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 abraid 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 very 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-