

*Staphylococcus aureus*, despite the absence of that organism in cultures of the patient's sputum. Microscopic examination of the cyst wall revealed fibrous tissue and chronic inflammation. There were multiple healed necrotic foci and the adjacent lung showed severe bronchiolectasis. There was no fragment of normal bronchial architecture in this cyst. These clinical and morphologic observations substantiate the diagnosis of post-infectious pneumatocele in this patient.

Although there is inadequate documentation of this child's initial illness, the author believes the evidence presented supports the conclusion that she had a chronically persisting post-infectious pneumatocele which is known to have existed for three and probably for six years.

## REFERENCES

1. WATKINS, E. JR. AND HERING, A. C.: "The Management of Staphylococcal Lesion Pneumatoceles by Intracavity Tube Drainage," *J Thor Cardio Surg*, 36:642, 1958.
2. SABISTON, D. C., JR., HOPKINS, E. H., GOODE, R. E. AND BENNETT, J. L., JR.: "The Surgical Management of Complications of Staphylococcal Pneumonia in Infancy and Childhood," *J Thor Cardio Surg*, 38:421, 1959.
3. FISHER, A. M., TEEVER, R. W., CURTIN, J. A., SCHULTZ, G. AND MILLER, D. F.: "Staphylococcal Pneumonia: A Review of 21 Cases," *New Engl J. Med.*, 258:919, 1958.
4. POTTS, W. J. AND RIKER, W. L.: "Differentiation of Congenital Cysts of the Lung and Those Following Staphylococcal Pneumonia," *A.M.A. Arch. Surg.*, 61:684, 1950.
5. WILLMAN, V. L., LEWIS, J. E., JR. AND HANLON, C. R.: "Staphylococcal Pneumonia: Surgical Considerations in Cases of Infants and Children," *Arch. Surg.*, 83:93, 1961.

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## ANNOUNCING THE 1964 ESSAY CONTEST

The American College of Chest Physicians is offering three cash awards to winners of the 1964 Essay Contest

First prize is \$500, Second prize \$300, Third prize \$200. Each winner will also receive a certificate.

A trophy will be presented to the medical school attended by the first prize winner. This trophy will be inscribed with the name of the winner and the name of his school.

The contest is open to undergraduate medical students throughout the world. Essays may be written on any phase of the diagnosis and treatment of chest diseases (cardiovascular or pulmonary). Five copies of the essay must be submitted in English; length is optional.

Contest closes on March 15, 1964.

Application and further information may be obtained from the American College of Chest Physicians, 112 East Chestnut Street, Chicago, Illinois 60611, U.S.A.

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DISEASES OF THE CHEST  
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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

## 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

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 fibers

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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 Tyndallometer 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

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 the parietal plaque can evoke a lesion on 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 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.

#### 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.

#### CONSENT

Asbestotic subcutaneous granulomatosis and asbestotic cutaneous verrucous scariosis 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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### SYMPOSIUM FROM THE INTERIM CLINICAL SESSION\*

#### AMERICAN COLLEGE OF CHEST PHYSICIANS

#### PHYSIOLOGIC STUDIES DURING CARDIOPULMONARY BYPASS ELIMINATING HEPARINIZED BLOOD

Drs. Arthur C. Shull, Jr. and Denton A. Cooley, Houston, stated that many adverse physiologic changes following temporary cardiopulmonary bypass recently have been attributed to the use of large quantities of fresh, heparinized, homologous blood to prime the various types of pump oxygenators. They have developed a technique of open heart surgery employing disposable oxygenators, primed with solutions such as 5 per cent dextrose in distilled water or 5 per cent dextrose in 1/4 isotonic saline solution, under normothermic conditions. Results of this technique in more than 200 clinical cases have demonstrated its superiority to techniques employing gassed homologous blood in almost every respect.

In order to determine certain physiologic changes

associated with this technique of open heart surgery, renal hemodynamic changes concomitant to this technique have been compared with those seen following temporary cardiopulmonary bypass with fresh homologous blood. A similar comparison has been made of changes in blood viscosity during and following bypass. Plasma electrolyte and both total body water and extracellular fluid volume changes have been studied as have changes in plasma hemoglobin levels during and following bypass.

The results of these studies would appear to offer further evidence that this technique of open heart surgery is superior to those employing pooled homologous blood. Both experimental and clinical findings were presented and their implications discussed.

#### BRONCHIAL GLANDS

Dr. Otto C. Brantigan, Baltimore, and co-workers have made a study of submucosal bronchial glands and the mucous goblet cells of the mucous membrane. The study was made in normal man and in pulmonary emphysematous disease in man with and without pulmonary denervation (sympathectomy and parasympathectomy). The study was made in various animals. The albino rat has no submucosal bronchial glands, but many goblet cells. The domestic pig presents submucosal bronchial glands more nearly like those in man than do other animals, but normally it has no goblet cells in the mucosa. The patient with pulmonary emphysema has submucosal

bronchial glands that are generally larger than the normal in normal subjects and in pulmonary emphysema in man, the submucosal glands show extensive staining with P.A.S. stain which indicates an acid or neutral mucopolysaccharide, fewer cells and glands stain with Alcian blue which indicates acid mucopolysaccharide. After pulmonary denervation, the pulmonary emphysematous bronchus shows a marked atrophy of submucosal bronchial glands and the mucosa and submucosal layer and a marked lessening of the Alcian blue staining material. In addition, there is the appearance of many goblet cells in the mucous membrane.

#### GASTRIC FREEZING IN TREATMENT OF SYMPTOMATIC HIATAL HERNIA

Approximately 20 patients with symptomatic hiatal hernia with or without associated peptic ulcer diathesis have been treated by gastric freezing after the manner of Wengertsen during the past ten-month period. An evaluation of the clinical results over this ten-month followup period was presented by Dr. Kenneth Cruise, Takoma Park, Maryland.

Pre-freeze evaluation and selection of these patients for cryogenic surgery was presented along with illustrations of some gross and microscopic changes seen in biopsy specimens of the mucosa. The results obtained during this period would not seem to support the excellent results reported by other investigators having smaller numbers of patients.

\*Portland, Oregon, November 30-December 1, 1963.  
Future issues of Diseases of the Chest will contain complete manuscripts of many of these papers.

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