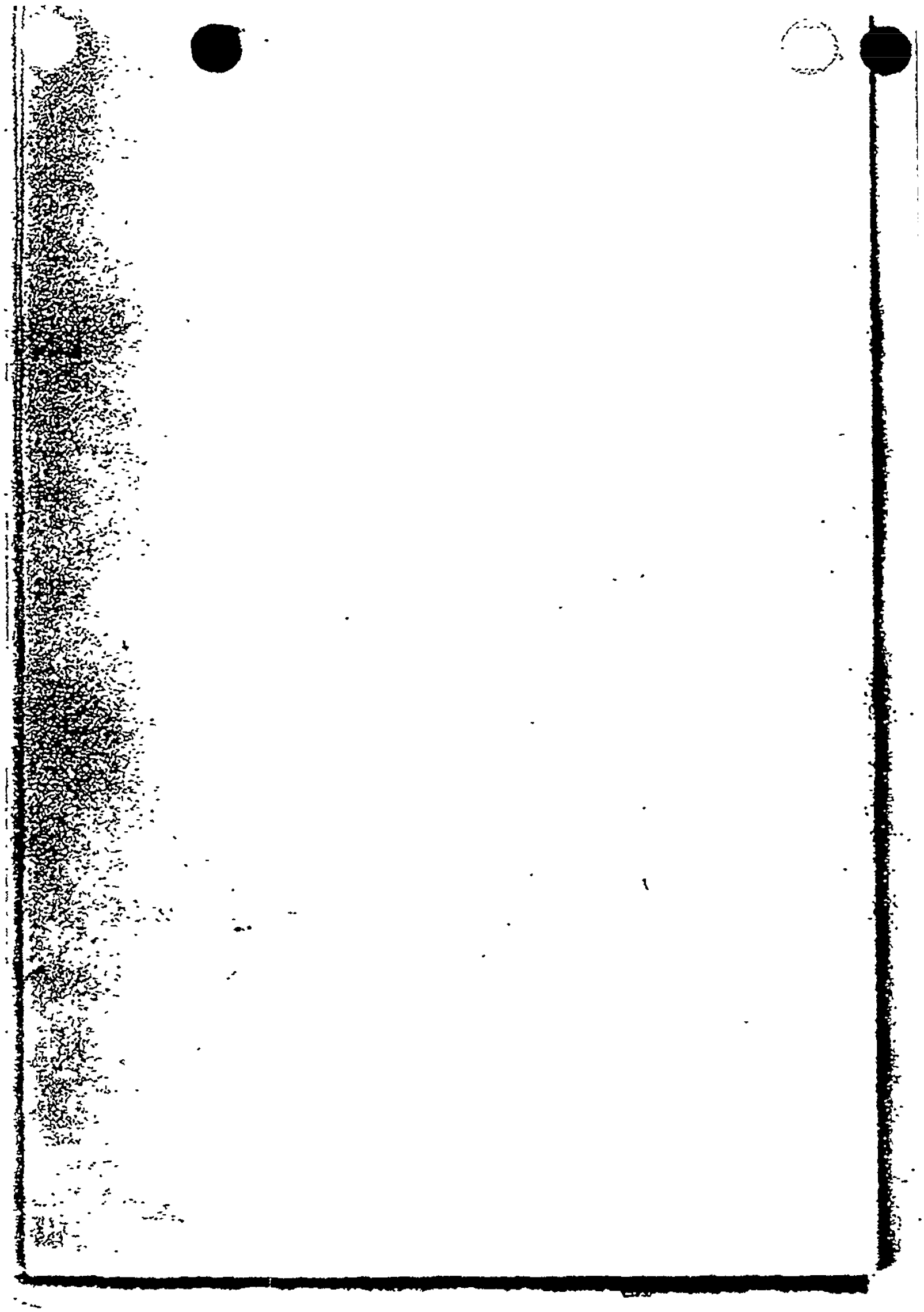
FILE NAME: Koppers/Beazer East (KBE)

DATE: 1932

DOC#: KBE024

DOCUMENT DESCRIPTION: Transactions of the 21st National Safety Council



1932

TRANSACTIONS

National Safety Council

Incorporated

TWENTY-FIRST ANNUAL
SAFETY CONGRESS

Washington, D. C.

October 3 to October 7, 1932

The Wardman Park and
Shoreham Hotels

CONTENTS

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Although the National Safety Council endeavors to eliminate from discussion at its conventions matters which are not pertinent to its purposes or which are contrary to its policies, the Council accepts no responsibility for the views expressed either in the papers presented or in the discussions thereon.

* *

NOTE: The sessions of the Street and Highway Traffic Section, the Child Education Section, and the Home Safety session are published in a separate small volume of these Transactions, available on request.

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Heavy Machine Shop Division, Group A. Lycoming Mfg. Co., Plant No. 1, Williamsport, Pennsylvania. The same division, Group B, United States Rubber Company. Shoe Hardware Company, Waterbury, Conn.

"Your Hour"

The next part of this session was a special feature entitled "Your Hour," led by T. H. McKenney, Supervisor of Labor & Safety, Illinois Steel Company, South Chicago. Question cards had been distributed and returned with safety problems, which Mr. McKenney read from the floor, and then asked for solutions. Questions and answers were as follows:

Question No. 1: "It was expected that poor working schedules would cause more accidents, but this has not been the case, why not?" One reply was that plants are not pressed for production. However, a strong percentage of the delegates were of the opinion that spreading the work and putting new men on new jobs was causing accidents. A vote of delegates on whether or not frequency of accidents has decreased during 1930 and 1931, resulted in a vote showing a decided decrease.

Question No. 2: "What practical means can be used to detect defects which may develop in crane wheels?" The replies included: visible inspection for cracks, flat surfaces, etc.; tapping wheels with hammer; also gauging the flanges. One well known company makes a monthly inspection of all parts of the crane.

Question No. 3: "Have any troubles or injuries been encountered due to metal caps on safety shoes?" Several well-known companies do not permit any safety shoes except those with steel toe. Flammable material in a composition toe is considered objectionable. Practically every man went on record as favoring metal tipped toes and a vote showed unanimous approval of steel toes.

Question No. 4: "What procedure should be followed if superintendents, foremen and workers do not co-operate in the accident prevention program?" The answers included the following: Our management is always with us but a good organization will also offset opposition to safety. Putting objectors at the heads of safety committees will often effect a cure. Safety must begin at the top. A safety engineer who cannot sell his wares to his men should move. The safety engineer must sell his wares to the management, and all executives are susceptible of being influenced if properly approached. Both the emotional approach and the discussion of safety on a cost basis are valuable to make the management listen.

Question No. 5: "In the investigation of accidents and near accidents, what is the best method of procedure—through the foremen, by a committee, by a safety inspection, or otherwise?" Many delegates favored an investigation by the department superintendent and his men, foremen, etc., with the safety engineer as secretary and assisting in the questioning. Another suggestion was by means of a trial. An immediate investigation is advisable, since conditions soon change, especially if someone is especially at fault.

Question No. 6: "Now that we are agreed that responsibility for accidents rests with the head of the department, what is the safety department doing to lighten the load of the foreman?" *It was agreed that the safety department should help the foremen in every possible way. It is important to combine safety and training.*

The final feature of the program was the presentation of the film strip entitled "Safety Kinks," by J. A. Oertel, Chief of Safety Bureau, Carnegie Steel Company, Pittsburgh. Mr. Oertel presented a great variety of machines and devices with appropriate remarks.

ADJOURNMENT

Mining Section

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D. D. Moffatt, Utah Copper Co., Salt Lake City, Utah.
Howard I. Young, American Zinc, Lead and Smelting Co., East St. Louis, Ill.

TUESDAY MORNING SESSION

October 4, 1932

The sessions of the Mining Section convened with General Chairman W. H. Comins, National Lead Company, St. Francois, Missouri, presiding. After welcoming the delegates, Chairman Comins asked for the report of the Statistics Committee which was presented by W. W. Adams, U. S. Bureau of Mines, Washington, D. C., Chairman, in part as follows:

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

Director, Saranac Laboratory for the Study of Tuberculosis, Saranac Lake, N. Y.

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, chalicosis 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

encounters increasing difficulty in forcing blood into the organs. In an attempt to compensate the overworked heart muscle becomes thicker and heavier and unless infection intervenes the time eventually arrives when the heart fails and death occurs from this cause.

The reasons for the prevalence of infection in the silicotic lung are not altogether clear. It is obvious that when the drainage system is so obstructed by nodules of scar tissue that more dust particles can only be removed with difficulty, the same must be true of bacteria which may be inhaled. As a consequence they remain in the lung and set up progressive infections. But this is not the whole story. The silicotic tissue apparently offers a peculiarly favorable soil for the continued multiplication of tubercle bacilli and perhaps other bacteria. Experiment has shown that even attenuated organisms of this type will produce rapidly fatal tuberculosis in a silicotic guinea pig. Furthermore, inhaled silica dust will cause a pre-existing latent tuberculous focus, harboring a few attenuated bacilli, to become progressive and spread.

The cause of this effect upon tuberculous infection has not yet been ascertained but probably it is in some way associated with the death of tissue caused by the toxic silica. Associated with this factor is an increased activity of the phagocytes which are stimulated by the irritating silica so that these cells tend to migrate rapidly and carry tubercle bacilli into previously uninvolved portions of the lung.

Asbestos dust, perhaps because of its fibrous structure, is not transported very far within the lung. It tends to lodge along the walls of the finer tubes and little of it is removed by the lymphatic drainage system. Tissue reaction, probably initiated by the solution of the fibers, occurs in their immediate vicinity. As in the case of silica this reaction consists of an overgrowth of the connective tissues which is later transformed into leather-like scars.

Aluminum oxide will serve admirably as an example of a non-siliceous dust whose particles are as hard and sharp as those of crystalline silica. When a measured quantity of such particles, 1 to 3 microns in diameter, are injected into the ear vein of a rabbit the majority of them come to rest in its liver and spleen. There they remain, apparently harmless, collected in large phagocytic cells and producing no reaction of the connective tissues for at least two years. Injection of the same quantity of the same sized quartz particles excites the formation of so much scar tissue that the functional elements are almost completely destroyed and the organs are reduced to nodular masses of leather-like consistence.

Carborundum, the carbide of silicon, when inhaled by guinea pigs for periods as long as four years, likewise fails to excite significant overgrowth of the connective tissues. It produces only a low grade inflammatory change with no nodules.

Instances such as those cited constitute the basis for believing that the cellular response to dust particles is not due to their hardness and sharpness but to chemical substances liberated by the action of the tissues upon them. The case for solubility has not been proven directly, for the detection of soluble silica in the tissues offers technical difficulties which today are unsurmountable. Nevertheless this substance is known to be a cell poison and in weak concentrations it will provoke an overgrowth of the connective tissues.

Mention should be made of the effect of inhaled coal dust. Most city dwellers breathe in sufficient quantities of this material during an average life-time to pigment considerable areas of their lungs. Deposits of grey or black dust will be found along the course of the lymphatic drainage system but this pigmentation is associated with little or no new growth of connective tissue. The coal miner's lung is much blacker but in most instances it also develops no deforming scars. When such reaction does occur analysis usually reveals appreciable amounts of silica in the lungs. The presence of excessive quantities of carbon dust apparently influences the localization of moderate amounts of silica so that the latter is not deposited in nodules as in pure silicosis but in streaks along lymph vessels as pure coal would localize.

Coal miners' fibrosis, due to relatively small amounts of silica, is therefore dif-

ferent from that of the pure quartz worker. When the amount of inhaled silica is excessive, the effect of the coal is negligible and the reaction approximates that in pure silicosis. There is said to be more reaction to hard coal than to the bituminous variety but there is evidence to suggest that this is due to the greater amount of siliceous rock which must be worked in mining anthracite coal.

Finally, consider the silicosis of granite cutters. This develops somewhat more slowly than that of the pure quartz miner and it has been maintained that this is due to the relatively small amount of uncombined silica in granite (25 to 40 per cent). But it is believed that other components of the glomerate granite may neutralize or at least inhibit the effects of the silica so that only after prolonged exposures to high concentrations does significant reaction occur. While typical silicotic nodules develop in guinea pigs inhaling pure quartz for a year, not even the earliest suggestion of nodules have appeared in the lungs of animals inhaling comparable concentrations of granite (40 per cent silica) for four years.

This brief discussion of some of the problems involved in pneumonokoniosis draws attention to the limitations of our present knowledge. It indicates that the reaction to an inhaled dust is determined by the chemical and probably physical composition of that dust. It emphasizes the need to further study of different types of dust both in the pure state and in measured combinations. The ultimate aim of such a study should be the neutralization of the toxic action of such dangerous substances as silica.

The Dust Content of the Atmosphere in Various Dusty Industries

By J. J. BLOOMFIELD

Sanitary Engineer, United States Public Health Service, Washington, D. C.

The speaker said in part: The properties of a given dust which determine its capacity to produce pulmonary pathology are, the nature of the dust, that is, its chemical and mineralogical composition, its particle size, and finally the quantity of the dust dispersed in the atmosphere.

One of the outstanding results of the last 20 years of research in the field of dust inhalation is the demonstration of the fact that, in general, the degree of health hazards associated with the inhalation of any dust, all other factors remaining constant, is dependent upon the mineralogical composition of the dust. For example, it is now well established that the inhalation of certain types of dust, such as granite dust, will in time produce fibrosis of the lungs, at times associated with tuberculosis. In other cases exposure to dust may result in the production of much less fibrosis without notable tendency toward subsequent tuberculosis: this is true of cement dust. And finally, there are certain types of dust, as typified by marble dust, which in the quantities and lengths of exposure so far observed produce little lung fibrosis. In general, it has been found that those dusts which are high in quartz content are the ones which produce a disabling fibrosis of the lungs most readily.

So far as the size of the dust particle is concerned, it is apparent that in order for any given dust to produce injury to the lungs, it must gain access to the parenchyma of the lung, the site where the harmful effects of the dust take place. It is known that not all of the particles of inhaled dust gain access or are retained by the human lung. In this connection it is of importance to have regard to the size of the dust particles present in the industrial atmosphere.

With reference to the quantity of dust present in the air of a workroom it is apparent that when the dust concentration is high the exposed person will inhale a greater quantity in a given period of time than he will when the dust concentration of the atmosphere is relatively low, and since the rate of production of the fibrosis