a proper staff, and not by some enthusiastic person who has not been trained for the work. Why not use more registered nurses in the smaller plants? The registered nurse is much better qualified to interpret a physician's plan of medical service than any lay attendant.

Much depends upon the physical condition of the employees. I am of the opinion that the physician, by reason of his professional attainments, is best qualified to handle some of the problem cases among employees. Mental symptoms, brought about by worry over home affairs, quite often produce accidents. In many cases the fear and worry of sickness in the family can be greatly allayed by a talk with the doctor or the nurse. Many of these cases have come under my personal observation.

It should also be absolutely the part of the physician to say when a man shall return to his regular employment after injury, and this decision should not be influenced in the slightest by its possible affect on any "no-lost time accident" contest or record. These contests are proper and have brought about outstanding results in the reduction of lost-time frequency figures. However, there have been cases of injured employees being urged unduly to return to some form of work merely to prevent the marking up of a lost-time accident. Nothing should be allowed to interfere with the employee's welfare. The physician has every motive to return the employee to his regular work as promptly as possible, but his higher duty is to conserve both the physical and economic welfare of the employee as well as the employer.

The safety director has need of the medical service, and the two should earnestly co-operate and seek to bring about a mutual feeling among the employees. Both departments are striving by all possible means to prevent economic waste chargeable to the disabilities and hazards of industry.

TUESDAY AFTERNOON SESSION

October 4, 1932

The delegates first attended an informal luncheon, and then gathered for the afternoon meeting in a Joint Session with the Metals Section. This session was devoted to a symposium on the dust problem in industry. Chairman Greenburg immediately introduced the first speaker.

The Effects of Inhaled Mineral Dusts

By LEROY U. GARDNER

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As long as men have worked in stone it has been appreciated that an unusually large proportion of them suffer from disease of the lungs. It was natural to assume that such disease would be caused by the dust generated, and this belief was strengthened by the discovery of black, grey or red pigments in the lungs, sometimes accompanied by the formation of scar tissue.

The term, pneumonokoniosis was introduced to describe the lung pigmented in this manner. With further observation pathologists attempted to classify various forms of pneumonokoniosis on a basis of the type of dust inhaled and such names as anthracosis, siderosis, chacicosis and even byssinosis and tobaccosis made their appearance. It was recognized that some forms of the disease were attended by severe symptoms and result in death but there was no correlation between the clinical and pathological pictures and the causative dust. Finally, statistical study demonstrated that of all kinds of industrial dusts silica generally produced a definite type of disease with a characteristic pathological lesion and complex of symptoms.

Today the clinical entity known as silicosis is one form of pneumonokoniosis which is quite clearly defined. Moreover, within the last three or four years asbestos dust has also been shown to produce another specific type of reaction attended by symptoms differing in some respects from silicosis.

The other forms of pneumonokoniosis still lack complete definition. The widespread use of radiography has brought together a mass of descriptive data from individuals exposed to many kinds of occupational dusts but these findings have not yet been correlated with pathological anatomy, chemical studies of the tissues and detailed analyses of industrial atmospheres. There is a tendency to assume that many of these pulmonary changes are due to the action of silica perhaps modified by the chemical components of the dust. We shall only be in a position to speak with assurance about them when the pure types of dust and their various combinations have been studied in human beings or in experimental animals.

For the sake of clarity no mention has been made of the infections which so frequently complicate some forms of pneumonokoniosis. In the prebacteriological era of medicine the use of such terms as "grinders' consumption" and "miners' phthisis" reflects the popular association of pneumonokoniosis with tuberculosis. The names were justified both by clinical and pathological observation for it has been subsequently shown that, in silicosis at least, a super-imposed tuberculosis may cause death in perhaps 75 per cent of the cases. Some observers even go so far as to state that silicosis itself does not develop unless the lungs are previously damaged by a latent tuberculous infection. This is probably an exaggeration, for typical silicosis can be produced in the experimental animal by the inhalation of silica without infection.

The role of asbestos dust as an excitant of tuberculous infection is not as clear cut as that of pure silica. While there are numerous reports of death in asbestosis due to a terminal tuberculosis, nevertheless surveys of living workers have failed to demonstrate any excess of such infection. Simple anthracosis is said to prevent the progression of tuberculous infection. Statistics from coal mining districts do show a tuberculosis rate much lower than that "normal" for the age group. As yet corroborated experimental proof of protective action of coal against tuberculous infection is lacking.

Not only the tubercle bacillus but other bacteria seem to develop with special facility in the tissues previously damaged by silica. For example, the pneumonia rate among silicotic native laborers of South Africa is many times that in non-silicotic natives living under similar conditions. In coal miners, likewise, pneumonia is a common and often fatal complication.

What has been said of the effects of inhaled inorganic dusts would indicate that the reaction of the tissues is not merely a response to mechanical irritation by particulate matter. Today it is generally accepted that the injury is chemical in nature and that only certain of the common types of industrial dusts possess properties capable of exciting reaction. In the cases of silica and the silicate of magnesium or asbestos the slightly alkaline body fluids probably effect a slow solution of the dust particles liberating silica in colloidal form. This substance irritates the connective tissue cells which respond by multiplying. The result is an overgrowth of the supporting framework elements at the expense of the more delicate cells specialized for specific functions.

For these two types of dust the character and form of the pathological changes has been carefully worked out. In *silicosis* the essential tissue change consists of nodules of connective tissue which develop first in the lympoid tissues situated in the drainage apparatus of the lungs. These nodules interfere with the physiological removal of foreign bodies and subsequently inhaled particles accumulate in the framework of the lung itself. Such reaction gradually decreases the normal elasticity of the organ and encroaches upon its functional elements. As a result the cardinal symptom of the disease, dyspnoea or shortness of breath develops. Because of the excessive amounts of scar tissue formed in the lungs the right side of the heart