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INDUSTRIAL HEALTH

RECENT DEVELOPMENTS IN PNEUMOCONIOSIS

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NEW YORK

Thirty years ago one thought of pneumoconiosis in terms of silicosis and the two words are often carelessly used as though their meaning were identical. From 1914 onward, silicosis received an increasing amount of attention and it was the general feeling among investigators in this field that silicosis was the only form of pneumoconiosis which was of prime importance. It was a clinical entity in itself, could cause disability and death and was frequently associated with tuberculosis which acted to expedite a fatal outcome.

In 1927, the term "asbestosis" first appeared in connection with a claim for workmen's compensation in Massachusetts, but this occupational disorder was not investigated until seven years later.[2] Here was another definite type of pneumoconiosis with a pathology distinct from that of silicosis and which also could produce disability and death.

During these thirty years a great deal of laboratory research and field study has been carried on in this country seeking to clarify the etiology and pathology of these two diseases. Abroad there is the outstanding work of the South African authorities and that of the English, the Canadians and the Australians, as well as similar investigations in France, Germany and other countries.

Field studies have crystallized ideas on etiology and the work of Gardner and his colleagues in Saranac have given a great deal of knowledge concerning the pathology of these two diseases. The roentgenologists, pioneered by the late eminent authorities, Pancoast and Sampson, have made their contribution to diagnosis. Also, engineering studies and experimentation have developed methods of controlling the dust exposures which are their underlying cause. Some of the earlier concepts have had to be changed but it is still evident that silicosis and asbestosis, to which must be added silico-tuberculosis, are the important form of pneumoconiosis from both the industrial and public health standpoints.

It is not entirely certain how silica produces its effects in the lungs, but the evidence is that the reaction is a chemical one. Likewise, the evidence indicates that asbestosis is produced mechanically.

The tendency of the silicotic person to become tuberculous has been recognized in every country in which this disease has been prevalent. Silicosis definitely predisposes to tubercle infection, the mortality from this cause ranging from ten to twenty times that of nonexposed persons. The nature of the relationship between these two conditions is not clear, but it has been generally thought that tuberculosis was a terminal condition. Of late, Dr. Gardner believes that tuberculo-silicosis in its chronic phases is neither tuberculosis nor silicosis, nor a mere superimposition of one condition upon the other, but that the two irritants act together to produce something that is entirely new and different.[3] This observation may have an important bearing on the attempts that are being made to clarify the pathology of other industrial dust-borne diseases mentioned later.

There have been no special recent developments with respect to asbestosis. The number of workers exposed was and is but a fraction of that exposed to silica. Mostly associated with textile processes, asbestos exposure could be and was controlled. It is a serious disease and must always be watched for where asbestos dust is present. With respect to tuberculosis and asbestosis, the two diseases may coexist, but there does not appear to be the causal relationship that exists between silicosis and tubercle infection.

Early ideas on silicosis were based upon experi-

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ence obtained in the hard rock mining industry in which the causative agent is quartz, SiO_2, and silicosis was thought to be a disease distinctive of the mining industry. Eventually, it became evident that many industries contained processes which involved exposure to silica dust and that frequently there was also exposure to other dusts associated with the silica. Different clinical patterns were defined. These substances present as dust along with the silica modify its action, the general tendency being to retard the action of the silica. It was thought some substances, notably free alkalies, might intensify the action of silica but it is doubtful if there is known at present any occupation in which the action of silica is enhanced by the presence of other substances. Fortunately, the general action of these mixed dusts is in the other direction. Such retardation has been described in the granite industry; iron oxide and coal dust likewise change the silicotic reaction. Furthermore, the presence of substances like gypsum and iron may cause aggregations with silica particles in the atmosphere so that they tend to fall out of suspension.[4]

When crystalline silica is heated in certain industrial processes, two other forms of silica are produced—crystobalite and trydimite. These conversion forms of free silica are very active and toxic and will produce silicosis more rapidly than ordinary quartz. This may account for the clinical picture seen occasionally in some industrial exposures.

There has been much conjecture and dispute about the effects upon the lung of silicates—dusts containing silica in a combined form. I believe it was Badham of Melbourne who first coined the term "silicatosis." Marked exposure to a silicate dust will, in some instances, produce roentgen ray evidence of change—exaggerated linear markings. These may tend to clear up after removal from exposure. They are not associated with symptoms or disability, although a study of the tremolite talc industry in New York State showed in some of the individuals exposed a roentgen ray appearance resembling early asbestosis both in fineness, diffuseness and ground glass appearance. Two fibrous silicates were present, tremolite and anthrophylite, which the investigators concluded were capable of producing a disabling pneumoconiosis. These cases also showed lung plaques varying from small linear deposits to massive deposits of variable outline and density situated on the visceral pleura, including the regions of the diaphragm and pericardium.[5]

Dreessen, investigating the talc industry in North Carolina, found roentgen ray evidence of dust reaction but no disability. From the medical and public health point of view, exposure to silicate dusts does not imply the hazards associated with silicosis. However, in any given case in which a roentgen ray change can be demonstrated following exposure to any type of dust, a medico-legal hazard may exist.

From time to time, attention is called to some industrial group which presents roentgen ray films showing nodulation and a general appearance resembling silicosis in which the evidence rules out silica exposure. These occurrences are troublesome and confusing both to the industry and the physicians, as well as to the individuals concerned.

Siderosis is a term that has been much used recently. Electric arc welders, in a number of instances, show on roentgen ray films an appearance that is so like silicosis that differentiation is difficult, if not impossible, by this means alone. Apparently the inhalation of fumes from the welding process over a period of from six to ten years may cause inert iron pigment deposits in the lymphatics without fibrous tissue proliferation. There is no progressive change in these cases after exposure has ceased, nor does this condition predispose to tuberculosis or other infection. There are no symptoms and this pseudosilicotic condition has little, if any, clinical significance.[6] It may have a considerable medico-legal significance. Similarly, a group of lime burners in one of our industrial states showed a generalized nodulation. Iron, carbon and aluminum were identified as present in the fumes to which they had been exposed. There was no clinical significance in these cases.

Both of these groups properly could be classified as having pneumoconiosis. This type, as well as that produced by the silicate dusts, has been called benign pneumoconiosis which is a satisfactory term, at least in the present state of knowledge, based upon both clinical and pathologic studies. Doubtless other dusts associated with other working conditions will be found from time to time, which may cause similar manifestations. All the factors involving working conditions, length and intensity of exposure, the composition of the dust, and the previous working history have to be considered before arriving at a diagnosis. This is particularly true when one encounters an individual patient, either in a hospital or away from his usual working environment. When a group of men are being studied in connection with their work and all the factors are known with reference to both working conditions and their physical condition, an investigator is able to weigh all the evidence and to render an opinion with a high degree of accuracy. But with an individual patient who presents a roentgen ray film that looks like silicosis or pneumoconiosis and who may have other organic disease and where the nature and extent of dust exposure is unknown, caution is most advisable and it is essential to backtrack on the work history as far as possible.

At this stage of the discussion of pneumoconiosis, it might be well to consider just what is pneumoconiosis.

In 1942, Dr. Gardner of the Saranac Laboratory stated that the pneumoconioses include all chronic changes in the lungs induced by prolonged inhalation of dust with no implication as to the type or severity of tissue reaction. The latter may vary

from simple phagocytosis to progressive fibrosis. Excluded from this category are the responses to living bacteria and fungi which occasionally are inhaled as dust.[7]

Definitions are frequently unsatisfying as anyone will appreciate who has tried to compose one. The foregoing definition is a good one although it is possible more recent experience may lead to questioning the words "chronic" and "prolonged." It has been the general experience, that the development of pneumoconiosis was a matter of years rather than of months. Also, it is not always easy to determine when a condition becomes chronic.

I have not mentioned fungous infections and will do so only in passing. Coccidioidal disease has become quite troublesome in some localities and is caused by inhaling dust in certain areas in which this fungus inhabits the soil. It would not commonly be thought of as pneumoconiosis although the method of causation is similar.

There is an occupational disease peculiar to the cotton textile industry known as byssinosis, presumably due to the inhalation of cotton dust, and which may or may not be a pneumoconiosis. The fibrosis is not marked, cotton dust has not been detected in the lungs of affected workers and the disease is not progressive after removal from exposure and it appears to have no tendency to cause tuberculosis. Byssinosis is more prevalent and has received more attention in Great Britain than in this country. In England, it is compensable and is credited with the ability to cause total disability and presumably death. It develops slowly over a period of from ten to twenty years and the roentgen ray appearance is not characteristic but corresponds to chronic bronchitis.[8]

It remains to be seen whether or not byssinosis has any appreciable significance in this country. It should not be confused with the infectious illness found among workers in low grade stained cotton, an acute infection due to a gram-negative organism, Aerobacter Cloacae, which has been the subject of two or three reports by the United States Public Health Service.[9]

Another disease which has been reported recently in this country is bagassosis. Bagasse is the produce remaining after the extraction of sugar from sugar cane. This is baled and workmen who later open these bales may present the symptoms of bagassosis, namely, cough, sudden severe dyspnea, occasional hemoptysis, night sweats, chills and fever. Prolonged weakness is characteristic and there is usually leukocytosis of the polymorphonuclear type. The sputum is scanty and mucoid. The roentgen ray film shows a diffuse mottling throughout both lungs. Recovery takes place over a period of a few months and the roentgen ray picture clears.

The short time of onset and the duration do not correspond with a pneumoconiosis; it is possibly an infection or of an allergic nature.

There are two other diseases recently reported which apparently are due to the inhalation of dust or fumes. They are not only very serious but quite distinct from any industrial disease so far recognized. Fortunately, the cases are few and the conditions which produced one of them at least appear to be no longer in operation.

Some employees of certain industries concerned in the manufacture of fluorescent lamps were found to have a pulmonary disease which had the appearance of sarcoid. There have been about twenty or twenty-five cases of this disease with, I believe, about five deaths. Intensive study, both in the plants and the laboratory, have failed to bring out any clarifying information with respect to exposure, the causative agent or the true significance of this condition. It would seem that these resulted from exposure during 1940 and 1941, that they began to manifest themselves in 1943 and that the manufacturing procedures have been changed and that there is no evidence of further trouble.

Various hypotheses have been advanced by the physicians and research workers who have been studying these cases. The disease may be an atypical silicosis due to the inhalation of exceedingly fine particles. Also, it may be due to the inhalation of fluorescent powders activated by some form of radiation; and it may be due to the toxic action of some substance present in the process. Suspicion has fallen upon beryllium. The factor of infection may be present; possibly a combination of two or more of these suggested causes may be responsible.[10]

Beryllium has not previously been considered toxic[11] but the Saranac Laboratory states that beryllium is a potent irritant and capable of producing chronic disease. More information will be forthcoming as the present studies develop.

Another disease has been detected in a group of workers who process bauxite at a high temperature. This process, by no means new, has never previously involved any health hazard as far as is known. In some plants in Canada, several cases were detected which presented the following symptoms: cough, dyspnea, cyanosis, to the point of disability and a distinctive roentgen ray appearance characterized by bilateral spontaneous pneumothorax. There have been from twelve to fifteen cases with four deaths so far. The postmortem examinations showed massive extreme fibrosis and emphysema. There was no evidence of tuberculosis.

I am indebted for my information about these cases to Dr. Andrew Riddell of the Health Department of Ontario who has kindly enabled me to obtain a film and lantern slides of one of these cases. Here, again, it remains to be seen whether this condition can be classed as a pneumoconiosis. The fatal cases lived about a year or a little longer after disability occurred.

There are certain definite implications with respect to the subject of pneumoconiosis and other air-borne occupational diseases. While silicosis and asbestosis are the two most important industrial

dust diseases, knowledge regarding them is still far from complete or thorough. It is being realized more and more that all industrial dusts are potential sources of trouble and that new types of exposures and new types of reactions are being discovered. Combinations involving active chemical substances, rare metals and radiant energy imply harmful possibilities, and even a dust commonly considered harmless may under certain circumstances or in combination with other substances, also in themselves considered innocuous, become actually or potentially dangerous. Any dust which can produce roentgen ray changes, even though classed as benign pneumoconiosis, must be considered seriously. With any dust, not only does there arise the question of its specific reaction but of its relationship to tubercle infection. And here, one moves from the medical and scientific field to that of medico-legal action.

Prevention of these dust diseases is entirely possible. Engineering skill and engineering methods can be applied to control or remove the dust at its source. This is often expensive, but so is the provision of safe drinking water. People have been educated in this country to spend enormous sums of money to provide safe drinking water but they have not learned yet to be as intelligent about the pollution of air as about the pollution of water.

Hand in hand with dust control in industrial establishments goes medical supervision. One of the lessons learned in war plants is that competent medical supervision and competent engineering control guarantee sound preventive medicine. Both are essential and satisfactory results cannot be obtained with one alone.

It is essential that industrial physicians, roentgenologists and other physicians who may come into contact with industrial workers be alert to detect and follow up unusual cases. The two diseases mentioned before were detected early in plants having a good medical service. One thinks of the possibility of individual cases in which the significance of the findings and the relation to occupation are apt to be overlooked.

One of the phases of the pneumoconiosis situation which has attracted a great deal of attention in the last few years is the use of aluminum dust both for the prevention and treatment of silicosis.

It is almost ten years since Denny, Robson and Irwin of the Banting Institute in Toronto demonstrated that by administering finely divided aluminum dust to experimental animals they could inhibit the action of silica. This was confirmed by other investigators, notably by Dr. Gardner of the Saranac Laboratory. The Canadian group used metalic aluminum and Dr. Gardner also used amorphous hydrated alumina. Several of us who are interested visited the Silicosis Clinic in Porcupine and were greatly impressed by what we saw. There is no question but that a number of the silicotic employees who had undergone this treatment experienced considerable relief of their symptoms, notably cough and dyspnea. It should be noted, however, that in other instances no improvement was noted and, in other localities, it has been difficult at times to induce the men who were receiving this treatment to continue with it.

It is natural that the novelty of this type of administration of a therapeutic agent would cause some hesitancy with respect to its adoption. There also has been a natural reluctance to try this mode of therapy on silicotic patients who are also tuberculous.

At the present time, this whole matter has been the subject of discussion by the Council on Industrial Health and the Council on Pharmacy and Chemistry of the American Medical Association and it is the intention of these two councils to issue a joint statement relative to the status of this subject at an early date. While there is naturally some divergence of opinion on various points among those concerned with the demonstration of this method, I believe that there is a general agreement with respect to basic considerations.

Silicosis is essentially a chronic disease and takes years to develop and, once the disease is well advanced, the general outlook is not very good, particularly with respect to the possibility of tubercle infection. Consequently, it is going to take a considerable length of time to demonstrate whether in human beings this method of treatment by the inhalation of aluminum dust or of amorphus hydrated alumina will fulfill the expectations raised by the results in experimental animals. It is apparent that where this form of therapy or prophylaxis is being tried, it is necessary to maintain careful and accurate medical supervision if permanent and worthwhile results are to be demonstrated. All who are active in endeavoring to develop the use of aluminum are agreed that it is in no way a substitute for engineering control of the dust hazard and for medical supervision of workmen exposed. It is appreciated that in some instances it is difficult to control entirely the escape of silica dust into the atmosphere but, in any event, it is insisted upon that engineering control and medical supervision be adequate before aluminum treatment is initiated.

As far as is known now, metallic aluminum is nontoxic. In response to an inquiry in *The Journal of the American Medical Association*, January 20, 1945, the statement is made that American medical literature contains no convincing evidence of pulmonary or bronchial injury from exposure to aluminum dust. There is considerable literature on the action of metallic aluminum on the lungs and some of the German investigators have claimed that aluminum is capable of producing a pneumoconiosis. This has been denied by other observers. There have been some reports that amorphous hydrated alumina may have an unfavorable effect upon tubercle infection. It is obvious that careful supervision of this inhalation method of treatment is obligatory. There also arises the question of possible medico-legal complications with respect to

aluminum therapy and, again, only time will give us the final answer.

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TUBERCULOSIS IN INDUSTRY

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Control of tuberculosis in industry in the past was often hampered and made ineffective by costly roentgen ray procedure. The standard 14 by 17 inch film, universally regarded as the most accurate plate size for the detection of lesions in early tuberculosis, was too expensive in material and personnel for large scale use, and was not practicable for the further important reason that any considerable number of workers could not be handled in desirably short periods of time.

Now, however, tuberculosis in industry can be controlled effectively by the application of a new and relatively inexpensive mass radiography technic. Photofluorographic methods employing 35 mm. and 70 mm. roll film, 4 by 5 inch film, and 14 by 17 inch roll paper film have given industry tools for the control of a disease that strikes down the most valuable industrial workers—men and women between the ages of 20 and 45.

In 1943, nearly one half of the deaths from tuberculosis (44.8 per cent) was in this age group of maximum personal effectiveness. Indeed, from early adulthood to the age of 35, tuberculosis is the chief cause of death. Between the ages of 15 to 49, it is one of the first three causes of death; and between the ages of 20 to 34, one out of every six deaths among white persons and one out of every three deaths among nonwhite persons are due to tuberculosis.

Recent surveys show that from twelve to fifteen in every 1,000 industrial workers are attacked by tuberculosis, and in consequence they may become inefficient on their jobs, are often absent and spread the disease to others. The cost to industry is incalculable and the expense in human life beyond all measure.

During the war there was no general increase in tuberculosis. Nevertheless there has been a larger proportion of deaths from tuberculosis in urban industrial areas than in nonindustrial areas.

Industrial communities are spawning grounds for this dread disease. Overcrowding, insanitary living conditions and unusual fatigue are contributing factors that precipitate the susceptible into breakdowns and hinder the already diseased from achieving maximum recovery.

Tuberculosis control efforts should be and have been concentrated on these areas in anticipation of an expected rise in tuberculosis mortality as a consequence of the war and its contingent health strains and pressures.

Soon after war was declared, the United States Public Health Service established a tuberculosis control office for the purpose of assisting state and local health departments in the development of tuberculosis control programs. Then, in July 1944, the Tuberculosis Control Division was created. The objectives of this Division are as follows: (1) case finding, (2) isolation and treatment, (3) after-care and rehabilitation, (4) protection of the family of the tuberculous person against economic distress.

The purpose of case finding is to discover hidden cases of tuberculosis. Such efforts, in the past, were directed toward the family members of known infectious cases. Since the introduction of mass radiography, however, case finding has had a much larger range. It has been pointed to large population groups. The two sizable portions of the population which can be reached easily by mass radiography are (1) persons admitted to general hospitals, and (2) persons employed in the large and small industries of the nation. This second group is at the present moment one of the chief concerns of the Tuberculosis Control Division.

When cases of tuberculosis are found, they must be given medical care and be isolated to assist the patient in renewing health and hope, and to prevent spread of the disease. To be sure, there is a lack of vision in case finding if treatment is delayed by a shortage of sanatorium beds. In every industrial community in which a tuberculosis control campaign is undertaken, plans must be made for the provision of a sufficient number of beds. Temporary facilities may, for a time, be utilized until the people, realizing the seriousness of the community's plight, will demand the construction and maintenance of the necessary hospitals.

After-care and rehabilitation are extremely important aspects of the whole fight against tuberculosis. It is well known that tuberculosis is a relapsing disease. The industrial worker who has arrived at an arrestment of his case must have help and competent advice, both medical and social, in his readjustment to self-supporting life. This is a large problem which will require the cooperation of private industry and local, state and federal agencies.

When the industrial worker is stricken with tuberculosis, his family often is required to depend on public funds for the necessaries of life. Accordingly, a sound medical program must be complemented by a generous plan of public assistance. If

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