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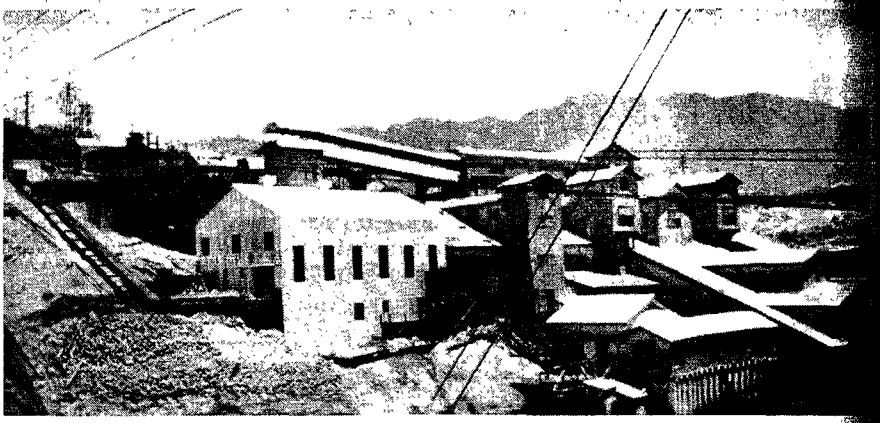

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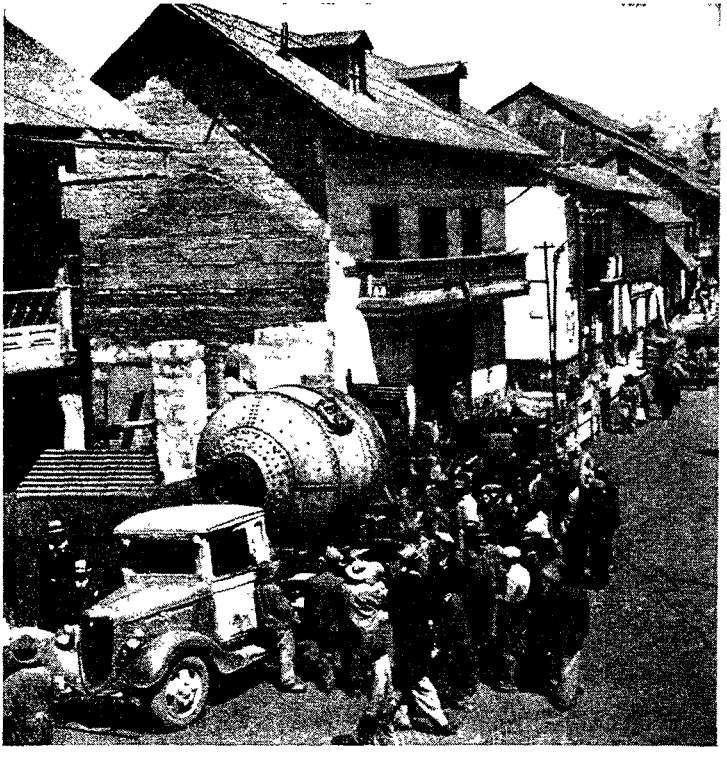

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MINING TECHNOLOGY

THIS periodical is the medium through which the American Institute of Mining and Metallurgical Engineers effects initial prompt distribution of TECHNICAL PUBLICATIONS (T.P.'s) sponsored by its Industrial Minerals Division and its Committees on Mining Methods, on Milling and Concentration and on Mining Geology. The contents of the annual volume of the TRANSACTIONS of the A.I.M.E. relating to the work of this Division and these Technical Committees will be chiefly selected from papers that appear in this publication.

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Technical Publication No. 887
(CLASS A, METAL MINING, No. 88)

Shaft Sinking on the Gogebic Iron Range

BY W. A. KNOLL,* MEMBER A.I.M.E., AND J. C. SULLIVAN†

(New York Meeting, February, 1938)

THE sinking of a new shaft at the Newport mine, Ironwood, Mich., was started in May 1931 and completed on Aug. 3, 1932. During this period, 2665 ft. of shaft in granite was completed, at an average advance of 6 ft. per 24 hr. In November of 1930, experimental work was conducted on 65 ft. of shaft through 22 ft. of overburden where ledge was encountered, and this work convinced the operators that all drilling could be done with vertical holes with the blasting relief to a 4½-in. hole drilled vertically in the center of the cut.

The reason for not adopting the conventional V-cut was to get away from damaging the shaft steel. The blasting in a V-cut naturally tends to throw the broken material upward, but with the cut as adopted the relief was in a horizontal direction to a large extent. The scheme of mucking employed made it necessary to keep the steel shaft sets within 50 ft. of the bottom at all times. This could not be done without serious damage with a V-cut when blasting in granite. The cut as drilled also broke 10.83 ft. per round of drilling, which was considerably more than the experience with a V-cut in the same material. The extended cut reduced proportionately the delays for charging, blowing smoke and handling drilling equipment in the shaft.

Another advantage was the ease in drilling vertical over inclined holes, and the preservation of the proper spacing of the drill holes at the bottom of the cut.

DRILLING

Air was carried down the shaft in an 8-in. flanged pipe line at a pressure ranging from 95 to 100 lb. This line was extended to within 100 ft. of the bottom, where a valve and a 4 by 4 by 6-ft. tee was connected to it. To each side of the tee was connected a nipple and elbow, the resulting span of the two elbows being about 4 ft. Lengths of short 3-in. hoses

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† Mining Engineer, Pickands Mather & Co.

ing fibrosis or the formation of scar tissue. This reaction is a result of stimulating connective tissue, the supporting framework that holds the functional cells of all organs in place. The connective tissue of the liver is composed of the same elements as the connective tissue in the lung, although it assumes different forms in the two organs. Since it has been shown conclusively that free silica has the same effect upon connective tissue regardless of the organ in which it happens to be located, it has been assumed that the reaction to other minerals will also be demonstrable on connective tissue in any part of the body. Experience is indicating that this is true except in regard to the fibrous silicates composing the asbestos group. Thus far these substances have had little effect outside the lungs and have produced fibrosis only in that organ. The capacity of any *particulate* mineral to provoke fibrosis can probably be determined with equal facility in all organs whose structure permits the accumulation and retention of injected material.

The minerals used for the tests to be discussed have been analyzed chemically and petrographically to make certain of their purity. They have been pulverized in mills, elutriated in water to secure particles 5 microns or less in diameter and again analyzed to check their composition. By weight 1 per cent and 10 per cent suspensions have been prepared in physiologic salt solution and sterilized at low pressures. Rabbits have been injected by ear vein with 1 gram of particles, administered in 20 doses over a period of 2 months. Guinea pigs have received a single 200-mg. injection directly into the abdominal cavity. At least four rabbits and five or more guinea pigs have been used to test each mineral. The animals have been killed at intervals during a two-year period to observe the reaction of the tissues and to determine whether changes of a progressive nature have occurred.

Capacities of Minerals to Provoke Reaction

All of the observations have not yet been completed but the accompanying tables indicate the scope of the investigation and the maximum period that each substance has been in contact with the tissues. The minerals tested have been grouped in the following classes:

1. Uncombined silica, of which most forms are active tissue irritants.
2. Silicates, which in most instances are either inert or productive of slight chronic inflammation.
3. Nonsiliceous minerals, which in general act like the silicates.
4. Mixtures of free silica with other minerals, in which the action of the silica is retarded and modified in varying degrees.

Signs Used in Tables

The capacities of these different minerals or combinations of minerals to provoke reaction in the tissues of living animals have been compared

and the results indicated by numerals and plus signs in the accompanying tables as follows:

± indicates the minimum reaction, consists merely of phagocytosis; i.e., the ingestion of the particles by wandering "dust cells." Inert substances apparently remain inside the bodies of such cells for indefinite period without exerting appreciable effects upon the surrounding tissues.

+ indicates that the mineral may be very slightly irritating. As a manifestation, microscopic accumulations of lymphocytes (chronic inflammatory cells) are found in the immediate vicinity of the dust cells.

2+ indicates that the fixed connective tissue cells directly adjacent to the dust cells have been irritated and there is slightly more evidence of chronic inflammation.

3+ signifies localized nonprogressive fibrosis. The injured connective tissue cells have begun to multiply as a result of irritation but the proliferation does not progress far and remains localized in a zone close to the irritating particles.

4+ is more widespread fibrosis, which is suggestive of silicosis but which has not attained mature hyaline form.

5+ *represents the standard reaction to normal quartz.* The functional cells of the organ are injured and destroyed; the phagocytes, irritated by the silica they contain, are stimulated to migrate and concentrate the particles in focal areas. More tissue is injured by the resultant high local concentrations, and fibrosis of a peculiar and characteristic type develops. Such fibrosis takes the forms of nodules about the collected masses of silica; it may also be diffuse when the particles are so numerous that the phagocytes fail in their attempt to remove them.

6+ and 7+ are necessitated by the very rapid fibrosis caused by crystobalite and tridymite. With these toxic substances the phagocytes that constitute the primary line of defense are apparently paralyzed at the outset. The moribund cells do not even attempt to remove the irritant and as a consequence the particles are not collected in focal areas. They remain scattered and injure the tissues wherever they lie, and the fibrosis that rapidly follows is diffuse rather than nodular in type.

8+ is used for the very acute inflammation that immediately follows injections of dispersed colloidal silica and sodium silicate. Often the process is so rapid and extensive that death promptly ensues. In nonfatal cases moderate degrees of proliferation or fibrosis may develop later but rapid elimination of the fluid irritant fails to maintain the reaction indefinitely.

Class I—Uncombined Silica

The following table lists the different forms of free silica which have now been tested and indicates their comparative capacities to provoke reaction. The responses to all except tridymite and dispersed colloidal

silica have been followed for at least a year. Tridymite has killed every animal within a period of 3 months. Colloidal silica in the standard dose used for all these minerals has been followed for only 1½ months.

TABLE 1.—*Free Silica*

Crystalline Forms		Cryptocrystalline Forms		Amorphous Forms	
Normal quartz	5+	Chalcedony	5+	Colloidal silica, dispersed	8
Tridymite	7+	Tripoli (Seneca, Mo.)	5+	Colloidal silica, gel	
Crystobalite	6+	Flint	5+	Opal	4
Vitreous silica	5+			Diatomite	5

In supplementary experiments it has been demonstrated that the dose of 1 gram arbitrarily selected for intravenous injection into rabbits is excessive with pure free silica. As small an amount as 0.05 grams of quartz will produce essentially the same effects, although at a somewhat slower rate.

Other injection tests have shown that the rate of reaction to silica is inversely proportional to the size of the particles. A splinter of quartz several millimeters in diameter, which has been embedded in the connective tissue beneath the skin causes no fibrosis within a period of one year. Intravenous injection of particles 10 to 12 microns in diameter excites the formation of microscopic tubercle-like nodules, which are cellular rather than fibrous in character and which increase very little in size during a period of two years. The same weight of particles 1 to 3 microns in diameter is responsible for the progressive fibrosis that has been described as characteristic of quartz. If a gram of particles 1 micron or less in diameter is injected in the same manner, acute degenerative changes develop in the organs where they lodge and most of the animals are dead within 3 months. Obviously the reaction to silica is related to the amount of surface in contact with the tissues.

The behavior of the amorphous silicas requires comment. In the dispersed colloidal form, silica is a very active tissue poison. In small doses it causes acute degenerative changes (8+), which are followed by a limited degree of proliferation simulating the early stages of the response to quartz. When such colloidal silica has gelled, it loses all capacity to affect the tissue. One year after injection the only visible reaction is the presence of phagocytes filled with the particles. Opal, a natural amorphous silica, is moderately irritating and sets up cellular changes, which temporarily progress like those induced by quartz. Later, however, the reaction subsides, leaving a limited amount of scar tissue. On the other hand, celite, an amorphous silica composed of siliceous diatome shells, provokes a progressive fibrosis indistinguishable from the reaction to quartz.

Class II—Silicates

The artificial silicate of sodium, not listed in Table 2, has an effect very much like that of dispersed colloidal silica (8+). When injected in the standard doses its effect is uniformly fatal. Minute repeated doses totaling 13 mg. when injected directly into the lungs of guinea pigs failed to produce fibrosis after a period of 4 months.

The other silicates, with the exception of the mica, biotite, have generally proved to be as inert as silica gel. Most of them have provoked only the initial reaction of phagocytosis (±) or have produced very slight degrees of chronic inflammation (+) in the immediate vicinity of the dust-filled cells. Talc and muscovite have caused a little more extensive inflammation (2+), but with the former this change has shown no tendency to progress in 2 years. The response to both muscovite and biotite has been under observation for only 6 months, but in this period biotite has excited definite proliferative changes (3+), which now look as if they would progress. The ultimate effect cannot yet be anticipated. It is of interest to note that the two sericites are almost inert. One of them was selected by Professor Larsen, of Harvard, as a fibrous type comparable to the form that Jones believed was an important cause of silicosis.

Table 2 lists the various minerals now under investigation and indicates their comparative irritating powers for the maximum period of observation. The "deaths" noted in the rabbits injected by vein are of mechanical origin and are of no significance to the subject under discussion. A larger unit dose of the same minerals has had no effect when injected into the abdominal cavity of guinea pigs.

The experience with the finely ground asbestos minerals, anthophyllite, amosite, amphibole, crocidolite and chrysotile, has been most instructive. In the liver and other organs none of them has caused even a suggestion of the fibrosis that characterizes the picture of pulmonary asbestosis. Furthermore, there has been no formation of asbestosis bodies, those peculiar structures always found in human and experimental asbestosis of the lungs and due to a deposit of iron and albuminous material on the surface of the fiber. The absence of reaction in extrapulmonary tissues to fibers ground as finely as these (3 microns) constitutes part of a chain of evidence which may ultimately demonstrate that the fibrosis in pulmonary asbestosis is due to mechanical irritation by long, flexible, mineral fibers. In the lung, which moves actively in respiration, such foreign bodies would be more apt to injure the tissues mechanically than they would when embedded in a relatively immobile organ like the liver. If the irritation from asbestos were chemical in nature one would expect that it would affect connective tissues in any part of the body and that fibrosis would develop most rapidly upon injection of very fine particles, which present such a large surface to the tissue.

TABLE 2. *Irritating Powers of Minerals*

Minerals			Effect on			
			Rabbits		Guinea Pigs	
Anhydrous and Hydrous	Group	Variety	Maximum Time Observed, Months	Tissue Reaction	Maximum Time Observed, Months	Tissue Reaction
Anhydrous:						
Polysilicates ($R_2Si_4O_9$)	Feldspar	Na-Microcline	24	±	12	+
Metasilicates ($RSiO_3$)	Pyroxene	Rhodonite	0		0	
	Amphibole	Anthophyllite	12	+	12	±
		Amosite-fibrous	6	±	4	-
		Na-Tremolite	6	+	8	±
		Amphibole	3	+	8	±
		Crocidolite	6	±	8	±
		Beryl	1	+	0	Not toxic
				Deaths		
Orthosilicates (R_2SiO_4)..	Garnet	Almandite?	24	±	12	+
	Topaz	Sillimanite	24	±	12	+
Hydrous:						
H_2O crystallization.	Zeolite	Analcite	24	±	12	+
H_2O essential	Mica	Muscovite	6	2+	1	2+
		Sericite-fibrous	21	+	12	±
		Sericite-platy	24	±	12	±
				Deaths		Not toxic
		Biotite	2	3—?	1	3+
				Deaths		Not toxic
	Serpentine and	Serpentine	6	—	12	±
		Chrysotile	9	+	4	+
				Deaths		Not toxic
	Talc	Talc	24	2+	12	2+
		Glauconite	12	+	12	+
				Deaths		Not toxic
H_2O [illegible]	Kaolin	Dickite	3	+	1	2+
		Halloysite	0		1	2+
		Montmorillonite	0		2	+
Anhydrous	Titanosilicate	Titanite	6		0	

The carbide of silicon (SiC), an artificial compound widely used as an abrasive, has produced practically no reaction. The masses of particles lie inert in the tissue for years without provoking more than a strictly localized, nonprogressive chronic inflammation of slight degree. Animals inhaling high concentrations of this dust for 2½ years have shown no more reaction in the lungs than has been discovered in other organs after injection.

The metal silicon, tested by subcutaneous injection, has excited neither inflammation nor fibrosis.

Colorimetric tests for the amount of soluble silica liberated in the heat-sterilized, saline suspensions of different forms of free and combined silica are now being made. As far as this part of the work has progressed, it has been impossible to establish any correlation between the degree of solubility *in vitro* and the capacity to provoke reaction in the living body. If anything, the least active forms of silica seem to be the most soluble. The inert silica gel yields 126.6 parts of soluble silica per million while the very active quartz gives a figure of 6.6. The solubilities of the silicates vary widely, as might be expected; halloysite showed a solubility of 4.5 p.p.m., while amphibole gave 43.7 and glauconite 52.3 p.p.m. It is emphasized that these figures represent solubilities in unbuffered isotonic sodium chloride solution. When similar tests have been made at the pH of the body in serum or other fluid, undoubtedly there will be different results, but it hardly seems probable that the existing ratios will be reversed.

Class III.—Nonsiliceous Minerals

Table 3 shows that, except for the toxic sulphides and dolomite, the nonsiliceous minerals like the silicates have very little effect upon the tissues. Dolomite is readily soluble and its high alkalinity undoubtedly is responsible for the effects produced in the concentrations injected. By inhalation, so few particles could enter the lungs at one time that the acid-base equilibrium of the body would not be disturbed.

Certain generalizations are permissible even at this incomplete stage of the study. Free silica is obviously capable of producing a rapid and progressive fibrous reaction, which no other mineral can even approximate. The silicates, as a class, are not irritating substances; exceptional species possess irritating properties whose nature has not yet been defined. Nonsiliceous particles are inactive, except the minerals that are definitely poisonous. These observations are not new; they simply confirm what has long been recognized.

The generally accepted belief that the properties of hardness and [illegible] are not responsible for [illegible] simply confirmed by [illegible]

of investigators, does not satisfactorily explain all the observations that have been presented in this paper.

Our demonstration of a marked variation in reaction to different forms of amorphous silica suggests that if solubility plays any part in the process it is not the only one. The *form* in which the soluble material exists is probably much more important as indicated by the loss of activity asso-

TABLE 3. *Nonsiliceous Minerals*

Mineral	Effect on Rabbits: Maximum Period Observed, Months	Rabbits: Tissue Reaction	Guinea Pigs: Maximum Period Observed, Months	Guinea Pigs: Tissue Reaction	Other Tests: Maximum Period Observed, Months	Other Tests: Tissue Reaction	Other Tests: Type of Test
Diamond	36	±					
Coal, bituminous	12	2+	12	+	41	±	Inhaled
Coal, anthracite (1 76 per cent SiO_2)	12	±	12	±		0	SubQ
Aluminum oxide	24	±	12	±		±	SubQ
Rutile			11	+	11	±	SubQ
Marble			1	0	3	0	SubQ
					31	±	Inhaled
Gypsum, satin spar	3	+					
Gypsum, calcined	6	0	5½	2+ (Calcif.)	24	±	Inhaled
Galena			8	Abscess and repair			
Chalcopyrite			8	±			
Iron sulphide			8	±			
Zinc sulphate (roasted sulphide)	11	2+	12	Abscess fibrous, repair			
Fluorite (SiO_2 0 8 per cent)	18	2+	11	2+			
Dolomite	40 days	± Deaths	0	Toxic Deaths			

ciated with gel formation in colloidal silica. Here again more observation is necessary before any conclusions can be reached.

CLASS IV—MIXTURES

The last group of minerals to be discussed includes natural and artificial mixtures of free silica and other substances. In many respects these are the most important, for mixtures rather than pure silica are encountered in most industrial atmospheres. Only part of the behavior of the mixed dusts can properly be discussed in this section dealing with injected material. It will be shown that *for the period of observation* the presence of small and sometimes of large amounts of silica have had little effect. The reaction during the first two or three years apparently does not differ from that of the nonsiliceous components of the dust.

Table 4 indicates the quantities of free silica administered in association with other minerals that have failed to produce fibrosis in various periods of observation. The injection of 3.3 grams of granite dust that was proved to contain 1 gram of quartz did not cause as much reaction in a year's time as the quartz alone provoked during the period required for its administration. One gram of the particular sample of South African mine dust employed contained 0.7 gram of quartz, and yet after 16 months of contact with the body the response had not attained a mature stage. The reaction was not nearly as extensive nor as advanced

TABLE 4.—*Mixtures of Silica and Other Substances*

Mineral and Silica Content	Effect on Rabbits: Maximum Period Observed, Months	Rabbits: Tissue Reaction	Guinea Pigs: Maximum Period Observed, Months	Guinea Pigs: Tissue Reaction	Other Tests: Maximum Period Observed, Months	Other Tests: Tissue Reaction	Other Tests: Type of Test
Hematite (6 per cent SiO_2)	12	±	12	+	42+8[a]	+	Inhaled
Anthracite (10 13 per cent SiO_2)	12	±	12	+	21	0	SubQ
Crude fluorite (10 39 per cent SiO_2)	12	3+	11	2+	11	±[b]	Inhaled
	15½	4+					
Granite (35 per cent SiO_2)	12	2+	12	3+	30	2+[b]	Inhaled
standard dose (0 3 gram SiO_2)	15	3+					
3x standard dose (1 0 gram SiO_2)	12	2+					
Ferruginous chert (50 per cent SiO_2)	22	3+			22½+	+	Inhaled
Standard dose (0.5 gram SiO_2)	12	4+	12	3+	26[a]		
2x standard dose (1 gram SiO_2)	19	5+	12	3+			
So African mine dust (70 per cent SiO_2)	16	4+	12	3+			

[a] 42 months in the dusting room +8 months in a normal atmosphere, and, similarly, 22½ months in the dusting room +26 months in a normal atmosphere

[b] In one or two animals nodular fibrosis (4+) developed in the lymph nodes draining the lungs but pulmonary fibrosis was lacking

as that provoked by 0.5 gram of pure quartz in 11½ months. At the opposite end of the series is hematite, a gram of which contained only 6 mg. of free silica. Since this quantity is only about one-tenth the amount of pure quartz necessary to cause fibrosis, it is not anticipated that silicotic reaction will ever develop.

The observations to date make it seem probable that if a mixture contains much more than the minimal effective dose of free silica, fibrosis may ultimately develop but at a very slow rate. The time required and the character of such fibrosis will probably vary with the properties of the adulterant dust. Some of these substances seem to be more potent than others in temporarily inhibiting the action of silica. For example, note that anthracite coal with 10 13 per cent of silica caused only a phagocytic reaction within a period of 12 months, crude fluorite with 10.39 per

cent. after causing a localized proliferation [illegible] more extensive reaction in another 3½ months. [illegible] seen to be as effective an inhibitor as anthracite coal, although this conclusion needs verification. These results have been too uniform throughout the various animals used for each test to doubt their validity.

The same mechanisms undoubtedly operate in the lungs to modify the action of silica inhaled with other dusts. In addition, siliceous and other kinds of particles tend to agglutinate with one another in the atmosphere. The resultant aggregates are so heavy that they fall to the ground or so large that they cannot be inhaled. This effect retards the accumulation of dust in the lungs. The right-hand columns of Table 4 indicate how very slight has been the response to prolonged inhalation of different mixtures. The atmospheric concentrations in these experiments have been high, always in excess of 500 million particles per cubic foot of air (light-field counts) and in many instances in excess of 800 million. In no animal has there been appreciable reaction in the lungs, and much less a suggestion of nodule formation. With crude fluorite and granite, early silicotic nodules developed in the lymph nodes draining the lungs; with pure quartz, on the contrary, an exposure of 12 to 18 months to a concentration of only 100 million particles per cubic foot has produced generalized nodulation throughout the lungs. The varying degrees of tissue reaction are paralleled by the amount of silica detected by chemical analyses of the lungs. After 22 months inhalation of chert, for example, the lung tissue revealed 10.25 per cent of ash, of which 3.18 per cent was silica. After 19½ months in one-third the concentration of pure quartz the lung yielded 12.27 per cent of ash with a silica content of 39.6 per cent. Thus it is concluded that the substances other than silica in mixed dusts play an important part in modifying its influence on the body, and that the hazard from such dusts is not only proportional to the quantity of free silica but is inversely modified by the other components.

INFECTION

To comprehend fully the effect of inhaled dusts one must have some knowledge of the influence of infections. Any portion of the body in which tissues are damaged by the scars of previous infections will not dispose of dust particles in the same manner as a normal area. Scar tissue tends to retain foreign bodies mechanically and the lymphatic drainage system may be so altered that it functions imperfectly. Mildly irritating mineral particles sometimes even [illegible]

[illegible]

Even more important than these considerations is the influence that silicosis exerts upon susceptibility to tuberculosis. Statistical analyses and clinical examination of silicotic human beings have amply demonstrated that such individuals have much more tuberculosis than other members of the population, and that the infection is often extremely chronic, causing symptoms only in the latter years of life. Experimental methods have duplicated these findings in animals and pointed out certain principles that are of some value in understanding the mechanisms involved.

Time will not permit a detailed discussion of this phase of the subject. Here it suffices to state that of the different minerals tested only free silica has a very marked influence upon susceptibility to tuberculosis. With calcium compounds there is a tendency to deposition in the areas of tuberculous degeneration and an accelerated healing of the infection. Some of the nonsiliceous dusts cause slight local extensions of the infection, which quickly heal to form small scars. The silicates as a class have not yet been proved to be any more potent than nonsiliceous minerals. On the other hand, free silica, in the body at least, seems to have a specific stimulating effect upon the growth of tubercle bacilli. These organisms grow with unusual rapidity in tissues being killed by high local concentrations of silica; in old silicotic scars they are able to remain alive for long periods of time but cease to multiply actively.

In human beings with quiescent encapsulated foci of tuberculosis, phagocytes may in time transport enough silica particles into the foci where the bacilli are localized to cause them to multiply and spread. The disease that results is the extremely chronic combination of silicosis and tuberculosis known as "silicotuberculosis." The same condition apparently can also be produced by new infection from without, in silicotic subjects with marked immunity to tuberculosis. Whether the mechanisms governing such immunity are altered by silicosis has not been discovered. In silicotic animals and in men with no immunity to tuberculosis, a first infection with the tubercle bacillus after the reaction to dust is mature produces an unusually acute reaction ("perinodular tuberculosis"), which terminates rapidly. Fortunately such cases are rare.

Mixtures of silica and materials like hematite and coal may cause widespread extensions of the infection followed by healing with the formation of massive scars. Possibly some of the so-called conglomerate types of simple silicosis in human beings arise in this manner. When they come under observation nothing but the scars of the infection are left; the organisms [illegible]

Being organs connected with the outer atmosphere, the lungs would soon become clogged with foreign material if they were not protected. They have a system of filters to prevent dust and bacteria from entering and another system for eliminating such particles as pass the first line of defense. The filtering apparatus is found in the tortuous passages of the nose, throat and wind pipe, which are lined with a sticky, mucous membrane, covered with vibratory hairs whose motion, like that caused by wind upon a field of grain, wafts foreign particles toward points from which they can be expectorated. The eliminating mechanism consists of the wandering phagocytes, or dust cells, mentioned in other organs and a system of fine drainage vessels, the lymphatics. The latter are found in the pulmonary framework, chiefly in the walls of the blood vessels, bronchi and some of the partitions dividing groups of air spaces. The dust cells move freely over the inner surfaces of the terminal air spaces and ingest any particles that may be present. They carry the foreign material to the lymphatic vessels, by which both cells and their contents are transported to lymph nodes. The latter act as sedimenting reservoirs; they are located both in the framework of the lung itself and in the connective tissue about its root. By these means the delicate membranes forming the walls of the air spaces are kept clean and in condition to permit a free interchange of gas to and from the blood capillaries that they contain.

The mechanisms are adequate for ordinary atmospheres. It is only when man thoughtlessly pollutes the air he breathes with excessive quantities of fine dust that his protective apparatus fails him. When this happens the dust cells begin to accumulate in the delicate connective tissues about the lymphatic vessels and are no longer eliminated. If the exposure continues, they form larger collections in these locations and then gather on the walls of the air spaces themselves.

The fibrous minerals of the asbestos group present an unusual problem to the protective mechanisms. They are too long to be transported by mobile dust cells, consequently they are rarely found in the lymph nodes inside or outside of the lungs. Most of them remain in or upon the walls of the terminal bronchial tubes, where they irritate (perhaps by mechanical means) and cause a fibrosis, which gradually extends toward the periphery of the lungs.

The injection experiments already cited indicate the effect that different forms of mineral particles may be expected to produce upon connective tissues. If any of the particulate dusts should accumulate in the lungs in appreciable quantities, it will have the same effect in this organ and its associated lymph nodes. The active forms of free silica that are inhalable should produce progressive fibrosis. Asbestos will cause fibrosis, though in another form. Other mineral dusts with a few possible exceptions will have little effect.

Nonspecific Pneumoconioses

The nonsiliceous materials and most of the silicates as they accumulate in the lymph nodes and in the connective framework of the lungs cause pigmentation, with or without microscopic accumulation of inflammatory cells in the immediate vicinity of the dust. The walls of the air spaces themselves are involved only after very unusual exposures. The gross effect upon a lung thus involved is to produce linear and focal deposits of pigmentation throughout the organ. The largest ones occur in the walls of the branching treelike blood vessels and in the lymph nodes. Smaller ones are found in the partitions between groups of air spaces. All of these deposits are soft and impalpable because there is no associated fibrous reaction. The shadows of such changes on an X-ray film appear largely as a thickening of the treelike branches of the blood vessels. The lymph nodules inside the lung are too small to cast an appreciable shadow; the larger ones at the root are not dense enough to offer any contrast with the shadows of other structures immediately about and over them.

In X-ray pictures, linear exaggeration is characteristic of the reaction to all the nonspecific types of dust. It may also be produced by some chronic infections and by hardening of the pulmonary arteries. From mere examination of a film one cannot determine the cause. This is not so unfortunate as might be inferred because such alterations do not interfere with the function of respiration and do not give rise to clinical symptoms.

Silicosis

With free silica the initial reactions are essentially the same as those with other dusts. But the irritating particles kill the phagocytes when too many of them are ingested and in smaller numbers they stimulate increased migratory activity. Wherever the cells concentrate enough silica particles in contact with connective tissue, the latter undergoes characteristic proliferation terminating in nodule formation. Since the lymphatic system collects and concentrates the particles in the lymph nodes, the first evidence of silicosis occurs in these organs. When only a little silica has been inhaled the only changes that appear may be found in the nodes. The development of fibrosis in the sedimenting areas obliterates the channels and the efficiency of the drainage system is impaired. Subsequently inhaled silica now accumulates in and about the lymphatic vessels inside the lungs. A mixed linear and nodular type of fibrosis ensues. The shadow on the film at this stage cannot always be differentiated from that produced by other forms of dust. Generally, however, minute nodules develop simultaneously in the walls of the air spaces. Only when the nodules in the latter position become large enough

to cast definite shadows is one justified in making a diagnosis of silicosis when supported by a history of exposure. When enough silica has been inhaled and large numbers of "discrete fibrous nodules are uniformly distributed throughout all portions of both lungs," the disease is mature.

Silicosis will then progress regardless of further exposure. But the progression is self-limited. The nodules will attain a maximum diameter of 4 to 6 mm., and then they cease to grow. They never progress until the whole lung is replaced by scar tissue. In this stage of "discrete nodulation" there are still large amounts of uninvolved lung tissue capable of carrying on the function of respiration. As a consequence, symptoms are not necessarily produced. The subject may be somewhat short of breath on sudden and unusual exertion. At his customary work he is generally unaware of any limitation. He has nonclinical silicosis.

This is the classical picture of silicosis that has developed in an otherwise normal lung. If portions of the pulmonary tissue have been damaged by previous infection or by other causes, the silica is deposited locally in excessive quantities. The irritating particles act upon the previously injured cells and accentuate the existing inflammatory reaction. A massive area of diffuse fibrosis ultimately develops in this location, while in other parts of the lungs the customary discrete nodules are formed. If the massive fibrosis reaches the pleura, this structure participates in the reaction and chronic fibrous adhesions develop, which fix the lung to the chest wall so that it cannot move with respiration. To compensate for the obliterated air spaces, those in other parts of the lung dilate, producing the condition known as emphysema. The man with areas of massive fibrosis and associated chronic pleurisy and emphysema is short of breath. This symptom may be so severe that he experiences it even while at rest. He is almost always disabled and often totally so.

Silicosis with Infection

One of the puzzling features of these cases of massive fibrosis is the origin. In the particular case, has the scar tissue developed upon the basis of an infection that has healed and is free of living bacteria or are the organisms that caused it still alive? This leads us to the subject of tuberculosis, the most common type of infection to complicate silicosis. Tuberculosis may arise from latent infections, which were established before the subject had become silicotic, or it may result from new infections, acquired subsequently. Tuberculosis complicating silicosis is apt to be unusually chronic, the area of disease is often detectable in an X-ray film for years before it gives rise to the ordinary symptoms and before tubercle bacilli appear in the sputum. For these reasons it is hard to differentiate from conglomerate silicosis developing in lungs damaged by a healed infection. The physical examination and the clinical behavior of the subject may give a clue but in many cases symptoms are absent and

only repeated X-ray films taken year after year to determine the rate and extent of progression can establish the nature of the underlying cause. When the conglomerate disease is due to an infection that is still active, it manifests itself ultimately by the symptoms of intoxication, fever, loss of weight, malaise, etc.; but by this time the localized area of fibrosis has broken down and perhaps formed a cavity; and tubercle bacilli have appeared in the sputum. Anyone can now recognize the condition as tuberculous. From this time on the patient becomes rapidly worse and is generally dead within a few years. There are other forms of tuberculosis in the silicotic, a very rare, acute form and a somewhat more common one, which from the outset behaves like tuberculosis in the general population. These offer few diagnostic problems because they cause characteristic symptoms.

Simple silicosis can and is being prevented. In its discrete form, as it develops in normal lungs, it causes so little disability that the existing cases are not a great problem. In its conglomerate form, on a background of previous injury it causes dyspnoea and is disabling. The cases that have already developed must be pensioned as they become disabled; the system of preemployment examination and the measures for reducing atmospheric dust concentrations will probably prevent more from occurring.

The great problem remaining before us today is the prevention of new tuberculosis in men who already have silicosis and the treatment of the silicotuberculosis that has developed but is now in latent stages.

The way has already been charted for the prevention of new infection. Treatment demands new methods, for those now used so satisfactorily for ordinary tuberculosis are not yielding encouraging results. This is the challenging problem that must be solved.