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Foreword

THE TRANSACTIONS of the Twenty-Fourth Annual Safety Congress, National Safety Council, 1935, are published in two volumes. This volume, first, contains the general sessions, the special subject sessions, and the ustrial Section sessions. It is supplemented by a smaller volume containing sessions of the Street and Highway Traffic Section, the Child Education tion and Home Safety.

All industrial members of the Council automatically receive the large volume. e smaller is sent to members believed to be chiefly interested in the sessions covers. It may, however, also be secured by other Council members upon uest.

Many members have found it worth while to distribute copies of these nsactions to supervisors, foremen and others in an administrative or supery position who may make practical use of them for reference purposes. r the benefit of those who desire extra copies the following quantity prices are oted: One to ten copies of the large volume, $1.50 each; eleven copies and r, $1.25 each; copies of the smaller volume, 50c each.

N ACCORDANCE with the plan of publication followed for the past several years the Transactions are published again this year as a condensed record the proceedings at the National Congress. The papers of each session or sion of the Congress have been edited carefully to eliminate extraneous matand to abbreviate the less essential portions because of space limitations. In er words, the material herein presented is not a verbatim report of all the eches presented at the Congress, but rather an abridged version, made as mpact as possible, yet without any injury to the technical aspects of any lress. The original manuscripts are on file in the library of the National iety Council, where they are available for additional reference. In several es, where illustrations were sent by the speakers to the Council, these also on file.

The National Safety Council at its Congresses seeks to eliminate from discsion matters which are not pertinent to the aim of the Congress or which are ntrary to Council policies, but it cannot accept responsibility for the views pressed either in the papers presented or in the discussions based upon the pers.

NATIONAL SAFETY COUNCIL, INC.

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under controlled and uncontrolled working conditions. It is apparent that where some measure of control is practiced by such methods as segregation or local exhaust ventilation, a material reduction in the exposure to mercury has been effected. It is our belief that in the case of the blowers' exposure, a reduction may be effected by mechanical enclosure and local exhaust ventilation, and that the shippers' exposure to mercury vapor may be lessened by a general system of ventilation sufficient to change the air in the store room frequently enough to bring the mercury concentration to a lower level. Unfortunately, in the present investigation, it has been impossible to find a plant in which an attempt has been made to reduce the exposure for these two occupations.

In some dusty occupations the methods of controlling dust have not been developed. In fact, operations such as removing the cores from very large foundry castings, sand-blasting, handling of used storage battery plates, paint chipping, and cadmium oxide manufacture appear to offer no practical means of adequately controlling the dust generated. In such cases, it is therefore necessary to furnish the worker with personal respiratory protection devices to prevent his exposure to the harmful effects of the dusts present. These devices consist of various types of respirators, masks, and helmets.

It is important to hold in mind the limited use of personal protection devices. Because a worker cannot with comfort wear a mask or helmet continuously, such devices must be employed intermittently. Their use is generally extended to those operations where all other methods have failed or supplementary to them, as in storage battery repair where the exposure to small amounts of lead breathed is known to be detrimental to health.

It should be pointed out that the U. S. Bureau of Mines is equipped to conduct approval tests of respirators used for protection against various dusts and fumes (Schedule 21). These tests are conducted against the dust for which the device is to be used and are rated, not on an efficiency basis, but on the quantity of dust which actually passes the respirator. The results of a study of masks or helmets of the positive pressure type, made during the sandblast investigation conducted several years ago by the Public Health Service in cooperation with the National Safety Council,[4] showed that the only practical safeguard to the worker inside the sand-blast room was to provide him with a mask or helmet of the positive pressure type. In studying the efficiency of such devices it was found that a relationship existed between the amount of air supplied to the helmet and the concentration of dust inside the helmet during blasting. To determine the optimum air volume to be supplied to such protective devices, it was necessary to obtain dust samples from inside the helmet while varying the air volume, at the same time maintaining the dust concentration in the sandblast room (outside the helmet) constant. The positive supply of 6 cu. ft. of dust-free air per minute will protect a worker under the operating conditions now in practice in sand-blast rooms. The ultimate criterion of protection, however, is the result of dust determinations of the air within the helmet, that is, the air actually breathed by the worker and not the volume of air supplied.

Too much emphasis cannot be stressed on the necessity of maintaining in good order the personal respiratory devices for the protection of the worker against various toxic dusts. Maintenance of exhaust ventilation systems, and other types of protective equipment, should be a rule in industry rather than an exception. Too often the term "good housekeeping" has been interpreted as signifying only the periodic removal of dust collected on floors, rafters, etc. Although such practice contributes to the general state of cleanliness of a workroom and should always be in force, the time has surely come when serious attention should be given to the installation and rigid maintenance of all types of dust control devices. In every plant there should be some responsible individual charged with the periodic inspection of all workrooms as to sanitation, ventilation, and maintenance of all dust removal and other protective devices. Perhaps the best criterion of the effectiveness of these devices is the periodic determination of the dust content of the air at the workers' breathing zone. Only by constant vigilance and an approach to the problem as outlined in this paper may one hope to make progress in the control of the dust hazard in industry.

Often the benefits of even a most extensive program of dust control are not immediately realized. This is especially true in dealing with fibrosis-producing dusts in plants where some of the workmen have already inhaled sufficient quantities to

cause disability. However, in dealing with such dusts as lead, cadmium, and mercury compounds, control measures may produce salubrious results in a relatively brief period. It is difficult, because of lack of sufficient reliable data, to indicate here the economic benefits resulting from a preventive program of dust control. It has been estimated by Dean K. Brundage, statistician of this office, that the minimum expectancy in savings to employer and employee from an indicated reduction of the accident rate and of the time lost on account of illness (or an equivalent reduction in mortality), demonstrated as attainable, is $20,000 per year per 1,000 employees. And this estimate is for plants whose accident rate is considerably below the average, in which there are no occupational health hazards. In plants where hazards are known to exist the savings should be far in excess of this conservative estimate. When one realizes that in this country there are approximately 15 millions of workers engaged in manufacturing, mechanical, and mineral industries, then it is evident that the magnitude of the problem has not been overemphasized.

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Silicosis and Silico-Tuberculosis
Medical Problems of an Important Industrial Disease

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A hidden element of the cost of production is industrial disease. Industry has reached a stage beyond the concern only of wages and hours. Far more important is conservation of man power by preventive medicine and improved engineering. Disease preventive measures are not a cost, but, in the long run, a great economic saving.

Incidence. In the United States it has been computed that there are from 800,000 to a 1,000,000 people employed in occupations where a silicosis hazard exists. In New York City there are about 65,000 such employees. In a representative group of granite workers in Massachusetts silicosis alone was present in about 15 per cent and silicosis complicated with tuberculosis in almost 8 per cent. Tuberculosis was the cause of death in over one-third of the granite workers which is four times the incidence for males of 20 years and over in this country. In foundry men studied in Massachusetts, silicosis was less frequent (about 9 per cent) and less advanced in degree than in granite workers. The duration of exposure in foundry workers with pneumonoconiosis has averaged many more years than that required to produce silicosis in an industry such as gold mining. The tuberculosis hazard in foundries is nearly as great as that reported for some of the other dusty trades, but the figures are much lower than those of miners of gold, silver, copper and lead, among whom the mortality from tuberculosis is 8 to 18 times the general expectancy. Death rates for all non-tuberculous infections have been reported higher among workers in siliceous dusts than in the general population. It is suggested that the worker in silica succumbs more often to acute pulmonary infections rather than surviving the chronic fibrosis.

Silicosis is defined as a pathologic condition of the lungs due to the inhalation of silica, whether free or combined in such a state as to be capable of setting up its characteristic pathogenic effects. The principal factors that determine the incidence of silicosis are (1) the percentage of free silica in the inhaled dust; (2) the concentration of silica particles less than 10 microns in diameter in the atmosphere; (3) the duration of exposure to the dust, and (4) the susceptibility of the individual exposed as modified by age, complicating infections, etc. The occupational disease resulting from such inhalation has been defined as "morbid results of occupational activity traceable to specific causes or labor conditions and followed by more or less extended incapacity for work."

Metabolism of Silica. Significant amounts of silica are present in all body tissues and fluids. It enters the body through the digestive tract and the lungs. Most of that entering the stomach is eliminated in the stools, but a fairly large amount is absorbed into the blood as shown by the constant excretion of silica in the urine. All vegetable foods contain silicon especially the hulls of grains, hay and straw. The low silica content of the liver, spleen and kidneys indicates the little retention of the absorbed silica in the body. Silica content of the urine of animals may be influenced at will by diet. The body possesses a very efficient mechanism for the disposal of silica because of the low kidney threshhold. Silica entering the lungs in particulate form is expectorated in part with its enveloping cells, but most of it is carried into the pulmonary lymph channels. Many such particles reach the lymph nodes and even the spleen by way of the blood. The finest particles however, may be dissolved in alkaline body fluids and carried away in solution, to be excreted in the urine. There may be a constant drainage of silica from the lung through the inhalation of extremely fine particles of silica in dusty atmospheres, so small that they are not seen under the microscope.

Attempts to influence the absorption of silica from the lungs by administration of alkali have been inconclusive. Elimination of silica by way of the sputum from patients having deposits of silica in their lungs appears to be higher than those having no history of exposure to dust. Only small amounts of silica are in the blood and this level is little different in normals than in silicotics.

Pathology. This disease is essentially a fibrosis of the lungs developing especially in such industries as hard-rock metal mining, granite cutting, metal grinding and sand-blasting. The pathological changes are believed to result from two causes, a blocking of the lung lymphatics by mononuclear cells laden with dust in addition to the action of colloidal silica, the exact nature of which is in doubt. The small particles under 10 microns are the only ones capable of penetrating the lung tissue.

Although silica plays the dominant role in the production of silicosis, the admixture of other dusts tends to modify the pathological changes in the lungs so that these resemble then those of other forms of dust inhalation, and the modification bears some relation to the percentage of free silica in the mixture. Silicates, as in asbestos, produce a definite change in the lung, as well as other dusts such as marble, coal, etc. Such changes are represented by a fibrosis brought about because relatively insoluble minute particles of minerals in sufficient concentration have been brought by the activity of phagocytic cells into intimate contact with the pulmonary connective tissue. This fibrosis is a diffuse cellular one that occurs in the walls of the smaller bronchi and of all their finer divisions and extends to involve the supporting connective tissue of the adjacent blood vessels and to some extent also the walls of adjacent air spaces. However, when the great majority of the inhaled particles are composed of or contain silica, there develops, in addition, a specific and localized type of fibrosis called the silicotic nodule—an orderly whorled arrangement of cells and fibres, and with sharp definition from the adjacent parenchyma. Many dusts create a generalized fibrosis but only one, namely one combined with silicon dioxide produces the special fibrosis of silicosis. Sericite, known as white mica, which is a hydrated silicate of aluminum and potassium, has not produced in animal experiments the silicotic nodule, but instead generalized fibrosis resulted.

Dusts must be differentiated into those which are *chemically active* and those which are inert when inhaled into the respiratory tract. Silica is a chemically active dust which must necessarily be *soluble* to a degree in the body fluids and its activity depends on its solubility. This activity which is manifested in the areas where dust particles are carried along the lymph stream by phagocytes, causes lesions of two

types, "toxic" and "sclerotic" both of which have been reproduced experimentally. Toxic lesions depend upon local necrosis and slow death and appear to favor the growth of tubercle bacilli; the sclerotic lesions produce the nodular fibrosis. The *inert dusts* are insoluble in body fluids and cannot exert chemical action in the lung tissue, but if they accumulate to a marked extent their effect is mechanical which may lead to a certain amount of diffuse fibrosis around the dust deposits. Certain dusts, such as carbon, may have physical effects, they may adsorb toxic substances and it has been suggested that on this basis there is a relatively lower incidence of active clinical tuberculosis in silico-anthracotics than in silicotic lungs.

Other dusts may even protect, as in the case of certain clays, gypsum and aluminum oxide. Pure pneumonoconiosis may be only a laboratory disease, as the pulmonary fibrosis of workers in dusty trades probably results from the combined action of dust and infection, whether it be tuberculous or not.

In silicosis it is not the mineral particles that are breathed in during life that record the cause of disease, but the particles that have been dissolved. Risk to work, efficiency and health may be greatly accentuated through the inhalation of finely divided particles even of a chemically inactive dust when there has occurred a lymph stasis which is known to follow exposure to silica. This lymphatic blockage leads to retention and accumulation of the inert dust. In soft coal miners who get what is called "miner's asthma" we have this accumulation of carbon. Such patients are very liable to have a high incidence of bronchitis with mechanical and physical changes due to retention of anthracotic dust. Furthermore, unequivocal cases of silicosis, with or without tuberculosis, can no longer be doubted.

Tuberculosis. Most apical tuberculosis is acquired before the age of 25 and so silicosis at the age of 30 makes a previous tuberculosis more serious. Most workers with tuberculosis going into the mines before the age of 30 die of tuberculosis by the age of 45. The silicotic develops a sputum that contains tubercle bacilli late in life and the children who are contacts with tubercular silicotics develop very little clinical tuberculosis. It is impossible to say definitely that tuberculosis engrafts itself upon the silicotic or vice versa, for we see apical tuberculosis in silicosis that spreads downward, while other silicotics show only the tuberculosis in the lower lobes. It has been found that most silicotics die of tuberculosis.

The diagnosis of silicosis is made primarily on two findings, the proper history of occupational exposure to siliceous dust and the presence of abnormal shadows on the pulmonary X-ray. The physical examination and the patient's symptoms are of less value. There are other diagnostic aids such as the finding of large quantities of silica particles in the sputum, quantitative determinations of silica in urine, and at post mortem, chemical analyses of the lung ash supplemented by petrographic examination, roentgen-ray spectrum analysis and special incinerating studies of lung tissue. We must at times rely on the pathologist to determine the amount and distribution of fibrosis due to silica and from microscopic studies give an opinion on the importance of this fibrosis as the ultimate cause of disease and death.

Lung fibrosis may be present without any silica. Silica may be present in lung tissue or the pulmonary lymph channels without associated fibrosis. Finely divided siliceous particles from lung tissue may contain innocuous silicate which cannot be distinguished from harmful silica particles. Hydrated silica which is not doubly refractive cannot be demonstrated with prisms. Therefore in the pathological section the presence of siliceous fibrosis can be suspected but cannot be specifically identified with the siliceous material that it may contain. Accordingly the microincineration method of Irwin with hydrochloric acid is now included in the microscopic examination of any lung as a means toward a surer diagnosis.

Diagnostic difficulties in clinical medicine may be more obvious if we examine first the occupational history. Quartz grinders working under conditions of massive exposure may develop silicosis in acute form even in a period of months, whereas in other occupations it may take 25 years. Workers in the same industry, indeed in the same room, experience different degrees of exposure dependent upon perhaps the dust-filtering capacity of the nose and functional condition of the lung as determined by constitutional characteristics and antecedent disease. The size and colloidal structure of the particles of silica, as well as the dosage and total amount of silica, will influence the rate of development of the disease. Apparently particles that are larger than 10 microns are not phagocytosed in the lung. So a definite

history of exposure must be established and in general hospitals where patients are migratory, conditions under which they worked are very vaguely described and there are not available data on dust counts and silica concentration, so that the history is often misleading.

As to symptoms, patients can perform strenuous labor despite extensive disease and the symptoms of dyspnea and cough are common to many diseases. Fever is absent unless infection occurs, but most important is the great disproportion between a patient's complaints and what is seen on the X-ray, the latter showing extensive abnormal shadows in comparison with the symptoms.

On physical examination, extensive disease may be present and few abnormal physical signs. The physical signs are merely those of a general pulmonary fibrosis with emphysema, such as restriction of costal and diaphragmatic movement, diminution of or intensified breath sounds and a hyperresonant note. Rales are usually absent unless infection is present.

As to the *X-ray*, there are 3 essential types of shadows described, linear strands, small discrete shadows, and homogeneous shadows of varying sizes; these correspond to the fibrous strands, silicotic nodules, and the conglomerate masses of fibrosis. The nodular shadows are usually characteristically around the hilum, or conglomerate nodular shadows of bat-wing appearance extend into both upper lung fields, or nodular shadows may be distributed in the upper two-thirds of the lung fields, perhaps more pronounced on the right side, with the lower third kept clear by emphysema. With infection present, the shadows are less sharp or the linear strands interconnect or fuse. Large conglomerate shadows appearing out from the hilum leaving the periphery of the lung clear throughout because of emphysema. The distribution of some of these lesions may be determined by the posture assumed by the workers, and infection may likewise determine an ultimate atypical distribution. Many variations from these patterns are seen in the X-ray, especially under excessive exposure or when other dusts are inhaled, or in the presence of infection. It is probable that the main source of the diagnostic difficulties is caused by the emphysema which muffles the physical signs and is responsible with emphysema, such as restriction of costal and diaphragmatic movement, diminution in great part for the absence of symptoms. It may blot out, even on X-ray, the silicotic lesions of fine size. Examples of such difficulties in diagnosis as encountered by us are the following:

Case I—Mr. T. A forty-five year old man entered the New York hospital complaining of mild cough and expectoration of four months' duration. He appeared acutely ill, his fever was 103 degrees and respiration 28. Rales were elicited over the upper half of both sides of the chest. Examination of his eye grounds revealed bilateral retinal tubercles; his sputum contained numerous acid-fast organisms. The X-ray revealed fine mottled shadows distributed throughout both lung fields and a small cavity at the left apex. A diagnosis of miliary tuberculosis was made. However, after one weeks' stay in the hospital, his temperature and pulse returned to normal and during the following month he gained 18 pounds. The signs in his chest now became confined to the left apex. In view of his unusual progress the diagnosis of miliary tuberculosis was doubted. His occupational history revealed that up until three years before entrance to the hospital he had worked for 20 years polishing leather on a sandpaper wheel. There were numerous machines in the work room and no precautions were observed to clear the very dusty air. His sputum was examined through the kindness of Dr. Barke of Raybrook, N. Y. who found it laden with numerous doubly refractile silica particles. He expressed the opinion that this was consistent with silicosis for he had found such numerous particles only in cases of silicosis. The patient subsequently died of a tuberculous meningitis. Retinal tubercles were demonstrated on microscopic section. The pathologist's report was miliary tuberculosis. Ashing of the lung showed increased silica content, consistent with undue exposure to dust (more than 2 mgm. silica per gram dried tissue).

Case II—Mr. R., aged 54, entered the New York hospital complaining of hemoptyses, dyspnea and chest pain. On physical examination there were rales and dullness over the upper third of the right chest anteriorly. He ran a low grade fever but was robust and felt quite well. The chest X-ray disclosed enlarged hilum shadows, particularly on the right, and diffuse mottled discrete shadows

throughout both lung fields with a circumscribed density near the right apex. He gave a history of having worked for twenty-four years as a cutter and sizer of asbestos-containing paper box boards. The rooms were in a continuous cloud of dust. Examination of the dust revealed 5 per cent silica content. The hemoptysis, we felt, was to be explained on the basis of infection or neoplasm, but we did not know whether we were dealing with one of those processes alone or an associated silicosis or asbestosis. A small nodule in the neck was subsequently removed and showed carcinoma. We are inclined to believe that this does not explain the whole process, as the man is still alive and certainly, from the X-ray standpoint, we cannot say that there is no silicosis.

Case III—Mr. C. A dish-washer, aged 46, entered Bellevue hospital because of cough and expectoration, associated with dyspnea. There was some dullness and rales at the right base posteriorly. Chest X-ray revealed a homogeneous shadow at the right base. His sputum contained no tubercle bacilli and lipiodol study revealed no abnormalities. Bronchoscopic examination disclosed a bleeding mass in right main bronchus. Symptoms and disease progressed during the following four years. Discrete mottled shadows first appeared in the upper right lung field and the shadow at the right base cleared somewhat. Three years later extensive abnormal shadows were present throughout both lung fields, particularly on the right. Diagnosis of chronic pneumonia of unknown etiology was made. At no time was a diagnosis of silicosis entertained, because as far as could be determined he had no history of exposure. Furthermore if there were silicosis it behaved very atypically having the lesion confined practically to the right base at the beginning and then spreading to the left lung. Autopsy however, revealed a typical silicosis associated with a small degree of tuberculosis. A picture of the lung showed how much more extensive the silicotic process was in the right lung. There was stenosis of the right middle lobe bronchus with bronchiectasis in this lobe. Chronic infection in the lung field probably accounted for the unusual localization. This case illustrates how necessary it is to have the history of dust exposure for without it here we are unable to even suggest a diagnosis.

Case IV—Mr. C., aged 45, complained of cough and dyspnea with slight expectoration and gave a history of having worked for many years repairing tires, using talc powder. It was difficult to obtain accurate details as to the possibility of silica exposure except that the rooms were filled with clouds of dust. Rales were present at both apices and a few tubercle bacilli were found in the sputum. Discrete and stringy shadows were disseminated throughout both lung fields. We know from the positive sputum that tuberculosis is present. The patient has, however, been well for a period of two years. The doubtful history of exposure together with the numerous discrete nodular shadows in his lung, which are consistent with silicosis, makes this patient a problem. Is this tuberculosis alone or is this tuberculosis with silicosis? The patient is still living a year later and working.

Conclusion. Cases such as these are exceptional, but their existence must always be borne in mind. If occupational history is inadequate and X-ray, clinical and laboratory studies prove misleading, the diagnosis may present great difficulty. However, a comprehensive study of all possible data usually clears up the problem with reasonable certainty.

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