FILE NAME: Oil Industry and American Petroleum Institute (API)

DATE: 1942

DOC#: API149

DOCUMENT DESCRIPTION: Book Excerpt - Cancers of the Respiratory System [Occupational Tumors and Allied Diseases]

OCCUPATIONAL TUMORS AND ALLIED DISEASES

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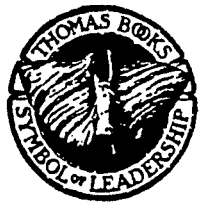

1942

CHARLES C THOMAS · PUBLISHER

SPRINGFIELD · ILLINOIS BALTIMORE · MARYLAND

Published by CHARLES C THOMAS
220 EAST MONROE STREET, SPRINGFIELD, ILLINOIS

First Printing

Printed in the United States of America

Type of Dust	Guinea Pigs	Rabbits	Rats	Chickens	Mice	Cats	Pulmonary Tumors
Chalcedony	242	28			40		0
Quartz	894	41	201	12	24	4	0
Marble and quartz	25						0
Hematite and quartz	28						0
Quartz and gypsum	88		27		6		0
Granite	298						0
Chert	196	3	52		10		1
Hematite	106		31				0
Asbestos	235						0
Soft coal	37						1
Fluorspar-crude			15				0
Carborundum	213						0
Marble	172		12				0
Gypsum	200		12				0
Total	2832	72	338 3338	12	80	4	2 0.06%

Asbestosis

Chemical and Technological Aspects. Asbestos, designating a group of silicate minerals of crystalline fibrillar structure, was known to the ancient world. At that time and during the following centuries it was used occasionally in the production of perpetual lamp wicks, shrouds for cremation, and other purposes partly of curiosity character. It was only since the latter third of the last century that this material has attained a major industrial importance, and has become of serious significance from the standpoint of industrial hygiene.

Asbestos is distributed widely in the world but occurs in relatively few places in sufficient quality and quantity to make mining profitable. The chemical nature of commercial asbestos varies with the site of its occurrence.

1. Amphiboles, found in Italy, South Africa, and Australia, occurs in five varieties: a) tremolite is a white, brittle, calcium-magnesium silicate; b) actinolite is a mineral consisting of fibrillar or radiating crystals; c) hornblende-asbestos or amphibole-asbestos is a flexible, greenish, fibrillar matter containing much calcium and a high amount of silicic acid; d) mountain leather, mountain cork, or mountain wood is of a greyish or brownish color occurring in the form of sheets of interlacing fibrils; e) crocidolite, which is mined mainly in Australia and South Africa, has a bluish color, a silky lustre, and elastic fibers of considerable length and tensile strength, and is a soda-iron silicate. These types are usually very acid resistant, but less fire resistant.

2. Serpentine and its subvarieties, picrolite and chrysolite, are hydrated magnesium silicates in which the magnesium may be replaced by iron in

some types. The serpentine has a bandy or slate texture and may contain traces of nickel and copper. It is mined in Canada, Russia (Ural Mountains), Germany, Corsica, France (Pyrenees), Siberia, and South Africa.

The Canadian asbestos, mined at Thetford near Quebec, where approximately 78 per cent of the world production is obtained, consists of 33 parts of magnesium, 41 parts of silicic acid, and 5.75 parts of iron oxide (Oliver); while South African asbestos is composed of 2 parts of magnesium, 50 parts of silicic acid, and 40 parts of iron oxide. Italian asbestos contains 42 parts of silicic acid, 38 parts of magnesium, and 3 parts of iron oxide. None of the various types possesses any free silicic acid.

The production of asbestos has risen enormously in recent years. It received this great impetus from its use in the automobile industry. The total production in 1868 was two hundred tons; in 1880, five hundred tons; in 1925, 330,000 tons; and in 1929, 430,000 tons. The United States is the principal consumer of this product. More than 12,000 persons are employed in the chief asbestos plants of this country. Occupational contact with asbestos is present in many industries using asbestos products. Asbestos is employed for a great variety of purposes, such as the manufacture of incombustible and insulating materials (fire resistant clothes, blankets, theater curtains, sheets, ropes, cords, twine, and threads), filter cloths, mill-board, wall board, shingles, tiles, mortar (together with cement and plaster of Paris for providing a fireproof wall lining), clinkers, gaskets, packing material of pumps, insulating material of steam pipes, water pipes, boilers, and electric wires, brake lining, paper, mattresses, adsorbent of dyes (for manufacture of fire proof and acid proof stains), ingredient of rubber products, and artificial wood (mixed with wood powder) (Lanza; Lynch; Stewart, Bucher and Coleman; Ullmann; Encyclopedia Britannica; Dewirtz; Nordmann; Alwens; and Cooke). The fibrillar types are spun, twined, and woven, while the brittle varieties and the dusty waste are compressed and molded into the desired shapes.

Conditions and Types of Exposure. The chief health hazard consists in the inhalation of asbestos dust, which is produced abundantly during the preparation of the mineral for the spinning process (purification and removal of stony impurities) and during various other phases of the production and manufacturing processes of asbestos and asbestos containing goods. Direct contact of the skin with asbestos is of minor importance. The number of actually exposed persons is rather large in the industries manufacturing asbestos products. Of the 12,000 workers employed in the major asbestos plants of the United States, Lanza estimates that 10,000 are exposed appreciably to the asbestos dust; while in Germany, where several thousand asbestos workers are present, only a fraction are said to be actually exposed.

Symptomatology: Cutaneous Lesions. The direct skin contact with mined asbestos is responsible for the production of painful warts formed around the asbestos needles, which penetrate the skin of the palms of the hands. The

majority of asbestos miners in the Ural Mountains are said to have on the inner surfaces of the right hand three to five or more papular, non-inflammatory, wart-like formations of round or polygonal shape reaching the size of a pea (Dewirtz). They consist of hyperkeratoses sometimes associated with acanthosis and prolongation of epithelial cell pegs. There is some lymphocytic infiltration in the cutis. Between the cornified masses asbestos needles are found. Many mitoses may be present in the thickened malpighian layer, and large giant cells with centrally located multiple nuclei may be present in this stratum. These lesions will persist unless the asbestos needles are removed. The warts do not exhibit any appreciable proliferative tendencies.

Pulmonary Lesions (Asbestosis). The asbestotic pneumoconiosis, resulting from a prolonged occupational inhalation of asbestos dusts and fibrils, is a condition more serious than the cutaneous manifestations. The first fatality of asbestosis was reported by Murray in 1899. It was not until 1914 that a record of an asbestosis death was published in Germany (Fahr). Fatalities from asbestosis as the main or contributory cause of death were published in the United States by Lynch; Stewart, Bucher and Coleman; and Shull. The Metropolitan Life Insurance Company recorded, between 1924 and 1936, seventeen cases in which asbestosis contributed to the lethal outcome of the main disease.

In Germany there was no appreciable interrelation between asbestosis and tuberculosis of the lung, such as exists regarding silicosis. In England there were 30 cases of asbestosis complicated by tuberculosis in a series of 100 cases of this type of pneumoconiosis reviewed by Wood. Similarly, in the United States a certain relation to tuberculosis was apparent, but the coexistence of the two diseases was not as frequent as in the case of silicosis (Stewart, Bucher and Coleman; Shull; and Lanza, McConnell and Fehnel). The development of a pulmonary asbestosis depends, according to Bauer, upon the duration and degree of exposure to asbestos dust, and on a certain personal disposition. A similar observation was made by Sweany, who remarked that some people develop an asbestosis rapidly, while others may be exposed to the same conditions for years without detriment. Individual differences in the filtering qualities of the nasal passages are mentioned by Lehman as possible reasons for such discrepancies in susceptibility.

Asbestosis usually develops after an exposure of three to fifteen years (Martz), but may not produce subjective symptoms until many years after the cessation of exposure to asbestos material. The first symptoms are dyspnea and a cough with expectoration. Later there is pallor, cyanosis, palpitation of the heart, loss of appetite and weight, and piercing pains in the chest during breathing. The shortness of breath becomes aggravated during physical exertion. In the beginning the physical examination reveals scarcely any pulmonary changes; but later there appears a diminution of resonance, especially over the lower parts of the lung (particularly the right side), where dry and

crackling sounds are perceptible in auscultation. The respiratory capacity is decreased, and there is a diminution of the respiratory shift of the pulmonary borders during respiratory movements of the lung. The heart is often enlarged due to the increased functional demand placed on it, because of the decreased respiratory surface and impaired circulation in the lung caused by the pulmonary fibrosis. The enlargement of the right side of the heart becomes accentuated as a cardiac insufficiency ensues. The roentgen-examination of the chest shows an early, diffuse, interstitial fibrosis having reticular honeycomb-like outlines and showing progressive tendencies, which later cause a filmy-hazy appearance of the lung. This condition has been termed "ground glass" appearance. The hilum shadows are enlarged in the more advanced stages. The movements of the diaphragm are reduced. The microscopic examination of the sputum executed with proper methods and thoroughness reveals the presence of golden brown asbestos bodies.

Pathology. The observations made at necropsy reveal a shrunken, blue-black lung of firm consistency containing little air, especially in the lower lobes which often are covered by a thickened pleura. The cut surface shows a widened, thickened, and prominent, interstitial, connective tissue. There are bronchiectases and small abscesses together with pleural adhesions and bronchopneumonic foci. The hilum nodes are enlarged, hard, and brownish.

Upon histological examination asbestos bodies are found in the interstitial connective tissue, and the peribronchial and hilum lymph nodes. These characteristic formations consist of a central translucent fiber or needle-like crystal (asbestos crystal) measuring 10 to 100 microns in length and 1 to 12 microns in thickness. The central fiber is ensheathed in a shiny golden or brown substance with varying architectural figuration. The whole body may have the shape of a rod with smooth blunt ends, or with one or both ends forming a single knob or a conglomeration of knobs. The shaft is often a series of rounded beads or discs strung closely along the central filament (Lynch). In other instances the asbestos body has the shape of a hoe or a club, or represents a long slender filament. The bodies give a characteristic Prussian blue reaction, which is attributed to their iron content. The iron content of the asbestos bodies is higher than that of the asbestos needles. The assumption has been made that the additional iron originates from decomposed erythrocytes. The nature and chemical composition of the asbestos body is still uncertain. McDonald advanced the theory that the central part of the body is represented by a particle of an asbestos fiber in the process of alteration and absorption by hydrolysis, while the enveloping matter was supposed to consist of silica in a colloidal or gel-like state. This latter conception was proposed, also, by Koppenhofer.

Beger contended that the sheath was composed of proteinic matter. The asbestosis bodies are found within the bronchioli, in the alveoli, and in the interlobular, peribronchial and subpleural tissues among the lymphatic routes.

They occur, moreover, in the peribronchial lymph nodes, within the walls of the sinuses, and embedded in fibrous areas. In the alveoli they are surrounded by phagocytic, mononuclear cells and foreign body giant cells massed especially around the larger bodies. The interstitial tissue shows a diffuse fibrous thickening and scarring. The asbestosis is, thus, an indurative, interstitial, diffuse fibrosis of the lung increasing in degree from the apex to the base.

Asbestosis and Carcinoma of the Lung. It is only in very recent years that serious consideration has been given to the possibility of a cancerigenic action of asbestosis upon the lungs. In an analysis of 18,280 death certificates for cancer of the lung and larynx in males from England and Wales from 1921 to 1932, Kennaway and Kennaway did not find any cases of lung cancer in asbestos workers. Since 1935, an appreciable number of cases of asbestosis associated with carcinoma of the lung, have been reported from England, Germany, and the United States (Lynch and Smith; Lynch; Gloyne; Wood; Horning; Nordmann; and Egbert and Geiger). The incidence of lung cancer among workers in whom an asbestosis was found at autopsy is relatively high, ranging from 17 per cent (Nordmann: 2 out of 17 cases) to 20 per cent (Wood: 2 or possibly 3 out of 12 cases). Teleky noted that there exists a minimum of 39 and a maximum of 78 autopsies in fatalities from asbestosis, the records not being clear in this respect. Teleky only knows of 6 cases of pulmonary cancer with asbestosis, and feels that this ratio is high in comparison to the number of necropsies. Koelsch mentioned the occurrence of 12 cases [Lynch (3); Egbert and Geiger (1); Gloyne (2); Dominici (2); Baader (1); Nordmann (2); Wood (3); and Horning (1)]. Lynch and Smith found among 35 autopsies with asbestosis 2 cases of pulmonary malignancy (six per cent), whereas there was an incidence of only 0.3 per cent of this type of neoplasia among approximately 2,300 autopsies of the general population. The period of occupational exposure to asbestos dust ranged for five cases from 7 to 21 years, but in an additional case only a year and a half. In several instances the lung tumor became manifest several years (10 to 12) after cessation of the work in asbestos. Fifteen to twenty-one years elapsed (average, 18 years) between the first exposure to asbestos and the death from asbestosis with carcinoma of the lung. The age at death depended upon the age of the individual at the time of the first exposure. The workers dying in the more advanced age groups entered an occupation with an asbestos hazard later in life, while those who died at an early age started to work in asbestos plants when still young. There was no evidence of a so-called old age predisposition, as two of these cases died at the age of 35 years and a third was 41 years old.

The majority of neoplasms were cornified squamous-cell carcinomas. Only one case was an adenocarcinoma, and a second was an anaplastic, round-cell carcinoma. The latest case reported by Lynch and Smith showed squamous cellular and glandular carcinomatous structures. This numerical distribution between the different histologic types of carcinomas varies distinctly from

that usually observed (Nordmann), but agrees with the shift toward a more frequent occurrence of the cancroids noted in recent years by Probst in pulmonary malignancies of unknown genesis. A multicentric development of malignant growths in asbestos lungs was found by Nordmann and Gloyne, and it is considered by Nordmann as a characteristic feature of this type of lung cancer. A multifocal sprouting of squamous cells lining the bronchial walls was seen by Nordmann, who regards these changes as precancerous in nature. A diffuse, proliferative thickening of the bronchial epithelium was recorded by Gloyne, who believed that this constituted a continuous prolongation of epithelial growth from some unknown starting point rather than a series of isolated proliferative units. These epithelial prolongations were hollow in places, showing a degenerating layer of cells surrounding a central empty space, and, in turn, being enveloped by rings of keratinized squamous cells. The carcinomas of three cases were very small [Gloyne (2); and Nordmann (1)], and had not produced any metastases. For this reason two of these cases (Gloyne) represented coincidental observations made during the post-mortem examination. The other cases were large tumors with extensive metastatic deposits in remote organs.

An evaluation of the evidence presented reveals certain features connected with these cases, which are suggestive of an occupational causation. There is an incidence of lung cancer in asbestosis of the lung which is definitely excessive. The relatively small number of necropsies on which this conclusion is based represents an urgent indication for more extensive post-mortem examinations in cases of asbestosis; especially considering that pulmonary malignancies seen in previously reported cases were, in several instances, very small and were coincidental observations. A second point favoring an industrial origin is the young age of an appreciable portion of the cases thus far recorded, and the relationship existing between the time of exposure to asbestos dust and the manifestation of the neoplasms throughout the entire series. The third factor consists in the shift of the histological types observed in the direction of a predominance of cancroids, and in the multicentric development of two (Nordmann; and Gloyne) and possibly even three (Lynch and Smith) of these pulmonary neoplasms. The existence of the cutaneous warty lesions suggests that asbestos needles embedded in epithelial tissue are apparently capable of eliciting non-inflammatory, epithelial proliferations. Nothing is known about the actual causative mechanism through which asbestosis may produce such epithelial lesions, and may transform them subsequently into malignant growths. The assumption of a chronic irritation of nonspecific nature or of specific silicotic character, such as proposed by Lynch, is scarcely acceptable and satisfactory in view of the negative evidence available in this respect. One of the cases reported by Nordmann in association with Horning showed symptoms of a marked and generalized, allergic disposition (bronchial asthma, and hematic eosinophilia), which may have played a

contributory role in the development of the neoplastic response. Asbestos bodies contain a considerable amount of iron, and there exists some evidence that points to a possible causative interrelation of pulmonary siderosis and carcinoma. The relatively small number of asbestosis carcinomas of the lung reported does not disprove an occupational origin of this condition. Previous experience with other industrial neoplasms (lung cancer of the Joachimsthal miners; and lung cancer of the chromate workers) has shown that such isolated cases may represent the first warning of a subsequent appearance of large numbers of similar tumors of undeniable occupational derivation.

Medico-Legal and Social Aspects. Asbestosis carcinoma of the lung is not included in any group of occupational tumors recognized by any country. The evidence presented is sufficiently serious, and the number of persons exposed to the suspected causative agent large enough to indicate a thorough and extensive clinical, statistical, and experimental investigation of the incidence and causative interrelation of asbestosis and pulmonary carcinoma. In view of the fact that several of the states with many large asbestos plants do not include asbestosis among compensatory occupational diseases, this study is even more urgent. Such a condition militates against an effective hygienic control of an important industrial hazard, and impedes the collection of pertinent and essential information in regard to the incidence, nature, and potentialities of an occupational disease growing steadily in general significance. Workers dying from asbestosis should be subjected to a post-mortem examination including a histological study of the pulmonary lesions, as multiple neoplastic manifestations in asbestosis may be mistaken for tuberculosis (Lynch).

Sanitary Measures. The following measures may be taken to eliminate or reduce the existing hazard caused by the inhalation of asbestos dust: humidification of the asbestos matter and the air to decrease the production of dust; introduction of a closed production system, wherever possible; establishment of an extensive and efficient exhaust ventilation; frequent cleaning of the workrooms to remove the accumulated dust; prevention of air currents caused by moving machinery parts; construction of rooms, machinery, and pipelines offering little opportunity for deposition and accumulation of dust; separation of operations in which the production of dust can be eliminated entirely from those in which this is not possible; wearing of respirators by workers engaged in dusty operations; and mechanization of operations under elimination of human labor to reduce the number of workers exposed. The dust hazard is most pronounced in store rooms for raw material, in rooms in which the raw fibers are handled, and in spinning, weaving, mixing, and filling rooms.

Siderosis

Occupational siderosis of the lung is of the black type, which is caused