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Tuberculosis as an industrial health hazard, with particular reference to the silica industries

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Tuberculosis as an Industrial Health Hazard, with
Particular Reference to the Silica Industries.

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1934

PREFACE

Thru personal experience in a certain industry in which dust formerly was liberated in rather large quantities, the author had observed that many of the workers after several years of exposure, contracted tuberculosis and eventually died from that disease. It has been a matter of interest and speculation to him what relationship existed between quartz dust and tuberculosis. This thesis is the result of that interest, and it was the hope of the author to learn why these workers died of tuberculosis. To what extent these hopes were fulfilled by the writing of this thesis, will be left to the reader's own conclusions after he has perused its contents.

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INTRODUCTION

Tuberculosis has been a greater enemy of the human race than almost any other disease. Its devastation has surpassed the wanton destruction of savages. The disease has been so prevalent that according to Mock (1920) autopsy statistics show that seventy to eighty-five per cent of all people have at some time during their life been infected.

It has long been recognized that certain occupations predispose to pulmonary tuberculosis, and in these "dangerous" occupations the death rate for this disease is far higher than in the general population. These observations have been made in British, European, and American experience by numerous investigators, Gye and Kettle (1922), Barnard (1934), and others, and that this is particularly so among those who work habitually in atmospheres laden with silica dust.

During the past few years numerous factors have stimulated interest in the phases of tuberculosis and silicosis in industry. The International Conference on Silicosis which met at Johannesburg, South Africa in 1930 and at which Great Britain, Canada, Australia, South Africa, Germany, Italy, Belgium, The Netherlands, and The United States were represented has stimulated interest in these phases. Then, over the past few years there has been such a rapid accumulation of claims for compensation, litigation, and experience in industrial disease cases, largely based upon silicosis, that the entire industrial field has been taken by surprise. For example, the report of the Committee of Five Insurance Companies (1934), citing the case of Wisconsin, ascertains

that approximately ten per cent of its total compensation losses will be chargeable to occupational diseases, most of which are for silicosis. Insurance rates for industries exposed to silica dusts have been increased for this reason, and it is stated that in Ontario and South Africa the cost has risen to a point requiring a rate that is almost prohibitive. It has been Watt's (1930) observation that the payment of compensation for miner's phthisis is a very great burden on the gold mining industry in South Africa, and may make the difference as to whether a low-grade mine may run at a profit or not.

Action for damages on account of silicosis and tuberculosis is being brought with increased frequency in the civil courts, where very large sums of money are claimed.

Bills for including dust diseases, particularly silicosis, in the compensation legislation are continually under consideration in the various states.

And on top of it all, Harrington (1933) concludes that knowledge of a definite, dependable, accurate character about dust disease is woefully lacking, and there is probably no more fruitful field for concerted, unbiased, disinterested investigation than that in connection with the health harmfulness from dust. Furthermore, the literature on dust disease is decidedly voluminous and one can substantiate almost any kind of position or statement or theory by available data in the published literature.

Therefore, not only are silicosis and tuberculosis of medical interest, but also of far reaching economic, legal,

legislative, and scientific concern.

A review of statistics, although quite incomplete, will show morbidity and mortality rates from tuberculosis that present quite conclusive evidence as to the destructive nature of occupational siliceous dusts. The Milner Commission found in the period 1902-1903 that 15.4 per cent of the miners on the Witwatersrand gold fields in South Africa were affected by Miner's Phthisis, and another 7.3 per cent were suspected cases, giving a prevalence of at least 23 per cent, which is felt is probably too low (Irvine and coworkers, 1930). Mock (1920) quoting from Crum shows that in Montana during the period 1907-1914, the mortality from tuberculosis among copper miners over the age of 15 was 37.9 per cent, while in the same registration area the general mortality among males from tuberculosis was 14.1 per cent. Kober and Hayhurst (1924) quoting from Niesztyka report that 76.5 per cent of all the deaths among the sandstone workers at Hanover are caused by tuberculosis, and their average age is only 35.1 years. Sayers (1930) states that in New York City an examination of 208 men exposed to rock dust in subway or tunnel construction, revealed silicosis in 57 per cent. In the lead and zinc mines of the Pilcher mining district of Oklahoma and Kansas, studies by Sayers and his co-workers (1933), show that 21.3 per cent of all the miners were definitely afflicted with silicosis, and 4.75 per cent with tuberculosis. Further statistics revealing the harmful nature of silica dust will be quoted from time to time through-out this paper.

ETIOLOGY

Tuberculosis is not a disease of industrial life alone, although unquestionably it has caused the greatest destruction among the poor working classes in our industrial centers. It has its highest morbidity and mortality rate among these poor working classes. They become centers of infection and spread the disease to all other walks of life. In this connection, Mock (1920) cites eight factors which are predisposing to tuberculosis among employees, as follows:

1. Hereditary predisposition and family infection.
2. Poor housing and living conditions and other community conditions.
3. Alcoholism and other excesses.
4. Unsanitary working places and working conditions.
5. Tuberculosis infected employees among the working force.
6. Prevalence of other diseases.
7. Injuries to the chest, and other injuries.
8. Specific occupational hazards.

It is with these specific occupational hazards that we are concerned with as predisposing to tuberculosis. Among the industries subject to exposure to silica dust with subsequent danger from silicosis and tuberculosis are:

1. Gold, Tin, Zinc, and to a certain extent, Coal mining.
2. Quarrying industries, especially of granite.
3. Sandstone cutting and grinding.
4. Refractories industries, as manufacture of silica bricks.
5. Millstone cutting and grinding.
6. Rock crushing.
7. Excavation and construction work in rock.
8. Pottery industry.
9. Sandblasting.
10. Foundry industry.
11. Flint dust workers, and various miscellaneous industries including porcelain, asbestos, and other industries.

There is considerable diversity of opinion as to what constitutes the harmful character of the dust, and numerous

theories have been advanced.

It was thought by many that some dusts were not harmful, such as coal dust, and that even some dusts had a beneficial action on the lungs, but much of the present day authoritative opinion is that all dusts if breathed in sufficient quantity and over a long enough period of time, will cause pathological conditions of the lungs. However, it is agreed by practically all, that the chief source of danger from dust is its silica (SiO_2) content, and that silicosis has far overshadowed other pneumoconioses in importance. The question then naturally arises why should silica dust of all dusts be singled out as the one producing a harmful pneumoconiosis with subsequent danger from tuberculosis?

It formerly was considered by many that the inhalation of dust, hard and gritty, also sharply angular, was the main cause of the pneumoconiosis, and that the damage to the lungs was by a mechanical irritation. Some believe that the relative insolubility of silica dust is a factor in its harmfulness.

Collis suggests that the chemical property of acidity due to the element silicon, may render the dust particles capable of entering into and modifying the colloidal structure of protoplasm. The trend of opinion, however, is in favor of the view that silica causes fibrosis by acting chemically upon the tissues and not by virtue of its physical properties (Gye and Kettle 1922). Gye's work has been based on the known facts that colloidal silica is the most easily formed soluble form of silica, and that living matter (soil bacteria)

is able to break up mineral silicates with formation of soluble silica. He judged it probable that the fibrosis brought about by finely divided silica was due to the slow formation in the tissues of silica sol, the sol acting as a cell poison, although direct proof of this contention as yet has not been found possible. Heffernan and Green advanced the hypothesis that pulmonary silicosis is brought about by the colloidal activity of fresh silica hydrosol derived from the hydration of quartz dust in the lungs and not by any poisoning of the cell protoplasm by soluble silica (Sayers 1930).

Harrington (1933) states that it appears that the quantity of dust in the air breathed more or less continuously, together with its lack of solubility, governs the hygienic harmfulness of dust to workers much more than do the specific physical or chemical qualities of dusts. Green (1934) believes the harmfulness of a particular dust is not connected merely with the amount of dust in the air, but the number, and more particularly, with the size-frequency (fineness) of its particles. It is believed by many that it is the finer particles of dust that produced the greatest damage in the lungs. According to McCrea no particles have been found in the lung tissue which exceed ten microns in diameter, and over 70 per cent of these found are one micron or less. In order to pass into the alveoli, dust particles must, therefore be very small. Otherwise, they are caught in the mucous secretions and are expelled by the ciliated epithelium.

The degree of silica content of dusts and the intensity

of exposure were found to be factors of utmost importance. Coal, limestone, cement, marble and other similar substances have long been observed to be relatively innocuous as far as pneumoconiosis and tuberculosis are concerned, and this is almost universally believed to be due to the absence, or low content of, silica. On the other hand, it has been observed that substances containing high percentages of silica produce the symptoms ascribed to silicosis, which very frequently is followed by tuberculosis. The period elapsing before symptoms of silicosis occur varies with the different occupations and intensity of exposure.

Granite dust contains about 30 per cent of free silica as quartz. Extensive investigations by Russell and his co-workers (1929) from the United States Public Health Service, were carried on in the granite industry in Barre, Vermont, which is claimed to be the granite center of the United States, during the period 1924-1926. They observed that since the adoption of the hand pneumatic tool for cutting granite in the latter part of the Nineteenth Century, there has been a rapid rise in the mortality from tuberculosis. These tools cut the granite much faster than the old hand chisels and cause more dust. The mortality from tuberculosis rose from 1.5 per thousand in 1890-1894 (which is not above that of the general population at that time) to 19.6 per thousand during 1924-1926, while the mortality from tuberculosis in the general population has decreased. Studies into the intensity of dust exposure revealed an average dust count of 59.2 million particles per cubic foot for all hand pneumatic tool operators, to 1.9 million particles for office

employees, while the general plant atmosphere gave an average dust count of 20 million particles per cubic foot. In the group with the heaviest dust exposure, it was found that after four years of service, all of the granite workers seemed to have developed at least an early case of silicosis, and by nine years, approximately 90 per cent had developed to more advanced stages of silicosis. In the group with the least exposure to the dust only two cases of silicosis occurred after ten years of exposure, and only one case of moderately developed silicosis after six years exposure. In the office workers there were no deaths from tuberculosis during the period of study, while in the group exposed to general plant dustiness only one death occurred, while the rest of the mortality from tuberculosis occurred in the group subjected to the greater dust exposure. Another characteristic observed was that there was no great mortality from tuberculosis up to twenty years of service, but after that period or approximately after the age of 50, it rose sharply. While above 50 years of age the mortality curve of pulmonary tuberculosis in the general industrial population began to decline.

According to Nicholson (1923) workers in the central district of Derbyshire show a high mortality from phthisis. They are chiefly occupied in quarrying, mining or cutting gritstone, burrstone, and ganister, which all have a high content of pure silica (85 to 96.4 per cent). In the case of gritstone and quartzite workers symptoms of silicosis after 5 to 10 years exposure, occurred, while in the case of ganister and granite workers, after 10 to 20 years, and in pottery

workers a period of over 20 years exposure is the general rule. Oliver puts the period in the rock drill workers of the Witwatersrand, South Africa, as from 6.8 to 8.8 years. Most authorities agree upon an average period of exposure of from 10 to 15 years as being necessary for the production of symptoms-- the American workers put it at 15 years. Statistical returns from Derbyshire and elsewhere in Great Britain show a phthisis death rate among gritstone workers alone in this area from 1900 to 1910 of 13.7 per thousand employed males, as against the standardized phthisis death rate of 0.77 per thousand in the district. In the limestone area it was 1.71, and in the coal area 0.68. Derbyshire gritstone contains 96.4 per cent silica, limestone only 0.2 per cent, and pure coal none.

Flint is practically pure silica. Flint knappers of Brandon have a death rate from phthisis, according to Collis, of 41 per cent as against a rate of 1.6 per cent for all males over 15 years of age in England and Wales in 1900 to 1902. Sayers (1930) quoting from investigations by Hayhurst and his co-workers in one of the sandstone districts of Ohio, reveals pulmonary pathology in 55.1 per cent of the workers, including 38.5 per cent with silicosis in various stages. This rock has an average content of 92.15 to 97 per cent crystalline silica.

Numerous other statistics and experiences might be quoted, but suffice it to say that there is general agreement that the greater the free silica content of the dust and the greater the intensity of exposure, the greater is the morbidity and the mortality from silicosis and tuberculosis. After consideration of the varied and diversified opinions

of its members, the Johannesburg Silicosis Conference in 1930 summed up the factors responsible for the harmfulness of silica dust as follows:

1. The amount of dust inhaled.
2. The percentage of free silica contained therein.
3. The size-frequency (or fineness) of the particles inhaled.
4. The nature and sort of such other substances (including vapors and gases) as may be inhaled simultaneously, or otherwise.
5. The powers of resistance of the individual concerned.
6. The presence or absence of a complication by an infected process.

And, furthermore, in view of the lack of sufficient knowledge, further investigations as to the causes of harmfulness of silica were urged by the Conference.

(Badham 1931). This much needed information, it is felt, is necessary to lead to more rational methods of estimating the hazards due to dusty atmospheres.

SYMPTOMATOLOGY

Before entering into the discussion of the symptomatology, of silicosis and tuberculosis, the following definition of silicosis by the Pennsylvania Compensation Commission (1933) aptly summarizes its clinical manifestations:

"Silicosis is a disease due to breathing air containing silica (SiO_2), characterized anatomically by generalized fibrotic changes and the development of milliary nodulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present), and by characteristic X-ray findings."

At first there is little to note beyond irritation of the mucous membranes of the nose and throat, the sneezing and coughing which ensues being nature's attempt to dislodge the dust. Silicosis develops slowly as a rule, so insidiously in fact, that it is difficult for the patient to assign a date to the onset of symptoms, of which dry cough in the morning or on leaving work at the end of the day is one of the earliest symptoms. Kober and Hayhurst (1924) speaking of the symptoms which a gold miner experiences, state that what appears to be a bronchial cold, with or without pleuritic pains, attended by a sense of increasing debility is an indication that the lung has become affected by mine dust. One feature stands out prominently throughout the whole course of gold miner's phthisis, viz; dyspnoea on the slightest exertion - a shortness of breath far in excess of the physical signs. The general appearance of the affected miner is frequently that of a man still in health. He may be slightly bronzed and looks weather-beaten, and yet

the disease has probably already got a good grip on his lungs for on physical examination the chest is observed to expand feebly, there is a diminution of one inch or more in the ratio between inspiratory and expiratory measurements, also impairment of the percussion note at the base of the lung toward the axillary area with deficient respiratory murmur. The pulse is almost invariably quickened.

In the later stages the respiratory murmur may become coarser, crepitation may be heard, also a cardio-respiratory murmur along the left border of the cardiac area. The expectoration varies. In the early stages, if there is little bronchial catarrh, expectoration may be scanty, stringy and pearly-white in character; in the later stages it may be mucopurulent, bluish-black and rich in particles of silica. Since in a considerable number of persons dying from silicosis no sign of tubercle is found in the lungs or in the body generally, silicosis must therefore in its early stages at least, be regarded as a non-tuberculous disease. When tuberculosis develops, not only is it secondary to the pneumoconiosis, but, there occurs an alteration in the symptoms. Emaciation, rise of temperature, night sweats, mucopurulent sputum - frequently containing Koch's bacilli, rales, and the other symptoms and findings of tuberculosis appear.

Granite workers (Russell and coworkers 1929) in the early stages develop a non-productive cough, and occasional pains in the chest, usually of a transient nature. The pains are part of the development of pleurisy which is coincident with the progress of silicosis. The cough is believed to be

probably not of a pulmonary origin, but, resulting apparently from mechanical irritation of the upper respiratory tract. Here again, as in the gold miners, dyspnoea is the principal complaint of the workers with silicosis, which in the early stages is noticeable only on exertion, and which increases in direct proportion to the exposure to dust. Silicotic patients are loath to give an accurate history of anything referable to their chest conditions, as they have a profound fear of tuberculosis. For example, many times they state they are not dyspnoeic, when it is evident they are. In the early cases there is generally a beginning of impairment of resonance and a diminished tactile and vocal fremitus with diminished breath sounds, which progressively diminish as the condition progresses, because of poor resonating quality of the lung tissues. Later there is a limitation of chest expansion, bilateral in nature. The average chest expansion of the granite worker on beginning his occupation is 7.7 cm; after 20 years of work it is 6.4 cm. and after 35 years in granite it is 3.6 cm., while the corresponding figures for vital capacity are 4, 3.8, and 3 liters.

There is every presumption that this decreasing chest expansion reflects the developing fibrosis. As the condition progresses this restriction of chest expansion becomes greater, and there is a hyperplasia of the muscles of the chest, due to the increased respiratory effort. In these more advanced cases there is still greater evidence of impaired resonance, and in the most advanced cases evidences of consolidation. When the condition is severe enough to cause loss of time from

work, it is usually a sign of incipient tuberculosis. In general, however, patients with silicosis appeared to be healthy and well nourished, and except in advanced cases, there is usually no evidence on casual examination of any impaired physical ability.

These symptoms in general seem to follow in the wake of silicosis whatever the cause and the industry involved. In particular, unproductive cough, dyspnea and transient pains in the chest seem to be the most outstanding symptoms.

PATHOLOGY

It is now generally agreed that dust found in the lung tissue gains access by way of inhalation. Other paths of entry, such as the digestive tract, may for all practical purposes be neglected. The respiratory apparatus possesses definite means for ridding itself of the dust, i.e., (1) the power of bronchial secretions to dissolve dust particles; (2) the sweeping out action of the cilia lining the bronchi and the bronchioles; and (3) the phagocytic action of macrophages which remove particles from the alveoli and carry them either within reach of the cilia, or through the alveolar walls into the lymph stream. These means may, however, be overwhelmed if the amount of dust inhaled is excessive, or if the period of inhalation is prolonged.

A great deal of experimental work has been concerned with the mode of entry of dust into the lungs. Now, there is general agreement that the very fine particles of dust which reach the lung alveoli are taken up by phagocytes, which, according to most authors, are derived from the endothelium of the lung capillaries, and according to others, from alveolar epithelium, and the dust-laden phagocytes migrate to the nearest lymphoid tissue and gain access to the closed lymphatic vascular system which drains the lung. Most dusts are removed from the lungs in this way through the lymphatic channels, but, silica, for the most part remains in situ, stimulating the formation of new connective tissue. (Gye and Kettle 1922). Other observers (Sayers 1930) are agreed that the phagocytes, when overloaded with dust, lose their motile power, collect in the lymphatic

channels, and lead to a reaction, with formation of fibroblasts in the surrounding tissues.

It has been found that Irrespective of the particular kind of dust which has led to the development of pneumoconiosis, one and the same structural change is invariably present in all affected lungs, if only sufficient time is given, namely, an increase of the fibro-connective tissue, an interstitial pneumonia in fact, accompanied by the presence of pigment. As Kober and Hayhurst (1924) describe it, the fibrous tissue tends to assume in places, concentric formation, the fibrosis being particularly well marked around blood vessels and small bronchi. The blood vessels become thickened as a result of endarteritis or in consequence of prolonged venous congestion. The fibrosis increases in response to the progressive nature of silicosis, and the sponge-like structure of the normal lung tissue is progressively replaced by the solid fibrous tissue.

X-ray pictures of chests of granite cutters (Russell and co-workers 1929) indicates that silicosis follows a rather definite form in its development. There is a general thickening of each hilum and of the trunk shadows, the latter being indicated by linear markings throughout. The enlargement of the hilum shadows is incident to lymphatic blockage and to the deposition of dust in the glands. Hilum shadows are increased in size and density very early, and become more pronounced as the exposure to dust continues. In advanced cases there was confluence of shadows, forming consolidations. The densities next in order in appearance and prominence were linear markings, or trunk shadows, which became diffused through-

out the lung as silicosis advanced. They extended from the hilum to the outer zone laterally and along the course of the larger bronchi in the lower lobes. These linear shadows in the outer zone at the same level as the hilum were conspicuous even in cases of comparatively short exposure to dust. Linear densities extend upward as silicosis advances, and the apices are the last to be involved. The angles of the diaphragm were not always clear, particularly at the cardiac end, because the thickening along the larger trunks overlaps and produces a hazy appearance. The overlapping of thickened pleura also adds to the densities in this region. Irregularities of the diaphragm were frequently observed, being present in 40 per cent of the cases, and as the condition advanced there was an increase in changes of the diaphragmatic shadows. It is evident from clinical and X-ray findings that a chronic pleurisy is a definite part of the pathology of silicosis. Pleural changes are attributable to the irritating action of the dust which reaches the pleura through the lymph streams.

Radiographic examinations of the chests of gold miners show three stages of pathology according to the classification adopted in South Africa (Nicholson 1923).

First stage. - Mavrogordato claims there is a pre-X-ray stage of silicosis because X-ray examination fails to reveal any evidence of silicosis until the fibrosis of the lymphatic vessels has begun. After this pre-X-ray stage the hilus shadows are always increased. There is also an undue prominence of trunk shadows and finer linear markings. The descending

trunk shadows are more noticeable on the right side, the heart shadow obscuring those on the left.

Second stage. - This is characterized by a more or less uniformly arranged mottling diffusely scattered throughout the lung structure, due to the deposit of dust in the lymph spaces, cells, and fibrous tissue spaces, together with some localized fibrosis. This mottling is of a characteristic nature and is called by the South African workers the "Snow-storm" appearance.

Third stage. - The fibrosis in this stage has become more general, lighted up here and there by emphysematous patches, with the mottling showing a tendency to become denser and to coalesce, forming denser and more uniform shadows. The condition in this stage is indistinguishable from advanced tuberculosis. The condition is always bilateral; it commences at the hilus and spreads at first toward the base, also in contrast to the behavior and spread of a tuberculous fibrosis which is more often than not unilateral, and which, commencing at the hilus, spreads first toward the apices.

The fibrosis of the lungs explains many of the symptoms and findings of the silicotic individual. The dyspnea can be attributed to a lessened aerating surface of the lungs because of the replacement by solid fibrous tissue. Sometimes there is such a marked replacement by fibrous tissue that it is a wonder how respiration can be carried on at all. The decreased chest expansion no doubt, is an expression of fibrous tissue formation, as well as are the impaired resonance, and decreased fremitus and breath sounds.

A study of the morbidity and mortality statistics shows strikingly the significance of tuberculosis as a complication of silicosis, and the question arises, how does the silicotic fibrosis aid the establishment of tuberculosis? Much work has been done on this problem, but, as yet the explanation is still to be sought. The disorganization of the lymphatic system in silicosis has been recognized by most authors as a potent factor in the increased susceptibility to tuberculosis. In addition, Gardner regards silica dust and the tubercle bacilli as irritants, and the two acting together are capable in a short time, of setting in motion a series of reactions on the part of the lung which neither alone is capable of initiating. By concentration of both in small foci each reinforces the action of the other, resulting in a chronic lesion which is slow to heal.

It might appear as if the avascular state of the fibrotic areas of an affected lung did not lend itself to the reception and multiplication of the tubercle bacillus, but, a suitable nidus is certainly provided in the catarrhal secretion within the pulmonary alveoli.

Gardner experimented on the inhalation of attenuated strains of tubercle bacilli by guineapigs that had not been exposed to silicosis, and on those that were subjected to silicosis. In the first group were found only isolated subpleural tubercles in lung tissue, massive involvement in tracheobronchial lymph nodes, and nothing in other viscera - an infection which was cured by resolution within two years and which never killed the animals. He found that in infected

animals subjected to the daily inhalation of quartz, the tuberculous lesions spread and generally pulmonary tuberculosis - chronic and fatal - resulted. From further experiments he concluded that the virulence of the strain of the tubercle used had not increased, but, that a temporary alteration in environment had been produced in the silicotic lung which favored multiplication of the tubercle bacilli. Kettle experimented with rabbits showing the modification of artificial military tuberculosis by silica. Rabbits inoculated first with intravenous amorphous silica and later with tubercle bacilli, develop more military tubercles than rabbits not receiving the silica injection. In mice with interstitial lesions produced by silica or calcium followed by tubercle bacilli intravenously, the tubercle bacilli were found in much greater number in the silica lesion than in the calcium lesion.

The results of animal experimentation have been subjected to some criticism because there is still a wide gulf between the findings of industrial lung and the results of animal experiments. No one has yet been able to produce by animal experimentation such a fibrosis as that shown by a rock driller's lung, for instance. But, nevertheless, such work should be carried on in view of our lack of knowledge of the relation between silicosis and tuberculosis.

Once tubercle bacilli invade the silicotic lung, the patient rapidly becomes worse and the case practically becomes helpless.

Two distinct groups of tuberculosis were found among the granite cutters at Barre, Vermont by Russell and his co-workers.

The principal differences being found in the ages of the patients, location of the lesions, and the course and prognosis of the disease. The first type of tuberculosis found - which forms the great majority of the cases - was comprised of workers who had a well developed silicosis. The average age of these workers was 49, with an average dust exposure of 28 years. The onset of the disease (tuberculosis) in this group was rather sudden, and ran a rapid course, terminating fatally. The general location of the tuberculous lesion was basal, in contrast to the usual apical location of the lesion. There were no periods of quiescence in any of the cases which came under observation, nor were there any recoveries. The second type of tuberculosis observed, which has been termed concurrent silicosis and tuberculosis resembled the disease in persons not exposed to dust. The lesions were usually located in the apices; the course of the disease was slower than in the older cutters, and the ages of the group ranged from 24 to 38 years, with a length of exposure to dust of from 3 to 13 years, averaging 8 years.

Institutional treatment appeared to have no effect in retarding the development of the disease. Patients were perhaps more comfortable and obviously were less of a menace to their families and to the public, but, the course and prognosis of the disease did no appear to be altered. The average period of time elapsing between the beginning of disability from tuberculosis and death was found to be about 15 months.

Pathological study was made of 8 lungs from employees of the Barre granite industry. Gross appearances: No case of

advanced uncomplicated silicosis was found, all these showing lesions of a coexisting tuberculosis. Five showed pulmonary cavities, occurring in two instances in the apices, and in three others at the base or in the mid lung. Generally the surfaces of the lungs presented extensive pleural adhesions. The tracheobronchial lymph nodes were moderately enlarged. The silicotic nodules of cases complicated by tuberculosis were much more dense, and the tracheobronchial lymph nodes appeared smaller and more shrunken as the silicotic lesions advanced. Microscopic appearances: In the earliest case presented, slight amounts of dust were found in the pleura, septa and adventitial coats of the vascular and bronchial trunks. These deposits were surrounded by a very slight amount of cellular connective tissue. In the alveoli a few free phagocytes containing dust particles were found. As the silicotic condition advances, many alveoli, especially those beneath the pleura, are found completely filled with phagocytes. The pleura show areas of thickening produced by masses of cellular connective tissue or by circumscribed nodules of non-cellular hyaline connective tissue. Massive collections of cellular connective tissue are observed beneath the pleura in the septa and about both bronchial and vascular trunks.

In half of this group the tuberculous process developed and proceeded in a manner similar to that ordinarily encountered; in the other half, the silicosis changed and modified the course of the complicating tuberculosis. These differences in the character of the lesions have suggested the hypothesis that tuberculosis as a complication of silicosis arises both from

endogenous and exogenous sources. The relative frequency of these two types of infection, however, cannot be determined from the study of such a small series of cases.

DIAGNOSIS

The subject of diagnosis of silicosis and tuberculosis in the industries exposed to silica dust has been dealt with more or less indirectly in the preceding discussions. Briefly, the essential features concerned in the diagnosis are:

1. History of exposure to silica dust.
2. Symptomatology.
3. Physical findings.
4. X-ray studies.
5. Sputum analysis.

With a history of employment for any length of time in any of the industries mentioned earlier in this paper, along with other indications, one should quite naturally think of a silicosis, or even a tuberculosis if the other indications are sufficiently pronounced. The industries in which silicosis and tuberculosis seem to have been the most prevalent and of greatest concern are gold mining, tin mining, granite quarrying, sandstone cutting and grinding, refractories industry - making of silica bricks etc., rock crushing, and excavation and construction work in rock.

As previously described the cardinal symptoms of silicosis are an unproductive cough, progressive dyspnea and transient pleuritic pains in the chest. With the complication of tuberculosis the additional symptoms of a productive cough, loss of weight, fever, night sweats and the other accompanying symptoms of that disease are superimposed.

It seems to be a rather difficult matter to present the employees for physical examinations. Many of the workers in these hazardous industries have the morbid fear of tuberculosis, and they are reluctant to learn the "bad news". Some workers refuse to be examined. The more common physical find-

ings in silicosis are a progressively decreasing chest expansion, impairment of resonance, diminished tactile and vocal fremitus and diminished breath sounds. With the superimposition of tuberculosis, there is a unilateral limitation of expansion, areas of dullness in addition to the general impaired resonance already existing, increase of tactile fremitus over the tuberculous lesion or cavity, change in breath sounds where cavity formation exists, in many cases bronchial breathing near the tuberculous lesion, and rales which is almost a constant finding. For a long period of time during which the worker has silicosis alone he appears to be a picture of health. No doubt this is a cause for his hesitancy to present himself for periodic physical examination.

Radiographic studies of the chest are the most valuable single means of diagnosis of silicosis in general. However, Mavrogordato and others believe X-ray examination does not reveal the earliest stage of silicosis, so the clinical findings must be depended upon for a diagnosis in early silicosis. Later, silicosis presents a rather characteristic manifestation according to X-ray studies. There is a general thickening of each hilum and of the trunk shadows. These trunk shadows are indicated by linear markings from the hilum to the outer zone laterally, and are diffused through the lung tissue. The South African workers ascribe a mottling or "snow-storm" appearance to the moderately advanced silicosis. In the advanced cases of silicosis consolidation of shadows takes place, and in the tuberculous stage, basal cavitation is characteristic.

The finding of tubercle bacilli in the sputum is, of course, definite evidence of the presence of tuberculosis.

PROGNOSIS

In the majority of cases silicosis is a progressive disease, even when the worker leaves the dusty industry permanently and takes to a life in the open. In some cases of early silicosis when the worker has left the industry there has been an arrest or apparent cure of the silicosis, but in other cases the condition has progressively grown worse until the individual finally broke down with tuberculosis, even though he had been removed from dusty industries for several years. This has been the common experience in South Africa, England, Australia, United States and elsewhere.

Russell and his co-workers traced the histories of twenty-four workers who had left the granite industry and carried on various non-dusty occupations. Eleven, at the time of their investigation, showed silicosis in various stages of advancement, while 13 showed tuberculosis, 7 of whom died prior to the termination of the study.

In South Africa, Dr. Irvine (Badham 1931) said of the silicotics who did not leave the gold mining industry that they progressed no worse than the men who left. Dr. George states that in 1922 and 1923, thirty-four miners from Broken Hill, New South Wales, suffering from uncomplicated silicosis, were settled on orchard farms. From the commencement the scheme was a failure. Up to the present time (1930), of these 34 men, 17 have developed or died of pulmonary tuberculosis, about 9 are still on their farms, and some of them have definite tuberculosis. It was observed in South Africa that of the gold miners with a pre-X-ray stage of silicosis, which is the earliest stage, who left the industry, most of them

progressed unfavorably. It is believed by some that in such cases there must be an "infective" silicosis present of a peculiar latent tuberculous nature. Sayers (1930) states that the outlook of the individual case is mainly dependant upon whether the infective element remains "bottled up" and inactive, or whether, on the other hand, it becomes active, or a further infection occurs from outside the lung. This difficult and as yet not fully understood complication - namely "infective silicosis," plays a very important part in the question of prognosis.

TREATMENT

It is quite generally agreed that there is no treatment for the cure of silicosis. Medicinal treatment or change of occupation after the condition has become established are of no avail. Possibly only in the very earliest stage can satisfactory results be obtained in some cases by retiring from the dusty occupation and taking up work in the open air. Once fibrosis has set in, it is likely to be progressive and eventually terminate in pulmonary tuberculosis. Prevention of exposure to dust, therefore, is the only treatment and cure of silicosis, and tuberculosis caused by silicosis.

To successfully combat the dust hazard a safe limit of dustiness must be determined. The investigations in the Barre, Vermont granite industry showed that the limit of safety lay somewhere between 9 and 20 million particles of dust per cubic foot of air. The South African standard for safety in the gold mines is 8.5 million particles per cubic foot, while the United States Public Health Service limit of safety is 10 million particles per cubic foot. It will be recalled, for example, that the pneumatic tool granite cutters at Barre worked in an atmosphere containing 60 million particles per cubic foot! It becomes apparent at once that rather drastic methods are indicated to control the dust at its source.

Numerous methods and devices for controlling the dust at its source have been devised. It is agreed that no one method is applicable to all circumstances; the methods must be adapted to the particular industry. It is agreed, however, that in most cases there should be a combination of methods.

The gold mining industry on the Witwatersrand in South Africa has led the way in the study of dust-caused tuberculosis because, as Watt (1930) states, this disease has been more prevalent there than in any other part of the world, and the opportunity for research and study has been made possible by the generosity and business acumen of the leaders of the industry. The vision and initiative of the Consolidated Gold Fields of South Africa some 30 years ago began putting into operation methods for dust control, so that today the absence of dust in their gold mines is as conspicuous as it was by its presence 30 years ago. It would be well to look into the methods of dust control in the gold mines on the Rand, and then discuss other methods of dust control as they apply to other industries.

Since 1916 four kinds of preventive methods have been combined to influence the situation on the Rand as described by Irvine and co-workers (1930). Two of these make up what may be called the "engineering methods" aimed directly against the production or dissemination of dust in mine air:

1) "Dry" methods of prevention - are measures described to secure an adequate standard of ventilation both general and local, to regulate the method and times of blasting, to secure the arrangement of shifts, and the times and manner of hoisting so that there should be no unnecessary exposure of any person to dust and fumes, and measures of a similar character.

2) "Wet" methods of prevention - by which one means measures which depend directly on the use of water to allay

dust at its source, or to remove it from the air when formed. The use of water in drilling machines and in blasting has been found to reduce the amount of dust generated in drilling and blasting by 97 or 98 percent by weight. However, it is felt that not all of the fine particles of dust are al layed by the water and which still are a source of danger, and that this phase requires further investigation.

The other classes of preventive measures one may call the "medical measures".

3) The "initial examination" of recruits and rejection of those suffering from respiratory diseases or those who appear unduly liable to contract such diseases.

4) The "periodical examination" which aims at the detection of all cases of silicosis or tuberculosis amongst the working miners, and consequent removal of all found suffering from tuberculosis from underground work, thus removing potential sources of dissemination of infection.

There is a tendency throughout the Rand mines to adopt mechanical means for handling and transport of the broken rock and it is conceivable that this fact may have become an important factor in the prevention of miner's phthisis by lessening exposure to dust.

Furthermore, a miner who is found to have simple silicosis has the option of taking an award and leaving underground work or of remaining at work and postponing his award. A miner who has been granted a lump sum award and ceases underground work within three months of his notification is entitled to further award of a monthly allowance if his disease progresses

to the advanced stage of silicosis or to tuberculosis with silicosis.

At the present time there is a strong move on the Witwatersrand to replace the "wet" method of dust allaying by the "dry" methods, because it is being felt that the high humidity produced by much use of water combined with the high temperature of the deep mines produces an inefficiency, discomfort and even heat stroke, and may even be a factor in causing simple silicosis to pass into the "infective" type. Various experiments have shown that certain exhaust types of dust removal and the use of dust traps as the Kelly dust trap, remove the dust at the source more efficiently than do the "wet" methods of dust control.

The regulations for dust control on the Rand are rigidly enforced, and as a result the number of cases of silicosis and tuberculosis detected among European miners has steadily fallen the past 10 years and does not now constitute a serious menace. The Kaffir natives employed in the mines are more susceptible to silicosis and tuberculosis than the European miners, but even including them there has been a marked reduction in the number of new cases of silicosis from 1920 to 1930. In 1918-1920 the attack-rate for simple silicosis at the end of 10 years service was 8.94 per cent, and at the end of 20 years, 14.13 per cent. By 1928-1929 the attack-rates had decreased to 2.55 percent at the end of 10 years service and to 6.68 per cent at the end of 20 years service, showing a marked reduction in the number of new cases of silicosis.

According to Russell and his co-workers after their studies

at Barre, observations in certain granite plants in other cities have shown that local exhaust devices can be economically installed which will reduce the dust count for the dust-making occupations to less than 20 million particles per cubic foot of air and the general plant atmosphere to approximately 5 million particles. In some granite plants Russell found the ventilating equipment to be of little value because of lack of upkeep and maintenance, clogging or improper placing of exhausts. Proper maintenance of ventilating apparatus, and of all dust-allaying devices, is essential and requires the cooperation of foremen and employees.

It has been recommended that stone-mason's work should be done in the open so that the dust may be rapidly diluted and blown away; closed-in sheds should be prohibited. Sandstone and millstone wheels should be discarded in favor of composition wheels which give off far less dust and need not contain silica.

The suggestion has been made that some dusts in conjunction with silica dust act as "antidotes" for the silica dust and render the latter harmless. Mavrogordato maintains that coal and shale dusts produce a catarrhal reaction, and that this reaction is helpful in eliminating inert silica dust along with coal and shale dust. However, in the opinion of certain members of the Johannesburg Silicosis Conference, these suggestions should be treated with great caution and reserve and call for further research in this direction. At the same Conference there was a concensus of opinion that alternate employment and periods free from exposure to siliceous dust tend to

increase the resistance and thereby delay the development of silicosis (Nicholson 1923).

The Conference went on record as recommending:

1. That there should be some standard method for determining dust concentrations which would permit of inter-industrial and inter-national comparisons.
2. Further research into photographic and photo-electric cell methods of dust determination.
3. Further research into the size-frequency or fineness of dust particles, as it is being believed more and more that the surface exposure of the dust particles is the chief factor in the amount of silica which goes into solution.
4. That personal protection of workers during working hours should be supplemented by secondary measures, as the provision of suitable change houses, regulation of working hours etc. In this connection, others have recommended that employers, physicians, city authorities and others exert their influence toward raising the standards of living of the employees. It is felt that the expense of making a working place safe may be of little or no value if the predisposing causes of tuberculosis in the workers' homes and community are not controlled also, such as living in dark, damp, stuffy ill ventilated rooms etc. This would necessitate in many cases increased wages, but it has been felt the improved health of the employee will in the long run compensate.

Barnard (1934) believes that the use of the X-ray is the most effective method now available for the diagnosis of tuberculosis in adults, but that its costliness has previously served

as a barrier to its extensive use. She believes that X-ray of individuals by entire industrial groups is a logical source of attack. She cites the X-ray survey made in New York City in 1933 under the direction of Dr. Shirley W. Wynne of approximately 20,000 people by which the individuals were X-rayed at the rate of 150 an hour at a cost of \$.75 per person. The use of this method by the Metropolitan Life Insurance Company in detecting tuberculosis among its employees has resulted in a decrease in the morbidity rate from 132 per ten thousand employees in 1928, to 43 per ten thousand employees in 1932.

Harrington (1933) states that exact figures showing the morbidity and mortality from dust diseases annually in the United States are not available unfortunately, "partly because many doctors apparently do not know how to diagnose the disease, but chiefly for the reason that usually in the regions most affected a concerted effort is made toward minimizing the dangerous conditions. In fact 'pussy-footing' about dust disease is now and has for many years been largely the rule." He further states that frequently doctors, merchants, engineers and business men in general in the community, while knowing the gravity of the situation, remain quiet from motives of self-interest. Death certificates of victims of dust diseases are made out as heart disease or Bright's disease etc, and newspaper reports indicate that death was due to "lingering illness". Further, he states, that we have very few and incomplete regulations concerning the dust hazard and most of these are ignored. State laws as to dust elimination

in the mines to prevent disease are usually non-existent, where such laws are in existence they are extremely meager, and in general are not enforced. He cites the situation on the Rand in South Africa, where the dust problem has not been "side-stepped". He recommends the same general methods of dust control and that they be rigidly enforced. He recommends that industrial organizations, the State and Federal Governments and other interested parties should cooperate so that authentic information about dust diseases and dust control shall be available for industrial application, and also to serve as a solid background for State regulations and laws which are now being formed without dependable data, and also in connection with the numerous court actions now pending and likely to increase.

Quoting from Current Comment of the Journal of the American Medical Association (1933);

"In more than one industry today the most important slogan in the 'new deal', from the standpoint of welfare defined in terms of human health and comfort, would be 'Stop the Dust'."

SUMMARY AND CONCLUSION

It has been observed for many years and in numerous countries that employees exposed to silic dust over a long period of time, are especially susceptible to tuberculosis, and that the mortality from tuberculosis among these workers is considerably higher than in the general industrial population. Some of the industries most concerned with the silica dust hazard are metal mining, especially the gold, tin, zinc and copper mining industries, granite quarrying and cutting, sandstone cutting and grinding, and workers in burrstone, ganister and gritstone.

Numerous theories have been advanced in attempting to explain the injurious properties of silica dust. Probably the most prevalent idea at the present time is that the harm is due to the chemical nature of silica dust, caused by the fine dust particles going into solution.

The symptoms that the silicotic worker is most apt to be troubled with are a progressive dyspnea, unproductive cough, and transient pleuritic pains in the chest. The diagnosis of silicosis is by history of exposure to silica dust over a period of time, the symptoms mentioned, physical findings and X-ray study of the chest. The X-ray discloses a characteristic fibrosis, seen by thickened hilum shadows and linear markings extending from the hilum to the periphery of the lung. The fibrosis displaces the normal aerating lung tissue which causes the dyspnea, impaired resonance, diminished fremitus, decreased breath sounds, and limited chest expansion. In the silicosis complicated by tuberculosis, there is a marked thick-

ening of the shadows, even dense consolidations, and cavitations, which, in the case of granite workers are frequently basal instead of apical as ordinarily found in tuberculosis. Why silicosis should predispose to tuberculosis is not known.

It is a common experience that the prognosis in silicosis is poor. Once the condition has become established, almost invariably it seems, the silicosis progresses to tuberculosis and death, even though the worker leaves the dusty industry and takes to open air occupation.

It is quite generally agreed that there is no treatment for the cure of silicosis, and that prevention is the only method of control. Prevention of silicosis and the subsequent tuberculosis in industry necessitates the elimination of dust from the working atmospheres. Most countries, and particularly the United States, have been slow in adopting measures to control the dust. The gold mining industry of South Africa has been the pioneer in this respect, and by rigid enforcement of methods and regulations, dust in their mines is conspicuous by its absence.

Of the "engineering" methods of dust elimination, the chief means are ventilating schemes and devices, exhaust apparatus, and the proper use of water in drilling, cutting and blasting rock. Of the "medical" methods of dust control are thorough initial examinations with the rejection of those with respiratory disease or apparent susceptibility to silicosis and tuberculosis, followed by subsequent periodic physical examinations to detect incipient silicosis and tuberculosis, and thereby remove "foci of infection" among the working groups.

Considerable research work is indicated to determine the harmful properties of silica dust, the relationship between silicosis and tuberculosis and to determine more effective means of dust prevention and control.

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