent on x-ray, are not associated with any disability. They should, however, be distinguished from the serious form of subpleural fibrosis and from pleural mesothelioma, both of which can cause significant limitation of pulmonary excursions.

RADIOLOGY

The radiologic pattern of asbestosis is characterized by a ground glass appearance and sometimes a fine stippling. The cardiac silhouette may also be blurred. If there has been some concurrent exposure to dust other than asbestos (as in mining), the pattern may be much coarser. A single standard therefore does not apply. The I.L.O. radiographic classification is quite unsuitable for grading the type or severity of asbestosis. The complications of asbestosis, of course, have their own distinctive radiologic patterns.

PULMONARY PHYSIOLOGY

Function studies of the conventional range have not shown good correlation between the radiologic signs, the intensity or duration of exposure and pathology discovered through biopsy or at necropsy.

Arterial oxygen saturation may stay normal even in advanced asbestosis. On the contrary, more often than not, arterial oxygen desaturation may precede the radiologic manifestations. Deficient gaseous diffusion across the alveolocapillary membrane may in some cases be the only abnormal finding. Impaired ventilation and reduced pulmonary elasticity are often found in advanced asbestosis. In some instances of asbestosis, there is a long latent period between the establishment of positive radiologic signs, the first demonstrability of physiologic disturbance and the ultimate clinical outcome of respiratory failure. Additional sequelae or complications may supervene to initiate the disabling phase of the disease. Intercurrent factors, such as aging or coincidental pulmonary infections, may upset the respiratory balance.

SEQUELAE AND COMPLICATIONS

Acute and chronic right-sided heart disease and progressive cardiorespiratory failure are the most frequent terminal sequelae of asbestosis. An association between asbestosis and tuberculosis has never been proven conclusively by any statistical or epidemiologic investigation and has been disproved in experimental animals. Active tuberculosis may heal and not become reactivated despite continuous asbestos dust exposure.

In the medical literature, there are more articles favoring a positive relationship between cancer of the lung and asbestosis than denying it. While it has been reported that there may be an enhanced prevalence of pulmonary neoplasia in some asbestos industries (e.g. crocidolite or amosite), or in some locations (e.g. South Africa, England), this does not appear to apply for the chrysotile industry in North America. This comment applies both with respect to intrapulmonary new growths and to pleural mesothelioma.

In contrast to other pneumoconioses, asbestosis is relatively infrequently complicated by bronchitis, bronchopneumonia or

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pneumonia. In far advanced asbestosis, asymptomatic noninflammatory bronchiectasis may, however, occur as a result of distension of bronchi and bronchioles by contraction of intervening fibrous tissue. Hypertrophic or obstructive pulmonary emphysema is not a feature of asbestosis. Between zones of fibrosis, however, alveolar spaces may become distended and distorted. This anatomic condition may be identified through biopsy or at necropsy, but is not usually associated with a clinically disabling equivalent.

PREVALENCE OF ASBESTOSIS

Apart from inequalities of the dust exposure in different industries, many other circumstances can explain the great variation in the alleged incidence of asbestosis. Reports of prevalence range from 8 per cent to 77 per cent in different industries or different countries.

The criteria for the diagnosis of asbestosis are far from uniform. The application of different radiologic criteria, for instance, may significantly influence the incidence rate as reported by separate radiologists. The method of tabulating the cases is a factor. In the same industry, an incidence of 24 per cent among the workers exposed to the dust can become only 8 per cent when the same cases are diluted among the total number of employees of the whole industry. The pathologist, who sees only deceased cases, or the consultant who reviews chiefly problem cases, are likely to have more pessimistic views of the disease than the clinician, who has under his care both those who become ill and those who develop limited disease only or who may even escape the asbestotic reaction despite significant exposure.

Even the more or less official statistics issued by different Compensation Boards have to be accepted with caution before being compared. Often the liberal interpretation of the aggravating factor clause, or the according of benefit of the doubt and other social considerations, will give

impressions of incidence which cannot be reconciled with authoritative medical opinions.

In order to have a prevalence rate which will be more representative and more exact, it would be necessary to collate the data which may be gathered from a small random sampling of pathology of deceased asbestos workers. Even such a study would only yield data applicable to the industry in question and similar investigations would be needed for each type of industry.

The divergent and even the contradictory opinions circulating on the different aspects of asbestosis and the numerous unanswered questions have created an embarrassing confusion for many of those concerned with the scientific and the practical problems of asbestosis.

DIFFERENTIAL DIAGNOSIS

Asbestosis can be simulated by any of the numerous varieties of diffuse interstitial fibrosis. For diagnosis, precise information is needed concerning the occupational exposure. When dealing with a group of industrial employees and when serial radiographs and clinical records are available, diagnosis may not be too difficult. In isolation, asbestosis can often only be recognized through biopsy or at necropsy. The finding of asbestos bodies in the sputum is indicative only of prior exposure and not of asbestosis.

PREVENTION

Asbestosis can only be prevented by avoiding prolonged inhalation of high concentrations of asbestos particles. Aluminum inhalations are of no benefit, as with silicosis.

TREATMENT

No specific treatment exists. However, considerable benefit may derive from treating symptoms or complications. It is better to keep asbestotic subjects active and ambulant as long as feasible. Prolonged immobility may lead to serious pulmonary restriction.

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COMPENSATION

In many states and countries, asbestosis is a compensable occupational disease. In some areas compensation is based on partial disability and in others is limited to total disability.

COMMENT

Asbestotic subcutaneous granulomatosis and asbestotic cutaneous verrucous acanthosis have been reported in occasional industries. Heavy exposure to some types

of asbestos dust may cause itching especially in zones of contact between clothing, wristbands, etc. This is due to superficial penetration of coarse fibers and has no clinical significance.

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