FILE NAME: Engineering (ENG)

DATE: 1934 Mar

DOC#: ENG021

DOCUMENT DESCRIPTION: Trade Journal Article - The Pathology of Various Mineral Dust Diseases

Accident Prevention | Health Conservation | Fire Prevention

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Vol. LXVII, No. 3 | March, 1934

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Published Monthly by

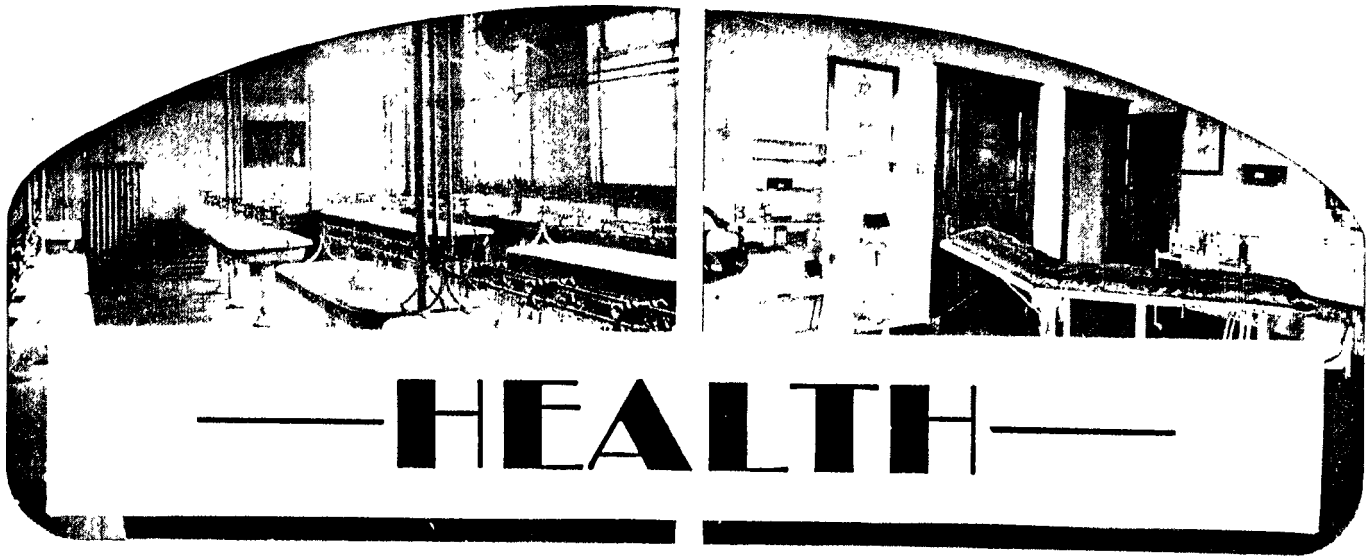
Safety Magazine Publishing Corporation

Editorial and general offices, 45 John Street, New York, N. Y. Publication office, 34 N. Crystal St., E. Stroudsburg, Pa.

ARTHUR C. CARRUTHERS, President — **AUSTIN F. HANCOCK, Vice President**

HARRY deG. ARMAND, Secretary

Contents of previous issues of *Safety Engineering* can be found by consulting the Industrial Arts Index in your library. Entered as Second Class Matter January 7, 1930, at the post office at East Stroudsburg, Pa., under the act of March 3, 1879. **SUBSCRIPTIONS; $3 a year in advance. In Canada, $4.00. Foreign subscriptions, $4.50. Single copies, 25c.**



—HEALTH—

THE PATHOLOGY of VARIOUS MINERAL DUST DISEASES

by LEROY U. GARDNER, M.D., of the
Saranac Laboratory for the
Study of Tuberculosis

Presented before the American Institute of Mining & Metallurgical Engineers, New York City, February 19, 1934.

THE lung is an organ developed to permit an interchange of gas between the blood and the external air. Being in free communication with the exterior it is more or less exposed to the action of atmospheric impurities, both particulate and gaseous. The following discussion will be confined to particulate contaminates.

Certain mechanisms are provided to protect the lungs from the accumulation of such foreign particles. The nose, through which the respiratory tract opens onto the surface of the body, is guarded by a coarse filter of hair. Behind this is a series of tortuous passages with moist walls which trap many smaller particles. In addition, the nasal cavities and the remaining portions of the upper respiratory tract, the pharynx, the trachea and the bronchi are lined by cells covered with minute vibratory hairs, the cilia. Wave-like vibrations of these hairs tend to carry particles lodging on their surface away from the lung and back toward the surface.

Particles which succeed in passing these barriers and penetrate to the terminal air spaces of the lung are ingested by wandering scavenger cells, or phagocytes, which come out of the partitions between the spaces for the purpose. These cells are capable of independent movement. They tend to carry the foreign particles out of the air spaces and into a special drainage system, known as the lymphatics. The lymphatics are minute vessels which drain into sedimenting basins known as lymph nodes. They are situated along the course of the vessels and bronchi, and at the root of the lung where the trachea divides into the two main bronchi.

For ordinary amounts of atmospheric pollution these protective mechanisms are adequate to prevent the significant accumulation of foreign particles in the functional part of the lungs. If an individual continues to work in very dusty atmosphere for long periods, his protective devices cannot cope with the situation, the mechanisms are themselves damaged and the dust particles collect where the air should be.

Lungs Can Absorb Much Dust

However, even excessive quantities of most kinds of dust are tolerated by the lungs. Loss of function seems to occur not from accumulations of particles inside the air spaces, or alveoli, but only after changes have taken place in the walls of these spaces. Such changes interfere with the permeability of the alveolar walls to oxygen and carbon dioxide and destroy their normal elasticity, so that the lung as a whole cannot expand and contract with respiration.

Of the common inorganic dusts to be found in industry only silica and some of the silicates, like asbestos, are known to produce dangerous reactions in the walls of the air spaces and the framework of the lung. Other substances apparently may accumulate in very large amounts and not result in incapacitation. They may produce pigmentation and perhaps cause the connective tissues along the lymphatics to thicken somewhat, but these changes do not interfere with function.

The Toxicity of Silica

Attention is therefore centered upon silica and its compounds. Silica in sufficient concentrations has been shown to be a cell poison. In colloidal form it is very toxic and when injected in large doses it may bring about active cell destruction and even death. Chronic silica poisoning causes the connective tissue cells to multiply and form scar tissue. Particulate silica will produce the same effects, if it is in a sufficiently fine state of subdivision, which suggests that its action is chemical in nature. Absolute proof of solution in the

AN ENLARGED LYMPH NODULE CHOKING OFF A LYMPHATIC VESSEL

body is still lacking although this is inferred from the available evidence.

To excite reaction in the connective tissues the silica particles must be concentrated in considerable amounts in immediate contact with cells of this variety. If the particles remain scattered through the air spaces of the lung there is no connective tissue reaction. But unfortunately silica particles act upon the phagocytes that have ingested them to bring about concentration of the irritant at the very point where it can exert its harmful effect. Phagocytes containing silica seem to move much faster than cells containing other types of dust particles. As a consequence silica is rapidly concentrated in the lymph nodes, both those inside the lung and those at its root. These structures contain connective tissue, which is stimulated, increases in amount and is gradually transformed into scar. The reaction in the nodes encroaches upon the lymph vessels associated with them; the lymphatic channels are narrowed or entirely blocked. Sheaths of new connective tissue develop about the damaged vessels. As this tissue matures it contracts and still further compresses the enclosed lymphatics.

The result is a progressive impairment of the pulmonary drainage system. If more particles are now inhaled they cannot be eliminated. They either remain inside the air spaces or they are carried into the walls of these structures. Here again they stimulate overgrowth of connective tissue cells. The result is scar tissue so dense that gases can no longer pass between the air space and the capillary blood vessel in its walls.

Nodules Form First in Lymph Nodes

The characteristic effect produced by silica is a nodule of very dense, glassy scar tissue. In ordinary silicosis of the lungs such nodules first form in the lymph nodes as already described. After they have developed to such an extent that the lymphatic system is obstructed, new nodules begin to appear in the thin walls of the air spaces. Under unusual conditions, where the atmospheric concentration of very fine silica is excessive, nodule formation and diffuse generalized thickening of the air space walls may constitute the first reaction. Apparently so much dust enters the lung that physiological mechanisms are quite inadequate. The lymphatic drainage system cannot begin to remove the particles fast enough to prevent an immediate effect in the functioning part of the lung.

These effects can be visualized in an X-ray film of a living subject being exposed to silica dust. In the early stages, before it is customary to diagnose the condition as definite silicosis, the roentgenogram reveals an undue prominence and beading of the radiating shadows cast by the blood vessels. This is due to the formation of new connective tissue about the lymphatics which course through the walls of the blood vessels. The beading is a visualization of small nodules in the associated lymph nodes. At the same time the shadow cast by the structures at the root of the lung is widened because of enlargement of the lymph nodes in this region. Later, when diagnosable silicosis has developed, the lung fields themselves are studded with great numbers of nodular shadows. As the latter increase in size and number they soon tend to obliterate the previously prominent linear blood vessel markings.

Silicosis a Progressive Disease

Silicosis is a progressive disease. If enough silica has been inhaled to initiate nodule formation, each focus continues to enlarge until finally a state of equilibrium is established. Nodules, which may not be visible by X-ray when a man leaves a silica industry, may subsequently increase in size so that they are readily detectable some years later. The progression of the process is favored and accelerated by the development of pulmonary infection.

Frequency of Complicating Tuberculosis

World wide experience with different groups of silicotic human beings has repeatedly demonstrated the frequency of complicating tuberculosis. In South Africa it is estimated that at least seventy-five per cent of miners with fully developed silicosis will die of this infection. In some cases the infection remains latent, presents none of its usual manifestations and merely modifies the character of the reaction to inhaled silica dust. In others it develops simultaneously with the silicotic reaction with a more or less typical localization in the upper portion of the lungs. Such cases may exhibit none of the characteristic symptoms of tuberculous intoxication for many years and are often only discovered in routine roentgenographic examinations of large groups of active employes. Ultimately these men develop symptoms, expectorate tubercle bacilli and finally die of tuberculosis, but the course of their disease is

protracted. There are still others whose roentgenograms show so much evidence of tuberculosis that the characteristic features of silicosis are obscured. Nevertheless they have had long exposures to dust and post mortem examination will reveal the nodular fibrosis produced by silica. These cases, like those of the preceding group, run a definitely chronic course and may not die of their infection until the fifth or sixth decade. Finally, there is another group, with well developed nodular silicosis, who apparently have never had a tuberculosis infection. Such men become infected, develop a rapidly progressive tuberculosis and die within six months. In them the symptoms of intoxication are more acute but bacilli may be very difficult to detect in their sputa and even in the lungs removed at autopsy.

Laboratory Animals Show Function of Tuberculosis

Approximations of these various conditions of infection in the human being have been reproduced experimentally in silicotic animals. By using an attenuated form of tubercle bacillus which produces only a transitory, self-limited infection in normal guinea pigs, it has been possible to show that silica dust specifically alters the course of such infection. If guinea pigs are infected by the inhalation of small numbers of such tubercle bacilli and immediately subjected to a prolonged period of silica dust inhalation the animals develop a chronic tuberculo-silicosis. This reaction makes its appearance after some five or six months exposure to the dust (eight hours daily) but the animals do not die for a year or two. They are comparable to the human silicotics of the first group. If the infection is administered first and the dust inhalation is commenced at different intervals thereafter it has been shown that the healing tubercles are reactivated and again become progressive. This result follows as long as the lung contains living bacilli. The disease produced is likewise chronic in its course and only kills its victims after many months. These animals may be compared to human beings who enter a silica industry with partially healed tuberculosis infections. Finally animals, which are first rendered silicotic by exposure to silica for a year or more and then infected by the attenuated tubercle bacilli, develop an acute tuberculosis which terminates fatally in a few months. Normal control guinea pigs, never exposed to dust, infected at the same time, never develop progressive tuberculosis. Their tubercles heal almost completely and they either die of other causes or must be killed two or three years later. The rapid tuberculosis which develops in the presence of preestablished silicosis has its counterpart in the last class of the human cases mentioned.

Effect on Tuberculosis Not Understood

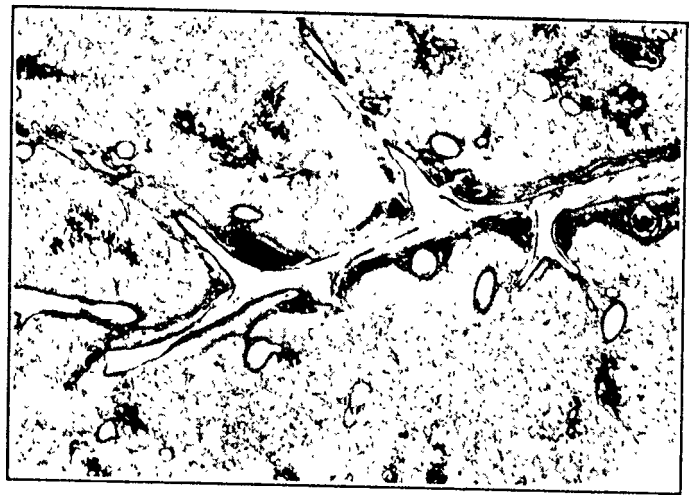
The cause for this effect of silicosis upon tuberculosis is not known. It is apparently quite specific, for marble, soft coal, aluminum oxide, gypsum, and hematite dusts have so far failed to influence the course of such infection. Asbestos, a silicate of magnesium, exerts a slightly stimulating effect but the resultant tuberculosis is rarely progressive and has usually healed after a year or more. The carbide of silicon has for some unexplained reason regularly produced progressive tuberculosis in animals infected in various ways with attenuated bacilli.

There are four possible explanations for the progression of the tuberculous infection: (1) alterations in anatomical structures of the lungs like the lymphatic system which may favor retention of bacilli as they are accidentally inhaled; (2) alteration of the tissues to offer a more favorable soil for the growth of the bacteria; (3) depression of the normal immunity mechanisms in the body, or (4) alteration of the bacilli so that they take on undue virulence.

Experiments at the Saranac Laboratory and elsewhere have quite conclusively demonstrated that bacilli recovered from silicotic lungs are not permanently altered. The attenuated organism which grows so vigorously in a silicotic environment will not produce progressive disease when transferred in bits of such tissue to a normal animal. Outside the silicotic lung it resumes its normal characteristic appearance and disease-producing capacity. Experiments to test the effect of silicosis on immunity are now in progress.

Silica's Reaction Is Easily Demonstrated

There is no difficulty in demonstrating that silica produces a reaction in the body which specifically favors the multiplication of tubercle bacilli. Kettle injected a definite quantity of fine silica particles beneath the skin of one flank of a white mouse, and in the opposite flank, the same quantity of aluminum oxide particles. A large dose of tubercle bacilli was then injected into the tail vein of the animal. The blood distributes these bacilli quite uniformly to all parts of the body, but if the animal is killed after several days large masses of them will be found at the site of the injected silica. Where the alumina particles have localized the bacilli are no more numerous than in any other part of the body. Apparently the reaction set up by the silica produces a favorable medium for the growth of these bacteria but later they disappear and may be very hard



EARLY STAGE. DUST CELLS HAVE MASSED IN LYMPH NODULES ALONG THE COURSE OF A VEIN

to find. The same seems to be true of the sputum of men with silicosis and tuberculosis.

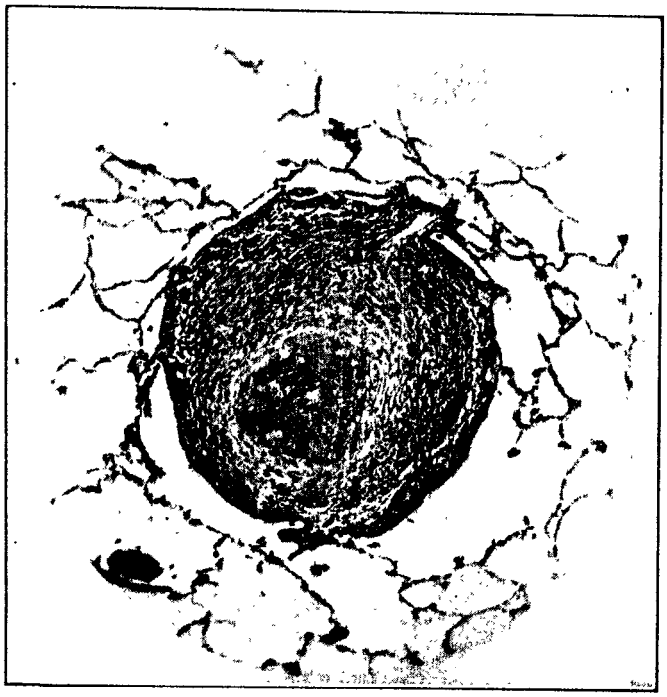
If silica is added to artificial culture media on which tubercle bacilli are to be grown the results are not so clearly defined. It seems as if this slow growing organism begins to develop more rapidly when the silica has been added, but the resultant quantity of growth is not much affected. Much more investigation of the whole problem of the relationship between silicosis and tuberculosis is needed.

Pathological Result of Asbestos Inhalation

Inhaled asbestos dust also produces fibrosis of the lungs but the scar tissue is not laid down in nodules nor does it have the peculiar glassy appearance which is characteristic of silicosis. In asbestosis one finds a widespread diffuse fibrosis throughout the framework of the lung. While not so spectacular as the nodulation of silicosis the fibrosis of asbestosis probably disables its victim more rapidly. The general thickening of the walls of the air spaces prevents interchange of gases throughout large portions of the lungs and brings about a state of deficient oxygenation in the whole body.

The reason for the peculiar distribution of fibrosis in asbestosis is apparently the fact that phagocytes do not transport the elongated fibres of asbestos for any distance. They tend to lodge along the walls of the fine cylindrical terminal bronchioles. There they cause fibrosis in the form of a collar, or cuff, about the tube and as this contracts it constricts the bronchiole. Air is thereby prevented from entering the peripheral ramifications of the tube and these portions then collapse. This in itself is a cause of fibrosis. As a result a generalized fibrous change involves extensive areas in the functional portions of the lungs.

How far asbestosis predisposes to tubercular infection is debatable. Animal experiments have not indicated an excessive susceptibility. Many autopsies on human beings have revealed a coexistent tuberculosis but statistical studies of large numbers of living workmen together with mortality records must finally settle this point.



SILICOTIC NODULE IN RABBIT LUNG, AFTER 13 MONTHS OF QUARTZ DUST EXPOSURE.

Sericite, a Silicate, May Be Harmful

Recently another silicate, sericite, has been incriminated in a series of papers by W. R. Jones. While it is too soon to comment authoritatively upon his thesis, it can be definitely stated that silicotic nodules can be produced experimentally without sericite. Petrographic analysis of the silica employed for the animal experiments made in the Saranac Laboratory has excluded contamination with this material. Normal crystalline quartz or, in some instances, crypto-crystalline silica have excited characteristic nodular fibrosis in guinea pigs, rabbits, white rats, cats, and domestic fowl.

Experience with other dusts, non-siliceous, is more limited but insofar as they have been studied it would seem that they do not tend to excite serious reaction in the lungs. They are ingested by phagocytes within the air spaces and quantities of particles may remain in this location for long periods of time. The walls of the spaces involved often exhibit a mild grade of chronic inflammatory change which is apparently not sufficiently marked to interfere with function. Many of the phagocytes leave the air spaces and enter the lymphatic system. They deposit their ingested particles in the lymph nodes within and at the root of the lung, but little or no scar tissue develops unless a sufficient quantity of silica has also been inhaled. These non-siliceous materials may cause pigmentation of the lung as, for example, the black lung of the coal miner and the red lung of the hematite miner, but mere pigmentation is harmless. The roentgenogram may reveal a slight accentuation of the linear blood vessel shadows and in some instances inflammatory changes in the air space walls may possibly cast a faint haze over the lung fields but none of these effects is at all definite. There is nothing to indicate that the inhalation of these non-siliceous dusts favors the development of tuberculosis. Other infections, notably pneumonia, are unduly prevalent in certain dusty industries. In how far the dust is responsible is not proved.

The carbide of silicon produces similar, non-progressive, chronic inflammatory changes in the normal lung. In experimental animals this dust has regularly and repeatedly increased the susceptibility to tuberculous infection. Whether the same effect obtains in human beings has not been ascertained.

In conclusion it should be reiterated that only silica and a limited number of the silicates are known to produce definite and serious pulmonary damage. In the case of silica this is associated with specific indisposition to tuberculosis and pneumonia. Of the silicates, asbestos is definitely recognized as a cause of pulmonary fibrosis. Its importance in predisposing to tuberculosis is not yet settled.