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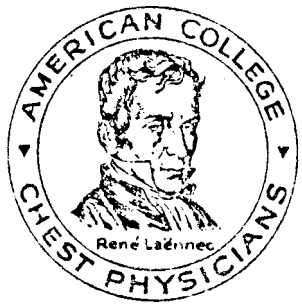

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Dust Inhalation in Relation To Pulmonary Disease*

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Everyone is inhaling some dust with almost every breath taken, but the results of this dust upon the lungs is considered part of the natural process of aging when it occurs outside of his occupation. The relationship of dust inhalation to pulmonary disease is therefore usually considered only from the point of view of industrial exposure. Observations on the effect of dusts in industry have consequently been limited largely to those pathological processes which can be clearly differentiated from pulmonary diseases of non-occupational origin. To these characteristic changes in the lungs produced by dust, the term pneumoconiosis has been applied.

Diagnostic Factors in Pneumoconioses

In order to establish the diagnosis of pneumoconiosis, two factors must be present: 1) pulmonary pathology which might have been produced by exposure to a particular dust, and 2) an occupational history of exposure to that dust. The chest physician is generally in a good position to evaluate the first factor, through the medium of signs, symptoms, x-ray inspection and laboratory tests. In determining the second factor, he is often handicapped by his paucity of knowledge of the nature of his patient's work and the materials handled. Most physicians are aware that a rock miner or stone cutter may be exposed to free silica in amounts capable of causing silicosis. They may not be aware, however, that a worker in a soap factory, which includes in its products scouring powders, may be handling large quantities of silex, which is almost pure pulverized silica. In other instances, the etiology of the pulmonary pathology may not be so readily recognized because the physician is unaware of the more obscure effects upon the lungs of a material such as talc or bagasse.

Another factor which makes it difficult to recognize the importance of an industrial dust exposure in the pathogenesis of a pulmonary disease is the close resemblance which frequently exists

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between that disease and some other pulmonary pathology of non-industrial etiology. When the x-ray picture is characteristic, as in an obvious case of nodular silicosis, the physician is apt to inquire carefully into the worker's occupation, and to enlist the aid of industrial hygienists or other experts in an effort to establish a dust etiology. When the clinical and sometimes even the pathological findings are almost identical with some non-occupational disease of the lungs, however, he is not so likely to seek far afield for an occupational exposure. It was not until the unusual incidence of workers dying from what was presumed to be Boeck's sarcoid in a plant manufacturing fluorescent lamps was noted, that the toxicity of one of the materials which they were handling was even suspected, and that the identity of chronic pulmonary granulomatosis due to beryllium was established.[1] Recognition of the dust etiology is even more difficult when the pathology is identical with that of a non-occupational disease, as for instance the primary lung carcinoma seen with increased frequency in chromate workers.[2]

Another way in which dust inhalation may affect pulmonary disease is by aggravation of a pre- or co-existing non-occupational disease. Today, the fact that silicosis predisposes a worker to tuberculosis, and renders that tuberculosis much more severe, is well established. We are just becoming aware, however, that some of the more innocuous dusts may adversely affect the course of tuberculosis and other pulmonary infections.[3] It is no longer considered good practice for an individual with arrested tuberculosis to return to work in any dusty environment. We all too frequently, however, see workers with chronic bronchitis, pulmonary emphysema or even bronchiectasis who are permitted to work in an extremely dusty atmosphere. Even the increased coughing induced by a dust which is slightly irritant to the upper respiratory passages, or necessary to clear these passages of the accumulated particles, may be sufficient to aggravate a chronic pulmonary disease.[4]

Over and above all of these specific aspects is the general effect of dust on the aging processes of the lung. I would like to quote an observation made by Dr. Edgar Mayer, in an article[5] he published a few years ago: "... respiratory infections and ... general dust exposure eventually provoke pulmonary changes. There exist only differences in degree, not in kind, between the so-called normal amount of pulmonary fibrosis so prevalent in the general population and those severe fibrotic processes which destroy the lungs of workers in industries wherein exposures exist to more concentrated and continuous effect of the same agents present in greater dilution in the atmosphere of industrial centers."

Dust Characteristics

A pre-requisite to the understanding of the effects of dust inhalation upon the lungs is a knowledge of the type of material under discussion. We have chosen to cover in this paper all forms of solid particulate matter generated in the course of industrial operations, because in so many instances their effects are similar or even identical. Fibers and fumes as well as dusts have therefore been included. Since some uncertainty exists as to the exact meaning of these terms in industrial hygiene, we shall start with a few definitions. The term dust is applied to the solid particles produced by handling, cutting, crushing, grinding or pounding organic or inorganic materials such as rock, ore, metal, coal, wood, grain, etc. Fibers are the particles similarly produced from organic or inorganic material composed of thread-like or slender elements such as cotton, wool, fur, bagasse or asbestos, and are also created in such operations as stripping, spinning and weaving. Fume is a term limited to the solid particles created by condensation from a gaseous state, generally after volatilization from molten metals, and often accompanied by a chemical reaction such as oxidation. Contrary to general usage, it does not refer to condensations to a liquid droplet form, which are called mists, or to a uniform gaseous dispersion, whether visible or invisible, which is referred to as a vapor or gas.

The common denominator of all these materials is thus the fact that they are present in the atmosphere as solid particles. An important characteristic of these particles is their size.[6] It has been shown both theoretically and experimentally that on inhalation those which are too large do not reach the lungs, but are filtered out in the upper respiratory passages. Particles which are too small, on the other hand, do not settle out, and are therefore theoretically not retained in the alveoli but are re-exhaled. The question of the innocuousness of these sub-microscopic particles has recently been open to question. It is a generally accepted principle, however, that the particles exert their greatest deleterious effect upon the lungs when they are between 0.5 and 3 microns in diameter.

The other important factor determining the reaction of a dust upon the lungs is its composition, that is, its chemical and physical characteristics.[7] Where it is a mixture of several different compounds, such as the dust created in a granite quarry, the percentage of the most active ingredient (in this instance free silica) is of the utmost importance. Even where the mixture is man-made and not a natural product, its composition has to be taken into consideration. For example, the sand used in an iron

foundry may consist of almost pure silica, but the dust released in a shake-out operation will have a much lower silica content because a large part of it comes from the iron oxide particles separated from the surface of the casting. The particle structure also appears to play a role: crystalline silica has long been considered a far more active substance than the amorphous form. Fumes from a melting operation are frequently much more reactive than the same material created as a dust by grinding or crushing. In some instances even the shape of the particles has significance, particularly when their effects are dependent to a large extent upon local mechanical irritation.

Protective Mechanisms

There are a number of mechanisms by which the lung parenchyma is protected from inhaled dust.[7] Many of the particles may be filtered out by the coarse hairs guarding the entrance to the nose, or trapped by the moist walls of the turbinates. Particles deposited on the surface of the mucous membranes of the trachea and bronchi are carried back towards the pharynx by the wave-like vibrations of the cilia, and also by the cough reflex induced by the irritation to the lining membranes.

Particles which succeed in passing these barriers and gain entrance into the alveoli are ingested by macrophages which arise from the alveolar walls and are commonly referred to as "dust cells." Some of these phagocytes, laden with dust, may pass up into the bronchioles and eventually be carried off in the sputum; the others pass through the walls and into the regional lymphatics. Some of the latter are arrested locally in the peribronchiolar tissue, but the remainder pass onward into the peribronchial and perivascular lymphatics, and are finally trapped in the lymph nodes at the root of the lung.

Types of Pathology

The type of pulmonary pathology which may be caused by the inhalation of a specific dust is determined by the manner in which this lung cleansing mechanism is disturbed, and by the type of reaction which results. Probably the best known is the fibrotic reaction produced by exposure to silica and similar materials.[8] This reaction represents a specific cellular response to the material. The particles engulfed by the dust cells stimulate the production of fibroblasts in any area where they are congregated; and these fibroblasts are eventually replaced by dense fibrous tissue. In the case of dusts other than silica, this fibrosis is usually fairly generalized; but with silica, it tends to nodular formations distributed along the lymphatics. In either case, the net effect is to make

the lungs less elastic, increase their bulk while decreasing their capacity, and decrease pulmonary circulation and aeration of the blood.

Less characteristic is the action of those dusts which act as general irritants,[9] particularly on the respiratory passages, but do not exhibit a specific fibrosing action. Included in this category are most of the organic dusts. Workers first exposed to them respond by sneezing and coughing, but usually quickly become acclimatized so that the irritant action is no longer found troublesome. Continued inhalation of these dusts, however, may result in chronic inflammatory changes in the mucous membranes with secondary hyperplasia or atrophy. Degenerative changes have also been noted, with disappearance of the ciliated columnar epithelium and its replacement by a cuboidal or squamous form.[10] The chronic cough induced by these irritants may also be responsible for emphysema and other secondary changes in the lungs.

In addition to this generalized irritant action, certain dusts may elicit a specific inflammatory response.[9] This may be of a chemical nature, as in the case of beryllium or Thomas slag, or may be infectious, due to bacteria or fungi carried in by the dust. In both instances, the pathology of the response depends upon the material producing it, and may be acute or chronic, reversible or permanently damaging. With some materials, for instance beryllium, the pathology is quite characteristic. With others, particularly those transporting micro-organisms, it may be indistinguishable from non-occupational infections of the pulmonary tract, and only a careful investigation of the patient's occupation will elicit the offending material.

Even when the dust particles exert no irritant action whatsoever, there may be slight pulmonary changes due to their mechanical effects.[11]

Pulmonary allergy is a type of response observed with certain organic dusts,[12] particularly those of plant or animal origin. The picture is one of a typical asthma, and only the occupational history differentiates it from identical reactions so commonly seen from pollens or house dusts. The distinction often can only be made by patch testing or similar procedures, which will demonstrate that the responsible antigen is present in the man' occupational rather than non-occupational environment.

Primary carcinoma of the lung has at one time or another been ascribed to a great many of the industrial dusts produced in industry. Proving or disproving the carcinogenic properties of any particular material is often an extremely difficult procedure, because the pathology in no way differs from that of a lung cancer of unknown etiology. Merely demonstrating the co-existence

of cancer and some other pulmonary disease such as silicosis, is not sufficient, since there is no reason why the incidence of malignancy in silicotics should be any lower than it is in non-silicotics. The only satisfactory proof of such carcinogenicity is the uniquivocal demonstration of an incidence of cancer in workers exposed to a particular dust which is sufficiently higher than that of a comparable group of non-exposed workers to have statistical significance. Great care must be taken in selecting the control group to insure that all extraneous factors, such as age distribution, sex and non-industrial environment, are identical. The high incidence of lung cancer in the uranium mines of Schneeberg and Joachimsthal has been known for some time.[13] More recently, an increased incidence of pulmonary malignancy has been demonstrated in workers exposed to chromate dusts,[2] although the statistical significance of the data which have been collected on this material is still being investigated. Both silica and asbestos have on many occasions been indicted as predisposing to lung cancer; but in the case of silica, it has never been demonstrated statistically, while accumulating evidence points more and more strongly to asbestos as a carcinogenic agent.[14]

Pulmonary Disease Due to Silica

Having presented the general types of pulmonary pathology which may be produced by dusts, I would like to devote the remaining time to a brief description of the specific pulmonary effects produced by certain materials. As has been previously pointed out, undoubtedly the most important of these is silica. This is the oxide of the element silicon, and is widely distributed, both free and combined, in the earth's crust. Crystalline silica occurs pure as quartz and as beach sand, and is also scattered in varying percentages throughout many forms of rock, such as granite, marble and sandstone. Dusts containing free silica can be created industrially in a multitude of ways, such as drilling and blasting operations in the course of mining and quarrying, cutting and shaping of stone, rock crushing, sand blasting or any other operations in which quartz, sand or silica-containing rock is processed. The disease[8] is characterized anatomically by generalized fibrotic changes and the development of miliary nodulation throughout the lung fields, and clinically by cough and progressive shortness of breath. Chest x-ray films show at first an exaggeration of the lung markings and lymph node enlargement, which is followed by nodulation, and finally by coalescence and conglomeration. Tuberculosis may be super-imposed at any stage, and is believed by some to be present in all areas where coalescence has taken place. As a rule, there is no fever or weight loss in

simple silicosis, and these signs may be indicative of a complicating tuberculosis. The patient tends to go downhill very rapidly with the development of an active tuberculous lesion.

Many observers have been of the opinion that silicosis could be caused only by the crystalline form of silica, but evidence is accumulating to the effect that the amorphous form as well is capable of producing pulmonary damage. Observations upon the effects of amorphous silica[15] have been made largely on workers exposed to diatomaceous earth, which is a light fluffy material formed by the accumulation of skeletons or shells of diatoms. It has a wide use in industry as filters, fillers and absorbents. In contrast to crystalline silica, it produces fibrosis of the lungs without discrete nodulation. The fibrosis is much more marked when the exposure is to calcined material, during which process microcrystals of chrystobalite may be formed. Disability appears to be less marked than with silica, and the fibrosis seems to diminish rather than enhance susceptibility to tuberculosis.

Another possible example of pulmonary damage due to a non-crystalline form of silica occurs in workers fusing bauxite in electric furnaces in the manufacture of alumina abrasives.[16] The fumes given off in this operation consist[17] of 25 to 40 per cent silicon dioxide and 40 to 60 per cent aluminum oxide, together with small amounts of iron oxide and numerous other impurities. Under the electron microscope they are revealed as fused particles varying in size from 1/100 to 1/2 micron in diameter. A high percentage of the workers exposed to these fumes have developed cough with expectoration, weight loss, anorexia, tightness in the chest, and dyspnea on exertion. X-ray inspections show a lace-like or granular increase in the lung markings, beginning at the apices. These gradually extend to involve the entire lung fields, and may be accompanied by pleural adhesions causing distortion of the thoracic contents. Spontaneous pneumothorax is a common complication. While the aluminum oxide has generally been advanced as the etiological agent responsible for this disease, it seems quite possible that this may be a response to silica fume with a particle size far lower than that generally considered injurious, especially since more than one form of silica have been shown to be damaging to the lungs, while aluminum oxide has always been considered a non-fibrosing agent.

Silica, when present only in combined form as a silicate, has usually been regarded as relatively inert. Considerable evidence, however, has been accumulating against this concept. Probably the best known of the silicates producing severe pulmonary damage is asbestos.[18] This is a mineral silicate occurring in the form of long fibers, which renders it capable of being carded, spun, and

woven into threads and cloth, or bonded with cement into building shapes. Inhalation of these fibers in the course of mining or processing asbestos has been shown to be responsible for the development of progressive pulmonary fibrosis with diffuse thickening of the alveolar walls. The outstanding symptoms are dyspnea and dry cough. Emphysema, pulmonary infections, and secondary cardio-vascular changes due to increased pulmonary resistance are the most common sequelae. Chest x-ray films show a "ground glass" haziness, rather than the reticulation and nodulation of silicosis.

Another combined silicate which has been shown to be not completely inert is talc, a hydrated magnesium silicate used extensively in its powdered form as a dusting agent. It may contain variable percentages of free silica; and when these are high, the classical picture of silicosis has been observed in workers mining, crushing, grinding or otherwise handling it. Even when it contains under 1 per cent free silica, however, as is usually the case, the dust, when inhaled, is capable of producing a fine diffuse pulmonary fibrosis similar to asbestosis.[19] It tends to be disabling, and is frequently accompanied by dyspnea, cough and fatigue. In addition, deposits of x-ray opaque material on the pleural surfaces in the form of plaques have been observed.

Still another silicate showing definite effects upon the lungs is mica, which is a double silicate of aluminum and either potassium or magnesium. This mineral, because it splits into thin sheets which are quite transparent, and have a high dielectric constant, has innumerable uses in industry, particularly in the manufacture of electrical equipment. For some time, it has been believed that the chest pathology so prevalent where it is mined was due to the free silica present in the rocks in which mica deposits occur. Even among workers exposed solely to pure mica dust in grinding operations, however, there has been observed[20] a high incidence of increased pulmonary fibrosis, with cough and dyspnea. The signs and symptoms are the same as those occurring in silicosis, but less severe. X-ray films show a fine granulation of uneven density with coalescence of the lesions in more advanced cases.

In all these exposures, the common denominator has been silica, whether free or combined. Recently, however, carborundum, which is an almost pure silicon carbide containing less than 1 per cent free silica, has been suspected of being an agent capable of causing pulmonary changes identical with silicosis.[21] This has been explained theoretically by the possible oxidation of the material in the lung. This may be the explanation for the occasional occurrence of silicosis among workers using carborundum grinding

wheels, which have been considered a completely "safe" substitute for the far more hazardous sandstone wheels.

Pulmonary Diseases of Non-Silicotic Origin

The pulmonary changes seen in coal miners has long been attributed to the free silica content of the shale through which they had to dig to get at the coal. Recent large scale studies in Wales have indicated,[22] however, that exposure to very heavy concentrations of coal dust with a negligible percentage of free silica, such as occurs among the coal trimmers on the docks, leads to a form of pneumoconiosis differing from the anthraco-silicosis of miners. There is a massive fibrosis of the lungs with nodular deposits of anthracotic pigment scattered throughout. The principal symptoms are due to the marked secondary emphysema, and the cardiac changes produced by pulmonary hypertension. Susceptibility to tuberculosis is increased. The x-ray picture is one of reticulation rather than nodulation, because of the lack of opacity of the nodules.

Specific pulmonary pathology has also been shown to be produced by exposure to fumes and dusts of certain of the metals, notably beryllium and cadmium. Both these substances actually are systemic poisons, but since industrial exposure to them occurs primarily by inhalation, the principal pathology is usually present in the lungs.

Beryllium is responsible for two types of respiratory disease.[23] One is an acute inflammatory reaction varying from a mild pharyngitis or bronchitis to a massive pulmonary edema, which occurs principally among workers exposed to soluble salts in the beryllium refining industry. The other is a chronic pulmonary granulomatosis developing after a delay of several months to years, which occurs primarily among workers exposed to dusts and fumes of the metal, oxide, and certain complex silicates (fluorescent compounds). It is characterized by a nodular fibrosis giving a characteristic x-ray picture, and a diffuse thickening of the alveolar walls. The outstanding symptoms are dyspnea, fatigue, anorexia, severe weight loss and intractable cough. The mortality is high, and some degree of permanent disability remains among nearly all those who survive. Interrelations between the acute and chronic forms, and the occurrence of an intermediate subacute type, indicate that they are probably all widely different manifestations of the same disease process.

The pulmonary pathology due to cadmium[24] is generally encountered when workers unwittingly create cadmium fumes without adequate protection: for example, in flame cutting or welding

cadmium plated iron. It first causes a cough and dryness of the throat, which then progresses to severe constricting chest pain and dyspnea, dizziness, chills and prostration. The pathological picture is that of pulmonary congestion and edema, with interstitial pneumonitis, hemorrhage and cellular infiltration.

The term siderosis[25] has been applied to a benign pneumoconiosis due to exposure to dust and fumes of iron and its oxides, usually seen among hematite miners, welders and other iron workers. Pathologically, siderosis consists of a deposition of phagocytized iron particles distributed in nodular fashion along the lymphatics. Outside of the mechanical effects of excessive pulmonary accumulation previously discussed, its importance lies chiefly in the necessity of differentiating it from silicosis, with which it had frequently been confused in the past, particularly among foundry workers. The nodules in siderosis are more discrete and sharply defined on x-ray, and there is no tendency to confluent nodulation or hilar enlargement.

Organic Dust Diseases

A few materials of organic origin have been found to be responsible for specific types of pulmonary pathology. Notable among these is bagasse,[26] which is the residue of sugar cane after extraction. This material has been extensively used in the manufacture of insulating board and other building materials. It shreds readily when handled dry, with the production of a fibrous dust. A high percentage of workers exposed to these fibers have developed, after about two months, an acute bronchiolitis with high fever, extreme dyspnea, productive cough, weakness and weight loss. X-rays show scattered miliary shadows throughout the lung fields which may go on to a confluent pneumonia. The disease usually clears up, but may be fatal or result in chronic fibrosis or bronchiectasis.

Both acute and chronic pulmonary diseases have been reported among cotton workers. The former[27] consists of an acute febrile illness with productive cough and dyspnea, resembling an acute bronchitis. It usually resolves completely, but may occasionally result in chronic bronchitis. It is believed to be due to the dust of the fungi or bacteria which grow on damp, unsterilized cotton. The chronic disease,[28] known as byssinosis, consists of a progressive fibrosis, usually associated with emphysema and bronchiectasis, which develops after many years of inhalation of cotton fibers. It is not yet known whether it is a specific fibrotic response, an allergic reaction, or the end result of repeated subclinical acute infections.

SUMMARY

There are a number of types of pulmonary pathology which can be caused by the inhalation of excessive quantities of particulate matter, varying from specific fibrotic reactions with a characteristic picture to an increased incidence of a form of pathology usually considered non-industrial in origin. Size, composition and structure of the particles are all factors determining the nature of the response. Pathological reactions generally result when the capacity of the filtering and phagocytizing mechanisms of the respiratory tract are exceeded. These reactions may be fibrotic, inflammatory, degenerative, allergic, carcinogenic or merely mechanical impairment of function.

The best known cause of pneumoconiosis is free crystalline silica, but amorphous silica, silicates, and even silicon carbide have been shown to exert deleterious effects. Coal dust, certain metals, and a few organic compounds, have also been indicated as the cause of specific lung pathology. A careful evaluation of both the clinical picture and the occupational history is necessary in every case of chronic pulmonary disease to rule out a possible etiological or aggravating factor in the patient's working environment, and to ensure an optimum prognosis by eliminating all future exposure to any harmful atmospheric agents found.

RESUMEN

La inhalación de cantidades excesivas de partículas de materia puede causar ciertos tipos de alteraciones pulmonares patológicas, que varían de reacciones fibrosas específicas, con un cuadro característico, a una forma patológica, de incidencia creciente, que generalmente no se considera ser de origen industrial. El tamaño, la composición y la estructura de las partículas son factores que determinan la naturaleza de la reacción. Generalmente resultan reacciones patológicas cuando se excede la capacidad de los mecanismos de filtración y fagocitosis del aparato respiratorio. Estas reacciones pueden ser fibrosas, inflamatorias, de degeneración, alérgicas, carcinógenas o, solamente, impedimentos mecánicos de la función.

La causa más conocida de la neumoconiosis es la sílice cristalina libre, pero se ha demostrado que la sílice amorfa, los silicatos y aún el carburo de silicio pueden causar efectos nocivos. Se ha sospechado que el polvo de carbón, ciertos metales y algunos compuestos orgánicos también causan cambios patológicos específicos del pulmón. Es necesario avaluar cuidadosamente tanto el cuadro clínico como los datos de las ocupaciones en todo caso de neumopatía crónica a fin de determinar si existe un posible factor etio-

lógico o agravante en el ambiente de trabajo del paciente, y para asegurar el mejor pronóstico posible mediante la eliminación de la futura exposición a cualquier agente atmosférico nocivo que se haya descubierto.

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