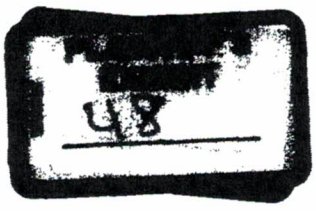

1934

TRANSACTIONS

National Safety Council

Incorporated

TWENTY-THIRD ANNUAL
SAFETY CONGRESS

Cleveland, Ohio

October 1 to October 5, 1934

The Cleveland, Carter and Statler Hotels

Publication Plan

THE TRANSACTIONS of the Twenty-third Annual Safety Congress, National Safety Council, 1934, are published in two volumes. The first, containing the General Sessions, the Special Subject Sessions, and the Industrial Section Sessions, is presented herewith. It is supplemented by a smaller volume containing the sessions of the Street and Highway Traffic Section, the Child Education Section, and Home Safety.

The large volume is being sent automatically to the Industrial members of the Council; the smaller volume is sent automatically to members who are believed to be chiefly interested in those sessions. It may also be secured on request by other Council members.

For the benefit of many members who have found it advantageous to distribute copies of these TRANSACTIONS to supervisors, foremen and others among their personnel, who may keep them for continuous reference, the following quantity prices are quoted: 1 to 10 copies of the large volume, $1.50 each; 11 copies and over, $1.25 each. Copies of the smaller volume, $.50 each.

THE PLAN OF PUBLICATION is the same as that followed for the last three years. It is a condensed record. The papers of each session or division of the Congress have been assembled, edited with care and discernment to eliminate extraneous matter and abbreviate the less essential portions, and the concise remainder is presented for study and reference. Although not "complete" in the sense that every word is reproduced, it is an interesting and valuable record which emphasizes the memorable parts of each paper or address. The original manuscripts are on file in the Council Library, available for additional reference as desired.

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 cannot accept responsibility for the views expressed either in the papers presented or the discussions thereon.

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TWENTY-THIRD ANNUAL SAFETY CONGRESS
NATIONAL SAFETY COUNCIL

Dust in Industry

FRIDAY AFTERNOON SESSION

October 5, 1934

The session devoted to a discussion of Dust in Industry was called to order by Dr. E. G. Meiter, Director, Industrial Hygiene Laboratory, Employers Mutual Liability Insurance Co., Milwaukee, who presided. Dr. Meiter briefly welcomed the delegates and then introduced the first speaker.

Types of Dust That Cause Occupational Diseases

By LEROY U. GARDNER, M.D.

Director, Saranac Laboratory for the Study of Tuberculosis, of the Edward L. Trudeau Foundation, Saranac Lake, N. Y.

The speaker said in part: Experience has demonstrated that the lung tissues can tolerate very large amounts of most kinds of foreign dust particles without serious interference with function. The lung may be intensely pigmented by colored dusts with no evidence that respiration is impaired. But dusts composed, in whole or in part, of silica have been shown to be dangerous because they stimulate the growth of the connective tissues of the lungs and bring about the formation of scar tissue. This destroys the normal elasticity of the organ which is necessary for respiration and it thickens the delicate membranes so that a free interchange of gasses is impeded. Fortunately, these results only follow the prolonged inhalation of very large quantities of silica.

Experiments have demonstrated that the injection of silica into the tissues of animals kills the cells where the substance lodges and excites an inflammation. When healing takes place a very dense and characteristic kind of scar tissue forms. Most non-siliceous dusts produce no inflammation and very little scar. It has been shown that such changes do not follow the injection of silica unless the particles are very fine, three microns or less in diameter, and unless enough of them localize in one area. With larger particles the rate of reaction is so slow that little change occurs within a year. As a general rule, the finer the particles, the more rapid the cellular response. The reaction to fine silica begins within four or five hours and it continues for a period of years.

If the silica is combined with bases in the form of silicates, it rarely produces such active and progressive changes. The silicate of magnesium, asbestos, does cause the formation of scar tissue in experimental animals and in the human lung, but most other silicates are not generally recognized as irritating. Recently it has been claimed that a hydrated silicate of aluminum and potassium, called sericite, is responsible for most of the cases of silicosis. This silicate, a transformed feldspar, is often associated with quartz and it has been identified in the lungs of persons suf-

fering from silicosis. However, at least two types of sericite have failed to excite significant changes when injected into animals. Experiments with this substance are still in progress. Kaolin, an alumino-silicate of potash, has also produced quite characteristic changes in the lungs of rabbits. But our present knowledge does not permit us to assume that silicates as a class will produce serious tissue damage.

Most observers now believe that uncombined silica, or quartz, is poisonous to the tissues because it is slowly dissolved in the fluids of the body. Certainly the majority of the experimental observations suggest a chemical injury, but it is difficult to understand how enough of such an insoluble substance as quartz could be dissolved within eight hours to produce a well marked, acute inflammation. If solubility were the only factor one should discover a more active response after the injection of the more readily soluble silicates, but such is not the case. Formerly it was held that silica produced its effect mechanically. The hard sharp quartz particles were believed to abrade the lung surfaces and excite the formation of scar tissue as a manifestation of healing. This theory is no longer tenable for when animals are injected with other hard, sharp particles like diamond, carborundum or aluminum oxide, there is no inflammation and practically no formation of scar tissue. Ultimately it may be shown that the irritation of silica is due to some unrecognized property possibly related to its atomic structure.

When quartz particles are inhaled they produce three specific effects which are not excited by any other type of dust yet studied. They alter the behavior of the pulmonary phagocytes; they stimulate the formation of circumscribed nodules of scar tissue; and they subsequently increase the susceptibility of the lung to infection, especially tuberculous infection.

Phagocytes that ingest too many quartz particles are killed by this irritating substance. As the cells die they liberate their burden and several new phagocytes come out to take it up again. By a repetition of this process enough phagocytes accumulate so that many of them ingest only a few particles. Under such circumstances the stimulus is not strong enough to kill but merely accelerates the motility of the cells. They naturally tend to leave the air spaces, enter the lymphatic vessels and accumulate in the lymph nodes. This process becomes more rapid under the influence of the irritating silica and very considerable numbers of particles are consequently localized in the nodes.

After six or eight years the quartz in the nodes has caused a marked overgrowth of the fibrous elements in these structures. Ultimately the reaction develops to such an extent that the node is entirely replaced by a mass of coarse, glassy scar tissue fibres, often several times as large as the original node. Such nodules encroach upon and compress the lymphatic vessels. The drainage system of the lung is choked and effective removal of subsequently inhaled dust is gradually diminished. Dust now accumulates around the lymph vessels and sheets of scar tissue develop in this location.

These preliminary changes do not constitute the disease, silicosis; they involve only the lymphatic system. The respiratory tissues are not appreciably affected. In good stereo X-ray films these preliminary changes may be visualized as a thickening and beading of the shadows cast by the blood vessels together with a widening of the shadow of the root of the lung. The blood vessels are thickened because the most important lymphatic vessels course through their walls. The beading is due to the minute nodules of silicotic scar tissue in the lymph nodes within the lung. The root shadow is widened because of the silicotic reaction in the lymph nodes in this location.

If still more dust is inhaled it can no longer be effectively carried off through the lymph vessels. The phagocytes now carry it into the walls of the air spaces. Often the cells group themselves in nodules but sometimes they are scattered. In these locations the silica again stimulates the formation of scar tissue. The result is a general thickening of the septa between the spaces and nodulation throughout the functional portion of the lung. This change seriously interferes with function as interchange of gas cannot take place through the thickened walls of the air spaces and the tissues lose their normal elasticity. Less involved air spaces dilate and their walls stretch, producing the condition known as emphysema, which is also incompatible with effective respiration.

With the formation of silicotic nodules in the functioning parts of the lung the

disease, silicosis, is said to begin. On the X-ray film such nodules cast clear cut, round shadows. As they increase in number and size they blot out the formerly accentuated linear shadows of the blood vessels. In a well marked case both lung fields may be thickly seeded with hundreds of such sharply defined, rounded, nodular shadows. Where there is emphysema, often at the bases, no markings are visible and the film appears black.

The condition of silicosis is a progressive one and it continues to develop after a man has left his dusty occupation. Serious results only follow, however, when he leaves with many nodules in the lung tissue itself. As such nodules increase in size they replace important functional elements. Obviously the continued growth of only a few nodules situated in the lymphatic system could have little effect other than to increase the obstruction already present.

The serious part of silicosis is its frequent complication by tuberculosis. This may arise from reactivation of a pre-existant latent infection or it may result from a new infection from without. The danger of infection increases with the amount of silicosis present. The course of the infection in the silicotic is usually chronic and it often fails to produce characteristic symptoms for months or years after its onset. Many times it is accidentally discovered in routine X-ray examination of groups of active workmen. However, evidences of disability are much more marked in silicotic individuals with infection than in those with uncomplicated disease. Occasionally tuberculosis may run a very acute course in the silicotic subject but this is also true in other persons. The cause of the susceptibility of the silicotic lung to tuberculosis is not perfectly understood, but it would seem that the action of the silica on the tissues creates a medium favorable for the growth of the bacteria. The tubercle bacilli multiply and a slowly progressive tissue reaction ensues, but the usual clinical symptoms of tuberculosis are often held in abeyance for a long time because the chances for absorption of poisonous products of infection are minimized by the obstructed lymphatic system. It is also possible that some of these poisons may be absorbed on the silica particles, as Cummins has suggested. Ultimately the infection gains the upper hand, symptoms develop and death from tuberculosis ensues.

The only other type of dust which is generally recognized as a cause of severe pulmonary injury is asbestos, a silicate of magnesium. This substance is fibrous in character and is more readily soluble than quartz. Apparently the protective upper respiratory mechanisms seem inadequate to prevent the inhalation of many such fibres and even quite long ones may penetrate into the finer bronchial tubes. These structures have irregularities in their walls which seem to hold the fibrous dusts. In this location the asbestos particles are surrounded and ingested by phagocytes. The cells do not seem to be particularly irritated for they are not especially numerous nor do they migrate rapidly into clumps as in the case of phagocytes dealing with quartz. Apparently most of them carry their burden of asbestos fragments directly into the walls of the tubes in which they lie. The mildly irritating dust sets up a proliferation of the connective tissue cells in the walls resulting in collar-like thickening of the tubes. Contraction of the new connective tissue narrows or closes the tube and thereby shuts off the ingress of air to the more peripheral air spaces. These spaces then collapse, a condition known as atelectasis. This in itself is a cause of fibrosis or scarring of lung tissue. The result is a fibrosis of numerous relatively small areas in the lung due to the collar-like constricting fibrosis about many small terminal bronchial tubes. The lymphatic system seems to play little part in disposing of asbestos dust and insignificant amounts of this dust are found in the lymph nodes.

Not a few cases of asbestosis die of tuberculosis or bronchopneumonia but the prevalence of the former infection is probably much less common than in silicosis. Surveys of large groups of asbestos workers fail to reveal such a high incidence of tuberculosis as found in similar studies on silica workers.

The inhalation of unusual amounts of non-siliceous dusts apparently fails to produce significant alterations in the lung. It is of course possible that injurious ones may ultimately be discovered, but none are recognized today. The non-siliceous particles enter the lung and are ingested by phagocytes as in the case of quartz. The cells are not stimulated to move very rapidly and as a consequence many of them remain within the air spaces for a long time. Some, particularly those con-

taining relatively few particles, make their way into the lymphatic system and accumulate in the lymph nodes. In sufficient concentration, or perhaps because they are mixed with small amounts of silica, they set up a low grade, non-progressive chronic inflammation. But this reaction results only in a little thickening in the lymph nodes and about the lymphatic vessels. The lung tissue may be variously pigmented, black from coal or red from iron, but there is no formation of scar tissue that interferes with function. The susceptibility to tuberculosis of the person with such pigmentation is not increased but it is apparently quite definite that pneumonia is unduly prevalent in coal miners and that chronic bronchitis results from the inhalation of cement. The X-ray films of men working in non-siliceous dusts shows either nothing at all or a more or less marked accentuation of the linear shadows cast by the blood vessels. This, of course, is due to the chronic inflammatory changes about the lymphatic trunks in walls of blood vessels.

The effects of mixtures of free silica and non-siliceous materials are less clearly defined and should receive more serious attention. It would seem unwise to assume, as has been done in the past, that the non-siliceous substances serve only as inert diluents and that only the quartz component of a dust is of significance. Experimental and clinical data are accumulating which suggest that at least some other dusts may materially modify the action of quartz, both in its capacity to produce nodular fibrosis in the lungs and to increase the susceptibility to tuberculosis. For example, animals inhaling heavy concentrations of a mixture of equal parts of pure silica and hematite dust for eight hours a day over a period of 14 months have shown not even the slightest indication of nodule formation in the lung, whereas pure quartz dust, inhaled for only two hours a day, produces definite nodulation. It also seems to be true that animals inhaling such a mixture are much less susceptible to tuberculosis than those exposed to pure quartz. In the case of the human iron miner, some lungs show definite silicotic nodulation, others only a very few nodules and many no nodules at all. All exhibit heavy deposits of iron particles with a cellular connective tissue reaction. When these findings can be correlated with chemical analysis of the lung tissues and the approximate amount of rock work which these miners have performed, some relationship between the exposure to silica and the fibrous nodule formation may be established. Tuberculosis in these iron miners is apparently less prevalent and much less severe than in men exposed to pure silica. What has been said of iron miners is also probably true of foundry workers and coal miners. It is generally accepted that men in the latter occupation have less tuberculosis than the average for the age period: Cummins believes that they have the infection but that it is so atypical and benign in its manifestations that it escapes diagnosis.

Mention should also be made of mixed dusts like certain slates and granite containing large amounts of free silica in combination with silicates. Experimentally these substances behave atypically and do not produce nodular fibrosis unless there is complicating infection. The same may also be true in human beings. Unquestionably such dusts are capable of producing typical silicotic nodules and they predispose to tuberculosis. From a theoretical standpoint they should receive much more intensive study to clarify our views on the whole subject. At the present time it appears that the action of their quartz component is modified and partially neutralized by the other substances which they contain.

Using Exhaust Systems and Respiratory Equipment To Protect Workers Exposed to Dust

By STUART W. GURNEY and DAVID S. BEYER

Liberty Mutual Insurance Company, Boston

The speaker said in part: A number of our states have had references to dust elimination in their labor laws for a good many years, but no state has as yet thoroughly enforced these laws. The codes of five states which we investigated provide that grinding, buffing and polishing wheels should be exhausted, although two of the states exempt from these provisions wheels on which water is used.