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PNEUMOCONIOSIS

By ROBERT B. HUNT, M. D.
Boston, Mass.

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SINCE the middle of the 16th century, it has been observed that men who have worked in a dusty occupation have been more prone to contract diseases of the lungs than those in any other occupation. Down through the following years has come the ever-increasing knowledge that men who have worked where dust is great and the exposure long, showed a considerable prevalence to respiratory infection. We note various terms which have been coined in the different types of industry referring to diseases of the lungs, such as grinder's rot and miner's asthma.

Whenever the incidence of tuberculosis rises sharply above the normal average in any community, there usually follows an investigation as to the cause. The British government experienced that condition in the gold mines of South Africa and Australia. A general survey of the areas showed a marked increase in tuberculosis, and following the report of the survey intensive study was made of the working conditions. When the report of that work was published, it stimulated a considerable amount of interest in the United States and investigations revealed that our miners were affected in a similar manner. Incidentally, it stimulated scientific and experimental work as to the cause and effect of various types of dust upon lung tissue.

During the early part of the last depression, thousands of men lost employment through no fault of their own. All workmen have a right to live and when finances were gone and men were confronted with want and starvation, they quite obviously turned to every factor which might provide them with sufficient funds to support themselves and their families. A general depression does not affect one class but all classes, and it might have been expected that many of the unemployed, aided and abetted by the legal profession, would eventually find their way to their physician's office, and that is exactly what happened in this country. The outcropping of thousands of claims for silicosis suddenly burst upon the unsuspecting world, and industry and labor were confronted with a serious economic and social problem. With this increase in the number of claims, there also arose a certain amount of hysteria among all of the factions more or less concerned with such claims. Those of a pessimistic nature might consider the outcropping as an emergency; but industry has met and conquered emergencies of greater moment

in the past, and I have every reason to believe that the solution of this problem will terminate to the mutual satisfaction of all concerned.

What is this condition called pneumoconiosis? To clarify and remove any reasonable doubt as to the terminology used in this discussion, let me explain the meaning of several words:

Pneumoconiosis refers to the presence of dust within the lungs whether that dust is harmful or harmless, organic or inorganic. It is a general term to denote the cause and location of the disease but is so broad in its scope and so non-specific that we try as far as possible to use a term more applicable to the individual case. It is not a diagnosis but merely a symptom.

Silicon is an element which never occurs in the free state, so we are not particularly concerned with that name.

Silicon dioxide, bearing the chemical formula SiO_2 and represented by quartz, causes a form of pneumoconiosis. When silicon dioxide combines with other elements, such as iron, aluminum, etc., it forms a *silicate.*

The lungs are equipped with a method of drainage which we call the lymphatic system. It is composed of a network of fine, connected tubes throughout the lung structure, following, in general, the course of the arteries, veins and bronchi and terminating in the glandular tissue of the mid chest. Another branch of the same system follows the outer contour of the lungs and pleura and ends in the mediastinal glands. The function of this drainage system is to rid the lungs of foreign material, whether it be bacteria, the result of infection, dust or any other foreign body which has entered the lungs.

Such a system would be of no practical value without a means to convey the extraneous material into the tubes, and the body has provided such an agency in the white cells of the blood. Each white cell, called a phagocyte or scavenger, is a mobile unit capable of ingesting foreign material and transporting it from the lungs to within the lymphatic system.

When dust enters the upper respiratory tract through the nose or mouth, it becomes immediately wetted, and, by far, the largest proportion enters the gastro-intestinal tract by the act of swallowing, and becomes inert. A portion of the dust accumulates on the mucous membranes of the nose and it is removed by the elimination of the nasal secretions and expectoration. The extremely small amount of dust that finds its way into the larynx meets considerable opposition in its downward progress from the cilia, or hair-like structures of the cells lining the bronchi. It is their particular function to retard the advance of foreign material.

Some of this dust is removed by coughing. Having evaded all of the protective agencies of the respiratory tract and arriving within the air spaces of the lungs, the dust particles are ingested by the white cells, or phagocytes, carried through the wall of the lymphatic system, and are eventually deposited within the glandular tissue. The smaller the size of the dust particles, the greater seems to be the activity of the blood cells, and conversely, the larger the particles, the more sluggish they become.

The unit of dust measurement is the micron, 1/25 thousandth of an inch. Experimentation has proven that dust greater than 10 microns in diameter is practically harmless as far as the lungs are concerned, but from dust averaging between 2 and 5 microns may be expected the maximum amount of lung pathology. Such microscopic dust is invisible to the naked eye.

The transportation and storage of dust continues until the lymphatic system has been filled to saturation. Other depositories must then be utilized. In that event, the phagocytes move out through the walls of the lymphatics and deposit their dust load in the partitions between the air spaces of the lungs. That process always occurs in both lungs and is never unilateral. It starts from the hilus area, extends outward toward the periphery and eventually may involve all lobes. The entrance of a foreign body within the lung structures stimulates the protective devices to greater action and soon the blood stream lays down cells about the dust deposits, in the endeavor to encircle the mass with a wall of thick fibrous tissue, in the hope that the progress and harmful effects might be checked or reduced to the minimum.

That protective cellular element is called connective tissue, and when found in the lungs is known as fibrosis. The formation of fibrosis is not peculiar to the presence of dust, but is present in its defensive role in practically all diseases of the lungs, whether it be tuberculosis, pneumonia, bronchitis, asthma, or the results of inflammation. It is rare indeed to find an X-ray of the lungs in which fibrosis cannot be demonstrated.

Connective tissue is non-elastic. It is quite obvious that the constant replacement of normal lung tissue by tough, non-elastic fibres, will in time produce embarrassment of respiration. It happens in tuberculosis, in asthma, in silicosis and in the absorption of other dusts.

The description of the lungs physiology in the presence of a foreign substance which has just been described is applicable to the absorption of many dusts, whether of a silicotic nature or silica-free. When silicon-dioxide is the prevailing dust, whether in the form of quartz, granite, sandstone, or flint, we become involved in a further process characteristic of silica alone. When the white cells group together and deposit their load of silicon-dioxide at a common point, the connective tissue cells encircle the mass, and we have formed the so-called silicotic tubercle. This is the first tangible evidence of silicosis. Tubercles and connective tissue may increase, which simply denotes that the process has advanced. We attempt to divide the silicotic process into three stages of development, but as there is no sharp line of demarcation between them, opinions will be as numerous as the number of persons who examine the films. It is quite suffiicent, however, for all practical purposes to simply note the presence of fibrosis

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and nodulation when examining the films.

When quartz is present, a chemical action is noted which is apparently characteristic of silicon-dioxide and not found in the presence of other types of dust, with the exception of asbestos which will be described later.

Silicon-dioxide is soluble in the weak body alkalies, and such reaction produces a toxin or poison. That poisonous chemical reagent so alters the immediate surrounding tissue that it loses its resistance and becomes incapable of protecting itself against the invasion of foreign bacteria. Pneumonia occurs not infrequently in silicotics, probably due to the frequency of the organism of that disease in the upper respiratory tract and the inability of the lung tissue to offer any defense. The disease in which we are particularly interested, however, is tuberculosis whether pre-existing or superimposed upon a silicotic process.

It is estimated that 75 per cent of all tuberculosis is acquired before the age of 15. If a person is comparatively young, say around 30 years of age and sometime during his life has contracted tuberculosis which has become non-active and walled off by the connective tissue barrier, the silicotic poison formed, exerting its influence on the outer wall and the action of the bacilli from within, often succeed in breaking through that defense. The tubercle bacilli are then liberated within the lung tissue and meeting little or no resistance spread rapidly and usually cause a fatal ending. If that protective barrier of connective tissue was sufficiently strong and tough to resist the action of the toxin, no particular harm would result.

Basing our conclusions upon the actual facts of medical and experimental knowledge to date, I think it is safe to say that the non-siliceous dusts, represented by coal, cement, lime and marble and the silicates of iron, aluminum, clay and many others, are essentially harmless when introduced within the lungs, but silicon-dioxide in the form of quartz, sandstone, granite or flint are particularly hazardous in the presence of infection.

Gardner divides the action of the dusts in the lungs into three classes: namely, the linear fibrosis caused by most inert material, the nodular type which is confined to silica and the diffuse as represented by asbestos.

Asbestos is a magnesium silicate containing about 42 per cent of silicon dioxide. Its entrance into the lung causes a diffuse fibrosis which is characteristic only of that substance. The exact cause of the reaction is not known, but one theory advanced relates to the loose combination between the silica and the magnesium resulting in enough irritation to cause areas of diffuse fibrosis. It may advance to a degree sufficient to cause incapacity and may terminate fatally without the intervention of infection.

The non-siliceous dusts and many of the silicates may produce varying amounts of fibrosis and may occlude many of the lung's air spaces. The former condition, if carried to excess, may influence the respiration to a certain extent. The lungs are composed of millions of air spaces and the obliteration of thousands would hardly be noticed. Tuberculosis, or operations upon the lungs, have wiped out entire lobes and yet produced no discomforts, so why need we become alarmed at the loss function of small areas due to dust obliteration?

It was formerly thought that the pathology within the lung created by the presence of silica was due to the mechanical action of the dust particles. The sharp edges and corners of the dust were supposed to so mutilate the tissue that it could not withstand attacks of infection. That theory could not account for the action of many of the dusts, such as aluminum oxide or diamond dust whose edges are sharper than those of quartz and yet were unable to aggravate tuberculosis into activity. That theory has been given up in favor of the chemical reactions. It was then thought that a colloid might be formed, but again that theory could not account for the variations in the reactions of dusts. At present, the chemical theory with the formation of definite poisons has been accepted. It at least gives us a fair explanation of the action of silica and the harmless dusts.

We may be obliged to change our opinions later concerning the actions of certain dusts in the light of advancing knowledge. Our present knowledge concerning the actions of some of the silicates is rather vague and not entirely satisfactory.

(Continued in the June issue)