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Silicosis and Other Pneumoconioses

French: *Silicose et autres pneumoconioses.* — German: *Silikose und andere Staublungenerkrankungen.* — Italian: *Silicosi ed altre pneumoconiosi.* — Spanish: *Silicosis y otras neumoconiosis.*

During the past few years many investigations have been made and interesting reports published concerning the occurrence and prevention of silicosis, a disease with characteristic modification of the lungs caused by quartz containing dust. Some of the more important papers, describing the conditions in different countries and different occupations, relate to the Witwatersrand goldmines[1]; the Schauinsland mines (gneiss quarries)[2]; the Joachimsthal pitchblende mines[3]; the "Tri-State district", a zinc and lead mining district in Oklahoma, Missouri and Kansas[4]; and porphyry quarries.[5]

The conditions in steel plants were investigated by CUMMINGS[6]; in rock drilling by NIGHMAN and KIESSLING[7]; among casting cleaners by HEDENSTEDT[8]; and an investigation was also made in the silica brick industry.[9]

A paper on mica and pegmatite workers, handling material containing quartz, was published by DREESSEN, DALLAVALLE and others.[10]

Very interesting is a "Restudy of a Group of Granite Workers", reported by RUSSELL.[11] A large number of granite workers, first examined in 1924[12], were re-examined. The re-examination corroborated the experience that the development of silicosis is not stopped by shifting to non-dusty occupations. The process continues towards a more serious silicosis or develops into a silico-tuberculosis.

Several investigations were made among coal miners, especially among soft-coal miners, who inhale coal dust mixed with quartz dust. D'Arcy HART, ASLETT, HICKS, YATES, BELT, and FERRIS have published the results of their examination of workers in South Wales coal mines[13], where 560 anthracite miners were examined in all details; 1,392 bituminous and anthracite miners were examined only by X-rays; and 470 coal trimmers were examined mostly by X-rays. Pathological pulmonary changes could be found by X-rays only in workers who had been employed for more than five years, and these changes were much more frequent in anthracite miners. The authors describe an increase in reticular tissue, different from the fibrosis of silicosis, caused by the combined effect of carbon and silica dust. The nodules were generally softer than in silicosis.

FLINN, SEIFFERT and others have studied the situation in three bituminous coal mines in the State of Utah.[14] The dust content of the air in these mines was very high. The quartz content of the dust, however, was not exactly determined. Among 507 coal miners, 58 per cent. of whom had been working 10 years or more in the mines, there were 16 cases (3.15 per cent.) of slight anthraco-silicosis, while in a previous examination of

anthracite workers, among 2,711 men, not less than 616 (22.7 per cent.) were suffering from silicosis; of these, 82 were in the second, or moderate, and 24 in the third, or advanced, stage.

Another study, dealing with the conditions among non-ferrous metal-mine workers in Utah was published later.[15] Among 727 workers examined, 66 cases of silicosis (9.1 per cent.) and 42 border-line cases were found.

CLARKE and MOFFET examined 774 workers in bituminous coal mines in Massachusetts.[16] They found only eight cases of silicotic nodulation: a rock driller who had worked 11 years, an undercutting-machine operator working for 16 years, and five coal-loaders working for 23 to 36 years in the mine, and one worker who had worked in other mines before.

The results of the examination of coal miners in the Donetz Basin in the *U.S.S.R.* were similar to those obtained in South Wales.[17]

It must be emphasised that the pneumoconiosis seen in coal miners is a silicosis altered by coal dust.

GRIESHAMMER[18] investigated, by means of experiments with animals, the reaction of the lungs to brown coal (lignite) dust. This dust caused neither fibrotic nor other permanent changes in the lungs.

There is only *one* dust, besides quartz, which is commonly known to cause severe lung diseases, but different from silicosis: the dust of asbestos, a magnesium silicate.

Interesting work on asbestosis has been done during recent years by the Italian authors VIGLIANI, MOTTURA and FAGIANO.[19]

Some clinical aspects of asbestosis were discussed by the Germans ROEMHELD, KEMPF and WEDLER.[20]

An important paper on "The Dust Hazard in Tremolite Talc Mining including Roentgenological Findings in Talc Workers" was published by SIEGEL, ROSS SMITH, and GREENBURG.[21] The talc of these mines is of the asbestine variety, mixed with tremolite and anthophyllite. The content of free silica is 1 per cent. or less. Among 221 tremolite talc miners and millers, advanced fibrosis was found in 32 men who had worked for 10 years or longer in these mines. Apart from this fibrosis, which was of a fine diffuse type with a roentgenographic appearance of granulation or nodulation, deposits of an opaque material, "talc plaques", were seen roentgenologically in the periphery of the lungs. The men suffered from dyspnoea, cough and fatigue. The illness had a disabling character.

PORRO, PATTON and HOBBS[22] examined 15 cases of pneumoconiosis, with five post mortem examinations. Talc mining and milling were the obvious cause of the pneumoconiosis in all these cases. The lungs were stiff, showing weakly outlined grayish bands and nodules in the parenchym. The authors point out that "diffuse haziness is the most constantly noted roentgenographic abnormality, and it is probably the earliest change detectable in pneumoconiosis due to talc".

GARDNER[23] also considers a homogeneous haze as the characteristic phenomenon of X-ray pictures in asbestosis.

The effect of free silica is a chemical irritation, not a mechanical one. The effect seems to be different in asbestosis. GARDNER[23] writes (p. 316): "Experimental observations seem to be indicating that asbestos fibres cause fibrosis in the lungs because they act as *mechan-*

ical irritants in an organ whose function involves considerable movement".

Talcs appear to be capable of producing a lung disease similar to asbestosis, but with some added silicotic signs. It must be emphasised, however, that no other silicates, except asbestos and talc, have been proved to produce serious changes in human lungs.

There are many problems connected with silicosis. One of them is the question how other dusts, inhaled simultaneously with quartz dust, modify the X-ray pictures and the pathological findings. The inhalation of pure quartz (stone-cutters, grinders of sandstone) gives X-ray pictures and pathological changes different from those found after inhalation of quartz dust mixed with coal dust (coal miners) or with clay (potters), although the principal fibrotic changes of the tissues are always the same. Further investigations are, however, necessary to clarify this problem.

In 1937 DENNY, ROBSON and IRVIN published their first article on "The Prevention of Silicosis by Metallic Aluminium".[24] A second article in 1939[25] reported extensive chemical investigations. Experiments *in vitro* had shown that by adding metallic aluminium to a suspension of quartz, the solution and the poisonous effect of quartz was stopped because the quartz particles were wrapped in an insoluble and impermeable coating of gelatinous hydrated aluminium. Animals could be protected against silica dust by adding 1 per cent. of aluminium dust to the total amount of dust.

These amazing findings were corroborated by studies undertaken by R. JAEGER and F. JAEGER.[26] If very fine silica and metallic aluminium were mixed *in vitro* in certain proportions, no free silica remained but all of it was transformed into water containing aluminium silicate (clay substance). Thus both aluminium and silica had been made innocuous. This fact, however, has no bearing on the damaging effect of aluminium as such, on which GORALEWSKI[27] has reported.

JOETTEN[28] tries to prove by means of animal experiments that there is no true antagonism between the inhaled quartz and aluminium, but his experiments are not convincing. Further investigations are necessary to solve this important problem.

The question whether silicosis can develop in the lungs without the co-operation of the tubercule bacillus is settled. There is no longer any doubt that silicosis can develop independently of tuberculosis. A small group of French physicians are the only opponents of this conclusion; but they have prevented the inclusion of silicosis in the list of compensable occupational diseases in *France* for a long time. COUROZIER and MAGNIN[29] assisted by MARTIN reported on a large number of observations contradicting the opinion of this group; and, in 1941 a report was submitted by the Industrial Hygiene Commission stressing the fact that in *France* silicosis constitutes a definite risk to approximately 200,000 workers. In its report the Commission also presented a draft Decree concerning the compensation of silicosis and silico-tuberculosis, together with a draft notice concerning the technique to be used in making radiographic and radioscopic examinations. This report, prepared by Professor DUVOIR has also been published in book form.[30]

During the past few years the medical aspects of silicosis have been thoroughly investigated: the differential diagnosis between silicosis and tuberculosis; the X-ray findings; the chemical analysis of

the lungs; the difficulties of expert opinion on compensation. All these questions are exclusively medical and will not be discussed here.

The effects of other inorganic dusts have also been studied and the results published recently. KOELSCH described "Iron Pneumoconiosis in Electric Welders".[21] In 18 out of 40 welders working with unsheathed steel electrodes, he found more or less marked deposits in the lungs. He explains this phenomenon as follows: "Fine iron particles are inhaled into the alveoli and then transported to the lymphnodes. They are gradually covered by fibrous tissue, but there is no proliferation of fibrous tissue and no generalised fibrosis as in silicosis, which develops only in welders working with certain sheathed electrodes."

A critical survey of the literature on pneumoconiosis caused by sulphur was published by SCHIAVINA.[22] Animal experiments with injection of sulphur into the pleural cavity have shown results completely different from those of any other dust injected in the same way.

The dust diseases caused by the inhalation of organic dust have received more attention recently. Byssinosis is now a compensable disease in *Great Britain* in spite of the fact that its cause has not been satisfactorily explained. The Workmen's Compensation and Benefit (Byssinosis) Act of 20 November 1940 provides for the payment of compensation or disablement benefit in the case of male workmen who have died from or become totally and permanently incapacitated for work as the result of the respiratory disease known as byssinosis. The compensation is given to men who have been employed for not less than 20 years in cotton card rooms or other specified parts of factories engaged in the spinning of raw cotton, and is administered by an administrative board. The Secretary of State may grant disablement benefits to those workers who, though disabled by byssinosis, have not been employed since the compensation scheme came into operation.

In the *United States*, NEAL, SCHNEITER and CAMINITA[23] have published a "Report of Acute Illness among Rural Mattress Workers using Low-Grade Stained Cotton". The workers used a very low-grade dusty stained cotton. Nearly three million families in 46 States of the Union were engaged in this type of work in 1941, and reports of illness came from 25 States. In one State alone over 700 cases of illness were reported. The disease is characterised by a sudden onset, occurring one and a half to three hours after the exposure, and is of short duration (24 to 48 hours). The symptoms are: headache, cough, pains in the joints, tightness around the chest, vomiting, diarrhoea, vertigo, rise of temperature, and high leucocytosis. The illness is caused by a specific bacteria, present in the cotton dust, which is called "cotton bacteria" by the authors of this report.

Another recently detected disease is "bagassosis".[24] "Bagasse" is broken sugar cane after the extraction of the sugar, and has been used recently as a heat-insulating material. It causes an acute inflammatory lung disease among the workers, giving the radiological picture of influenza-bronchopneumonia. The pneumonia usually undergoes a slow solution and recovery follows, but chronic cases also seem to occur with long-lasting dyspnoea.

A group of workers called "grain-handlers" were examined in the *United States* by SMITH, GREENBURG, and SIEGEL.[24] These workers unload grain-carrying ships or work on grain elevators, and some of them were suffering

from "grainfever", *i.e.*, general malaise, pains, chills, fever. These symptoms used to set in two or three hours after the men stopped working, and usually disappeared the next morning. In some cases the symptoms appeared when the men first began to work, in other cases at the beginning of every season or whenever they had been away from work for several weeks, or when the dust was concentrated, or when certain grains were handled.

This disease may be a foreign-protein reaction, an allergic reaction similar to "zinc fever". The X-ray pictures of the lungs sometimes showed pulmonary fibrosis or non-specific lung infections. Another effect of this kind of work was an irritation of the mucous membranes of the conjunctiva and the upper respiratory tract.

New Diagnostic Methods

The diagnosis of silicosis and of other changes in the lungs caused by dust is possible only with the help of X-ray pictures. Since the usual X-ray pictures are comparatively expensive, a new method has been elaborated, the microphotography of the X-ray picture seen on the usual radioscopy-screen. The great advantage of this method is that a large number of pictures can be taken in a short time at low cost. The cost of the usual X-ray pictures (14 x 17 inches) is 65 cents, while a microphotographic picture (4 x 5 inches) costs 6 cents, and one (35 x 35 mm) 3 cents. This method is therefore used for mass examination of soldiers, workers and children.

Different sizes of microphotographical pictures are used in different countries. In *Great Britain* the usual size is 35 x 35 mm. The "Technical Sub-committee on Apparatuses for Mass Miniature (Indirect) Radiography", in its report on tuberculosis in wartime[36], states that good results are obtained when certain conditions for a good picture are fulfilled, but that "no diagnosis other than suspected abnormity" should be made from the miniature film. The same holds true for silicosis. In *Germany*, where smaller films (24 x 24 mm) are used, the results were not satisfactory, according to Saupe[37], for detecting slight degrees of silicosis. In the *United States of America* two sizes of microfilms are used: 4 x 5 inches and 35 x 35 mm. It must be emphasised, however, that under certain circumstances the diagnosis of silicosis is more difficult than the diagnosis of tuberculosis. A report by Plunkett, Weber and Katz[38] gives a clear picture of the advantages of microfilms. It may be said, in conclusion, that microscreen photography makes mass investigations possible at low cost and that this work can be done in a very short time (50 and more pictures in one hour); but if the screen picture is not very clear the usual X-ray picture has to be taken.

Reliable apparatus for collecting dust from the air exists, but the counting of dust particles of a size under 0.001 mm under the microscope is very troublesome. Therefore, a simplified technique of counting has always been sought, so far without complete success.[39]

Another difficulty is to distinguish, in dust samples as well as in the lungs, between quartz particles (which are the only ones that cause damage) and other dust particles. X-ray diffraction has proved to be a good method.[40] X-ray diffraction is also used for the measurement of these small particles.[41]

Preventive Measures

Many years ago the United States Bureau of Mines established schedules of

minimum requirements for respirators and other protective apparatus, and since then it has approved many protective respiratory devices. From time to time the list of such approved devices is published.

In 1940 a "Committee on Devices for the Protection of Workers from Dust" was set up in *Germany*. This committee publishes its findings concerning both suitable and unsuitable devices in the journal *Staub* (Dust), with descriptions and pictures of both. Among those declared unsuitable are many old and well-known respirators. Among five devices for sand-blasting only three were found suitable. The directions for the construction of such devices prescribe an overpressure and a supply of at least 80 litres of air per minute in the headpiece. They have to be constructed in such a way as to make it impossible to go below this amount. For the time being the use of air taken from compressed air mains is permitted, but if the apparatus is connected to such an air main a high-grade filter of activated carbon has to be inserted.

On 1 August 1940 new regulations were published in *Germany* concerning the establishment of new plants using asbestos.[42] They prescribed the installation of devices making it possible to measure the depression at various points of the ducts of the exhaust system. The machines must have individual drives to avoid raising dust. Pneumatic or other enclosed conveying systems must be installed as far as possible. Before erecting new premises or making important changes, the establishment must consult the Accident Insurance Association or the Office for the Combating of Dust.

It may be mentioned here that in several countries the establishment of a water supply system and the use of wet drilling is prescribed for mines.

In the *United States* a symposium on "Tuberculosis in Industry" was held in June 1941, in the Saranac Laboratory and the lectures given on that occasion were later published by the National Tuberculosis Association.[43] Besides lectures on clinical and statistical problems, there were lectures on predisposing factors (dust, fumes, gases, fatigue, trauma) and on certain industries (tobacco, needle trades, gypsum and cement industries, steel plants, foundries). According to the summary of the findings by L. U. GARDNER, Director of the Laboratory: "Of the environmental factors . . . the standard of living is universally accepted as significant. Of all the accessory environmental factors peculiar to industry, only free silica is accepted without challenge." Fumes and gases, lead, high temperature and humidity could not be proved to favour the development of tuberculosis. Of the dusts only those high in free SiO_2 increase the susceptibility to tuberculosis. In modern tobacco and cigar factories there is little excess tuberculosis.

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