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BOOK CHAPTER

MODERN OCCUPATIONAL MEDICINE

Fleming, D'Alonzo and Zapp, 1962

Chapter 33

Occupational Chest Diseases

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Permanent disability resulting from occupational factors always has been and still remains an important problem in industrial medicine. In the United States alone more than 4 million industrial workers are exposed to potentially hazardous substances. The current bibliography on the subject exceeds 8,000 references, and it is obviously possible to review here only those occupational chest ailments which are of greatest topical importance. At the same time, emphasis will be placed on basic principles and pragmatic issues.

I SILICOSIS

Because of the predominance of silica in the earth's crust, it is natural that silicosis should constitute an important occupational chest disease. Of the more than 1,000 known minerals, more than 300 are compounds of silica. It is indeed fortunate that many of these naturally occurring varieties of silica, as well as the element silica, are biologically inert. It should, however, be cautioned that epidemiologic surveys and experimental inquiry have concerned themselves with fewer than 10 per cent of these substances. In recent years, numerous synthetic siliceous substances have been introduced.

Many theories of the biological action of SiO_2 have been suggested, e.g. *(a)* the mechanical injury concept, which presumes that angular quartz particles can lacerate vital cell components. *(b)* the solubility theory, silicic acid being presumed to be the pathogen. *(c)* the polymerization theory, the slow formation of polysilicic acid being invoked to explain the retarded development of silicotic nodules. *(d)* the piezoelectric theory, based on the assumption that the well known piezoelectric forces characteristic of quartz may cause local tissue injury. *(e)* the unsaturated valency theory, which postulates the existence of a layer of highly reactive SiO molecules at the fracture angles of quartz particles. *(f)* the colloidal silica theory, which is based on the concept that the crystalline quartz particle is covered with a film of toxic colloidal silica. *(g)* the protein denaturing theory, which postulates that the physical field at the surface of the quartz particle induces organic chemical molecular rearrangements in proteins; *(h)* the auto-immunity theory, which postulates that plasma cells carrying immune bodies are the prime agents in tissues in which silicotic fibrosis occurs.

None of these theories furnishes a complete explanation. All of them aim primarily at explaining the silicotic nodule, a minor, late component of the disease. Few of them take cognizance of the fact that quartz particles cause immediate disruption of mitochondria and the death of phagocytes which ingest them. This process continues until the quartz particles have acquired a proteinaceous coating from the cytoplasm of previously killed cells. When this coating is digested off by the phagocyte, the latter again becomes prone to injury. Successive phagocytes will again be killed until their cytoplasmic protein, in turn, furnishes a new coating for the silica particle. This process continues ad infinitum. The mineral body contributes fibrocytes to help isolate the toxic particles by means of hyalin collagen. At the same time phagocytes continue to obliterate but are killed as soon as they ingest the silica particle.

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FIG. 56

ASBESTOSIS

Roentgenography

A, Diffuse fine linear pulmonary fibrosis. The main zone of collagen deposition is in the subpleural zone on either side of the lumina of vessels. Fibrous trabeculae dip inward along vessels and bronchi. Dilated bronchi within zones of consolidation. Diagnosed fine focal perivascular fibrosis. *Miner exposed to chrysotile dust 23 years.* Initially symptomless. Died of cor pulmonale.

B, Asbestosis. Dense linear perivascular fibrosis with minimal vesicular emphysema. *Textile worker exposed to amosite dust 11 years.* Progressive exertional dyspnea. No cough. Died of cor pulmonale.

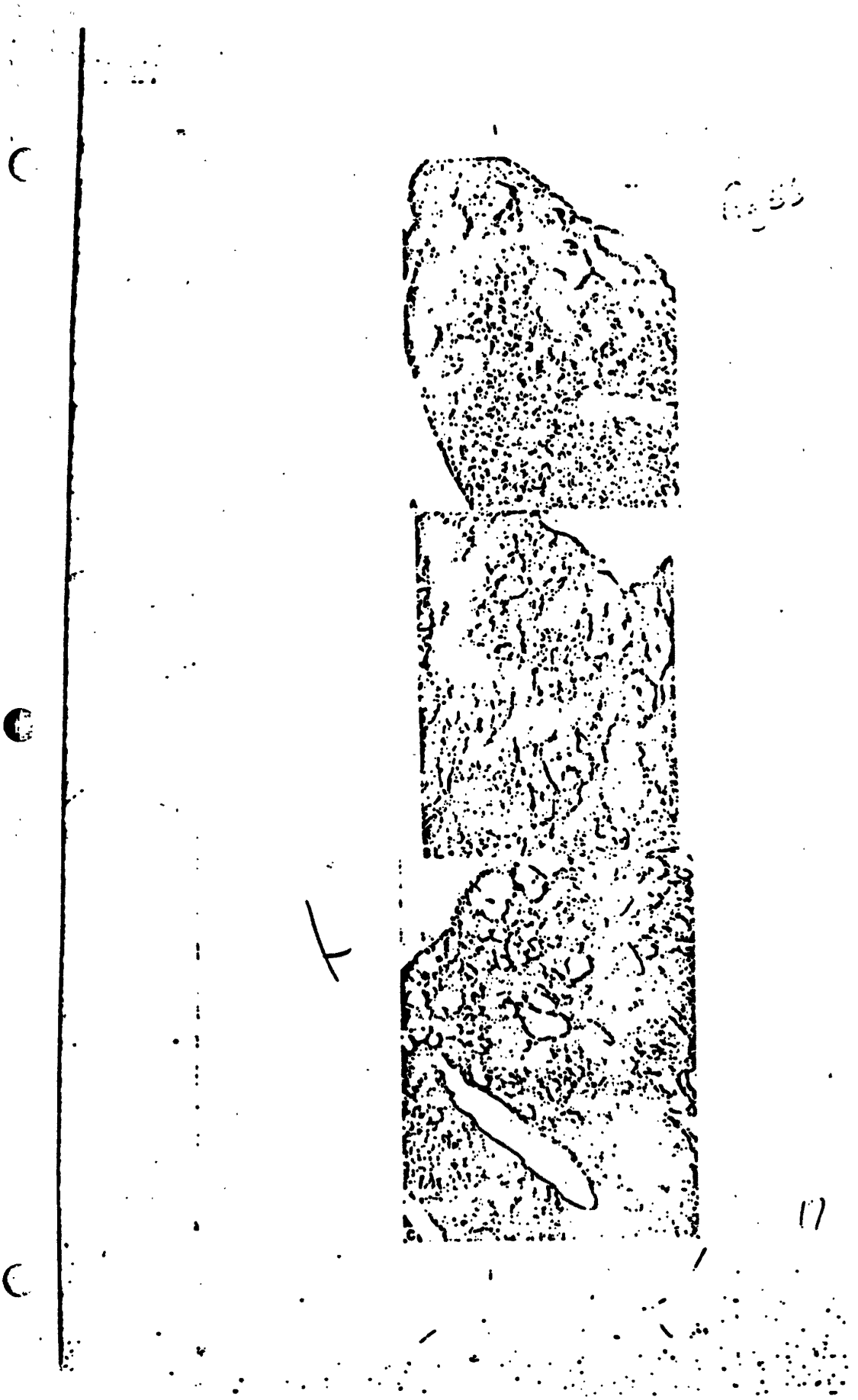
C, Tuberous. Irregular interstitial fibrosis, mainly perivascular and peribronchial. Marked distension of lower bronchi and bronchioles. *Mill worker exposed to crocidolite talc dust 17 years.* Terminal progressive dyspnea and productive cough. Died of cor pulmonale.

Asbestosis: This is by far the most important among the silicatoses. Six varieties of asbestos fibers are commonly implicated, viz. chrysotile, amosite, crocidolite, anthophyllite, aktinolith and tremolite. Since more than 85 per cent of the asbestos used in the U.S.A. is chrysotile, cases occurring in this country are most probably chrysotile-derived. Crocidolite is more commonly mined and used in Europe and Africa. This information leaves scope for precise identification of the industry which has caused the asbestosis since the different types of asbestos fibers can be distinguished in biopsy or autopsy material.

Asbestosis is provoked wherever exposures to asbestos dust occurs. However, in textile industries there appears to be a greater prevalence of severe disease. In contrast with silicosis, the asbestotic reaction in the lung is excited not by minute particles but by relatively long fibers. Thus, if asbestos be pulverized to particles no longer than 3 microns,

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very little disease develops and the process advances much more slowly than when the aerosol contains a predominance of fibers of 10 to 50 microns. Again, if the fibers are introduced into the lung in a high caliber form (*e.g.* conjoined multiple fibrils), less disease results than when the individual fibers are split into their component fibrils of submicron caliber.

Asbestosis is essentially an interstitial pulmonary lesion in which all component tissues are involved with focal emphasis of the process. The lesions range in severity from mere alveolar mural cellular infiltration of a microscopic nature, not detectable by radiography or even on gross anatomical examination, to massive consolidation with associated vascular occlusion, bronchiectasis, and carcinomatosis.

The interstitial lesions result after fine short fibers have been ingested by phagocytes and transferred into the lymphatic channels of the alveolar septa. Here fibrocytes proliferate, new capillaries form and reticulin, collagen and new elastic fibers are laid down. This type of lesion cannot readily be distinguished from other interstitial pneumonitides unless the asbestos fibrils are detected by oil immersion or electron microscopy. Rarely, some of these fibrils will be rendered obvious by conversion into an asbestos body through proteinaceous encapsulation and ferrous pigmentation. If the disease remains limited to this stage, disability may remain limited being merely due to increased work of breathing.

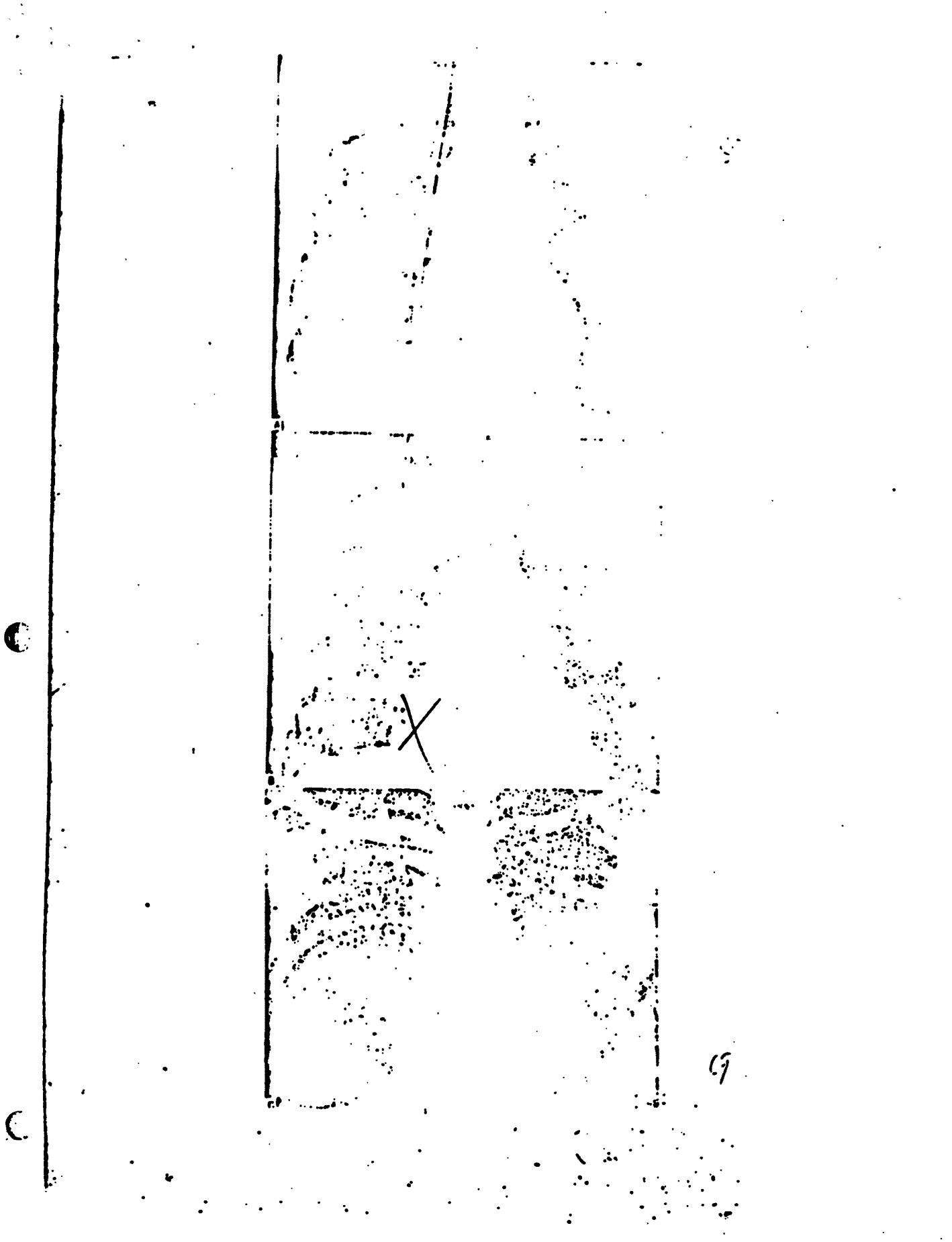
With extension of the process three major complications arise. There may be progressive invasion of venous adventitia with perivascular fibrosis, intimal hyperplasia, and ultimately occlusion of the vascular lumen. Relatively large venous channels may become involved but arteries tend to escape early damage. This obstruction to the venous channels leads to progressive elevation of pulmonary tension and marked cor pulmonale. Heart failure is the usual cause of death in these cases. The asbestotic origin of the venous obstruction is readily proven by demonstrating the asbestos fibers in the peri- and endovascular granulation tissue. Perl's stain often is sufficient for this purpose as it will demonstrate the presence of iron deposits in the protein sheaths around the asbestos fibers. Another tell-tale feature consists of the deposition of abundant perivascular elastic laminae and fibers.

While the stage of the alveolar mural invasion is not radiographically detectable, except for blurring of lesser pulmonary markings, the stage of perivenous fibrosis is characterized on the x-ray by the development of a coarse web and the effacement of the normal vascular pattern. The extent of the latter damage is often underestimated until tomography or angiography are resorted to.

Massive fibrosis tends to develop mainly in the parts of the lung which are in constant agitation, such as the lung apex, the supradiaphragmatic zone of the basal lobes, and the paracardiac components. The consolidation is effected by progressive interstitial invasion, atelectasis, trapping of fibers within alveolar spaces where phagocytes surround them and organization takes place, and by abundant pleural thickening on both sides of the lamina elastica. In these zones carnification is often so complete that all semblance of pulmonary architectonics is completely lost. Major blood vessels can only be identified by elastic stains which reveal their elastic skeletons.

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FIG. 57

ASBESTOSIS

ROENTGENOGRAPHIC FEATURES

A, CASE 1 (UNIVERSITY ASSOCIATION). *Linear* pulmonary fibrosis and ground glass opacification. *Compensating* shrinkage of upper lobes. Incipient cardiomegaly. Diaphragmatic adhesions. *Textile worker heavily exposed to chrysotile dust 7 years.* Slight exertional dyspnea and tachycardia. Discontinued work.

B, ADVANCED CONFLUENT ASBESTOSIS. Marked interstitial infiltration. Ground opacification. Parenchymal infiltration and "shaggy heart." Reduced lung volume. Marked cardiomegaly. *Same case as A, 6 years after discontinuing work exposure to chrysotile dust.* Marked cardiopulmonary disability. Died less than a year later from advanced chronic cor pulmonale.

C, ADVANCED CONFLUENT ASBESTOSIS. Extensive bilateral diffuse infiltration of lung fields. Thickened pleura and subpleural cyst. Part of pulmonary opacification due to congestive cardiac failure. Heart markedly enlarged. *Negro miner exposed 8 years to crystalline dust.* Marked dyspnea and anasarca. Died two years later from pulmonary carcinoma at the age of 38.

Radiographically, these consolidated zones present as diffuse and spreading opacities along the upper mediastinum, above the diaphragm and on either side of the heart, whose silhouette becomes blurred: the so-called "shaggy heart" feature. Calcification may occur in these dense zones.

Symptomatically, these patients usually are merely dyspneic on exertion unless there is marked accompanying vascular obstruction. In such cases cor pulmonale will be the predominating lesion. The thorax is usually small and immobile while breath sounds, vocal fremitus and resonance are markedly impaired over the affected zones. Bronchitis and bronchiolitis are not common in asbestosis, though some of these air passages may become involved in the fibro-granulomatous reaction. Advanced cystic bronchiectasis may develop as a result of the fibrotic distortion of the lung. At first there is more bronchial distension and some fluid or mucus may become trapped in the dilated ducts. Later infection supervenes with all the attendant inflammatory processes leading to chronic suppurative bronchiectasis. The symptoms in these cases do not vary much from conventional varieties of bronchiectasis except that hemoptysis is less common and cor pulmonale develops early.

Tuberculosis may occur in asbestotic subjects, but is not more prevalent among these than it is in the general population. The lesions are not any more severe than those usually seen and often are limited by the fibrous barriers caused by the asbestosis.

Pulmonary carcinoma has been observed with such high frequency in employees of the asbestos industry that a causal relationship has been accepted by most authorities. Indeed, pulmonary carcinoma is compensable as an occupational disease in England, Germany and South Africa. Most of the carcinomata are squamous cell epithelioma ta. Pleural mesothelioma is also quite prevalent, especially in crocidolite industries.

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Since the lymphatic system is not completely destroyed in asbestosis, e.g. [illegible] may spread diffusely throughout the lung in spite of extensive fibrosis. This does not occur in silicosis.

Asbestos bodies not *infrequently* are demonstrable in the sputum, where they may serve as a diagnostic clue. However, severe asbestosis is compatible with their absence. Again, asbestos bodies may be quite prevalent without coexisting asbestosis. This is possible because the majority of asbestos bodies are formed in the alveolar spaces when fibers have become engulfed by phagocytes. These asbestos bodies may become incarcerated in granulomatous tissue by atelectasis and secondary inflammation.

In industries in which asbestos is handled extensively, warts are not uncommonly experienced at points of cutaneous friction, e.g., hands, collar, belt, cuff zones. Many of these acanthotic lesions are indistinguishable from benign warts, but occasionally the tell-tale causative asbestos fibers are identifiable. Later these warts, if left untreated, have a strong tendency to progress to malignant anaplasia. They should, therefore, be radically extirpated as soon as they are identified.

Important features in all cases of asbestosis are the existence of a lag period before the onset of the symptoms and the inexorable progression of the disease despite cessation of dust exposure. There is evidence to support the view that the first few years of exposure to asbesto- fibers are as important determinants of the final outcome as are most of the remaining years of exposure. Thus, in one verified case, a single month of exposure to asbestos dust in a textile plant in one country was followed thirty years later in another country by fulminating fatal asbestosis. The intervening period was one of good health and no further asbestos dust exposures had occurred. Continued exposure will, of course, aggravate the process. The severity of the asbestosis is thus a function of the duration of the exposure multiplied by the period which has intervened between the inception of exposure and the onset of the disease. This principle at the same time accounts for the progression of asbestosis after exposure has been discontinued. Cases are currently cropping up in men and women who had worked as pipe insulators on liberty ships during the second world war without since having had any further asbestos exposure.

Fig. 58

SILICATOSIS

Roentgenographic Features

A, Tuberculosis. Multiple, dense irregular opacities of unequal size and fuzzy outline irregularly dispersed. Thickening of interlobar fissure. Associated emphysema. *Tale miner 25 years.* Lesions progressed steadily during final 10 years, during which pulmonary disability developed. Patient expectorated white chalky material identified as tremolite.

B, Rock Worm. Silicosis. Dispersed linear and focal infiltrations with focal semiconsolidation and interspersed emphysematous zones. Contraction of upper lobes. Cardiac rotation. *Exposed mainly to rock and coal dust for 23 years.* Progressive dyspnea and cough. Died from cor pulmonale. Characteristic rock wool fibers within fibrotic perivenous granulation tissue.

C, Silent Pneumoconiosis. Dispersed fine mottling, decreased vascular markings, increased hilar shadows, dense opacity right base, identified by lateral view roentgenography as atelectatic middle lobe. Bronchography revealed annular constriction of right middle lobe bronchus with distal bronchiectasis. *Slate quarry man 18 years.* Right middle lobe resected. Bronchus stenosed by dense collar of fibrosed lymph nodes containing abundant petrographically characteristic slate particles. Rest of lung showed small, perivascular, fibrotic nodules.

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