THE COMMISSIONER OF PUBLIC HEALTH.

RECORDS.

Asbestosis.

Asbestos is a hydrated silicate of magnesium in combination with traces of iron, nickel, calcium and aluminum. The substance is mined in many parts of the world, including Canada, where most of the American supply (chrysotile) is obtained. Mined asbestos comes in long, thin, fibrous strands which can be spun or woven. Its pliable texture and high resistance to heat and chemicals make asbestos an important industrial product in the manufacture of mattresses, brake-lining, fire-proofing material, electrical insulation, in jacketing boilers and steam pipes, and in a variety of building fixtures. Although the dust hazard associated with the asbestos industry does not nearly compare in prevalence with that of the silica industry, the rapid growth of the former puts asbestosis among the important forms of dust diseases.

Inhaled asbestos fibres range in size from 10 to 200 microns or more. The dust given off from asbestos during industrial operations consists mostly of small fragments of fibres from 1 to 10 microns long and under 2 microns diameter, together with longer pieces up to 50 or 60 microns, and particles 0.5 to 0.75 microns diameter. The action on the lungs is mechanical rather than chemical. The large particles, unable to enter the alveoli, lodge in the lumen and obstruct the respiratory bronchioles. Atelectasis of the distal alveoli is followed by a fibrotic reaction in the collapsed tissue. Nonobstructed alveoli undergo compensatory emphysema. Contrary to that seen in silicosis, there is practically no nodulation unless silica is mixed with the asbestos dust. The hilar lymph nodes are not much enlarged for the reason that the lymphatics are not actively engaged in the pathologic process.

The gross appearance of the lung is characterized by scattered areas of diffuse fibrosis, affecting chiefly the lower lobes, emphysema of the uninvolved parts and intense pleuritis. Depending on the extent of coexisting anthracosis and silicosis, there are associated changes and pigmentation of the lungs and lymph nodes. Infection with pyogenic organisms and tubercle bacilli modify the pathology. Tuberculosis has been found in about one-third of the autopsied cases, but there is some doubt as to whether asbestosis per se favors the development of tuberculosis, or whether vulnerability to tuberculosis is primarily due to poor working conditions and incidental factors.

A striking feature of the pathology is the presence of "asbestos bodies" seen on histologic examination of lung tissue. These golden yellow or brown bodies have been shown by Gloyne to be composed of a central core of asbestos fibres, covered by a layer of iron-containing material which is believed to be derived from blood pigment of the tissues. Asbestos bodies may be found in the sputum of asbestos workers, but their presence does not necessarily indicate lung disease. They vary considerably in shape, and are from 24 to 60 microns long and 12 to 24 microns diameter, golden-yellow in colour.

The roentgen appearance of early asbestosis is not revealing. Advanced disease often shows distinguishing characteristics. The fine pulmonary fibrosis, patchy areas of interspersed emphysema and overlying pleuritis are reflected roentgenologically in a "ground-glass" appearance of a uniform quality, in places showingdenser opacities which, however, seldom assume the nodulation of silicosis. The lower portions of the lungs are chiefly involved. A marked pleural reaction manifests itself in obliteration of the costophrenic sinuses, an unevenness of the

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diaphragm and a felted or "porcupine" appearance at the periphery of the cardiac silhouette, the last caused by the pleuropericardial adhesions.

The onset of the disease is insidious with gradual increase in cough, expectoration, dyspnea, loss of weight and, in time, inability to work. The physical examination is not revealing. Wood and Gloyne draw attention to a peculiar earthy complexion of the face and a slight, violet tinge in the cheeks and lips of some individuals. Occasionally, asbestos corns occur in the skin of the hands caused by the penetration of asbestos fibres into the superficial epidermis. It takes, on the average, [redacted], between five to ten years for asbestosis to develop, although it may occur in as short a period as two years where working conditions are bad. Whether the disease can progress after contact is broken with asbestos dust is still unsettled. Death results from tuberculous or nontuberculous infection, congestive heart disease or other intercurrent diseases. The measures listed for the prevention of silicosis apply also to asbestosis.

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DIRECTOR.
TUBERCULOSIS CONTROL BRANCH.

X Reference: 'Diseases of the Chest' - Rubin.
'Dust and its Effects on the Respiratory System' - Gill.

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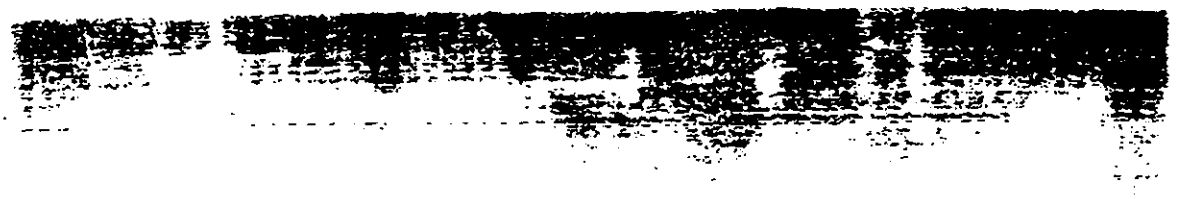

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