FILE NAME: Chrysler (CHRY)

DATE: 1937

DOC#: CHRY164

DOCUMENT DESCRIPTION: Journal Article - Industrial Dusts and Lung Diseases

J. Mich. State Med Soc 36: 546-549 (1937)

The following instances were considered as attributable to the operation:

Febrile puerperium	42
Puerperal sepsis	6
Wound infection	14
Pneumonia	2
Pulmonary embolism	1
Thrombophlebitis	4

There is a 17.8 per cent maternal morbidity directly attributable to the operation, other than the usual postoperative febrile reaction.

Summary

As a result of this analysis of 158 abdominal cesarean sections the following points are considered significant:

1. The panoramic study includes the earliest case recorded in the University of Michigan Hospital.

2. The incidence of cesarean section has increased from 1.6 per cent prior to 1924 to 2.5 per cent since that time. This increase may be accounted for by the fact that certain cases of placenta previa and abruptio, as well as court order for sterilization of feeble-minded pregnant women are now accepted as justifiable indications for operation in this clinic.

3. The maximum number of sections in any one patient in this series was four.

4. The most frequent indication for the operation was cephalopelvic disproportion.

5. A combination of indications, which singly do not necessitate operation, may force the issue for operation.

6. Since 1931 our preference has been for the low type of operation.

7. In the entire group of 158 cases, maternal mortality was about the same in the low and classical types of operation.

8. Pre-operative physical condition of the patient is important.

9. Integrity of membranes is important.

10. Elective section carries the best prognosis.

11. Sepsis is the most frequent cause of death following cesarean section.

12. Corrected fetal mortality was 8.8 per cent (absolute—12.6 per cent).

13. Corrected maternal mortality for the 35 year period was 5.06 per cent (absolute —6.33 per cent).

14. There have been no maternal deaths following cesarean section in the last four and one-half years.

INDUSTRIAL DUSTS AND LUNG DISEASES*

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The lungs, as described in Chinese medicine, "have a fishy smell and a hot taste. They store the energy and are the seat of sorrow. The lungs produce the skin and the hair, form the kidneys, and control the heart. They have the nose as their opening, convert the fluids into nasal secretions, supply the skin, and nourish the fine hair. The lungs are attached to the third vertebra and hang down in eight lobes. They are gray in color and are pierced by eighty small holes."

Whatever may be the fallacies of these statements, many an American industrialist and many a worker willingly will accept the Chinese doctrine that "lungs are the seat of sorrow."

Every work-day in this country approximately 500,000 employees go about their duties exposed to silica dust in harmful quantities. This number is distributed over at least sixty different occupations, including, among others, foundry workers, core makers, granite cutters, rock drillers, sand pulverizers, sand blasters, and vitreous enamelers. In addition, at least 2,000,000 other workers are employed in dusty trades in which no silica exists or wherein the quantity of silica is below that capable of producing that form of pneumoconiosis known as silicosis. These workers may be found in coal mines, limestone quarries, carbon black factories, glass plants, iron mines, coke ovens, gypsum mines, seed and grain mills, pigment factories, zinc smelters, lead refineries, and on and on through scores of other work places. As one becomes familiar with the extent of dusty trades, it becomes amazing, not that there is so much dusty lung disease, but that so little arises.

All of the many scores of industrial dusts entering the lungs in harmful quantities may be divided into four classes for the purposes of this present discussion:

*Read before the Section on Radiology, Michigan State Medical Society, Detroit, September 24, 1936.

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1. Dusts that enter the body by way of the lungs without producing their chief effects upon the lungs. By way of examples of this type may be cited lead, arsenic, mercury, and manganese.
2. Dusts which, after coming in contact with the respiratory tract, including the lungs, lead to allergic manifestations. Already this has been covered by Dr. Towey, and need not be discussed in any detail here. However, by way of casual examples, may be cited pyrethrum, leather, cotton, fur, and many dyes, of which paraphenylendiamine is the best known.
3. Dusts which enter the lungs and become the causative agents of immediate inflammatory reactions. This class embraces such agents as soda ash, lime, acid and alkali dusts, and obvious others, such as resins in dust form.
4. Dusts which, after entering the lungs, set up reactions that eventuate in the production of fibrosis,—the so-called fibro-genetic dusts.

For lack of time it is necessary to limit further discussion to this last-mentioned group. Also, for the same reason, only the briefest mention may be made of any aspect of the action of these dusts or the conditions in industry under which lung injury arises.

Of all the mineral dusts leading to lung injury, silica and asbestos only stand out as sources of direful lung disease characterized by extensive fibrosis. Associated with these dusts are the respective terms "silicosis" and "asbestosis." Both are forms of pneumoconiosis, which term, from its very derivation, is applicable to any degree of abnormality of the lungs produced by any dusts. However, many pneumoconioses are not associated with unusual quantities and distributions of fibrotic tissue.

Silicosis is a specific disease, widely defined as, "a disease due to breathing air containing silica (SiO_2), characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present), and by characteristic x-ray findings." Manifestly, error attends the use of such terminology as "coal dust silicosis," "lamp black silicosis," "lime dust silicosis," etc. When silicosis appears in the coal miner, which is fairly common, it must be attributed to the silica content of the overburden of the coal seams. While this is true, the coal dust itself plays an important rôle in accelerating the appearance of the silicosis. Anthracosilicosis is a characteristic occupational disease of anthracite coal miners. Similarly when silicosis appears in a cement worker it must be attributed to the silica content of the ingredients entering into cement. While this is true, it is granted that cement workers may present increased quantities of fibrosis, quite apart from the typical features of silicosis and attributable to dusts other than SiO_2.

Mineral dusts other than the two mentioned are of comparative insignificance as sources of disabling industrial lung disease. If the damaging properties of silica dusts be arbitrarily rated at 100, then, with the exception of asbestos, most other dusts may be represented on this same scale by 5 or 10. Among these 5 per cent or 10 per cent dusts may be found aluminum, lamp black, asphaltum, whiting, titanium oxides, gypsum, coke, and coal. On the other hand, some silicates, as exemplified chiefly by clays, may be rated somewhat higher and perhaps in the order of 15 per cent to 20 per cent, possibly because of the content of free silica admixed with the silicates, and possibly from actions within their own rights. Possibly, glass dust should be placed in this category rather than elsewhere.

From this concept that most dusts are only about 5 per cent as dangerous under industrial conditions as is quartz silica, it becomes advisable to abandon such outmoded terms as siderosis, aluminosis, chalcosis, byssinosis, anthracosis, as descriptive terms of significant pneumoconiotic disease.

Asbestosis as one of two outstanding industrial fibrogenetic lung diseases must here be dismissed with the cursory statement that its clinical course, its x-ray manifestations, and its association with tuberculosis are all quite dissimilar to those characteristic of silicosis.

The period of exposure requisite to the production of a demonstrable silicosis varies from a few months, such as two or three, to a lifetime of seventy years. Rarely, when the exposure is to essentially pure

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quartz silica, in minute particle size range, an acute silicosis may be brought about which is atypical as to its clinical course, and particularly as to its appearance on the roentgen film. Such were some of the cases in the West Virginia holocaust; such also characterized some of the cases in the New Jersey outbreak occurring five or six years ago. In the causation of the more nearly typical form of silicosis, the period of exposure is likely to lie between seven and twenty-five years. The time element is greatly influenced by the percentage of free silica in any dust, the size of the particles inhaled, the hours of labor, the arduousness of the task, the age at which the worker entered the trade, the size and shape of the upper respiratory tract, particularly in respect to the vibrissæ, and, to a slight extent, by the race. Every one of these points might be made the basis of extended discussion. However, only one statement may be made with reference to each.

Some other minerals associated with the silica dust appear to retard the rate at which silica exerts its action, among others, iron oxides, gypsum, some clays, and possibly alkalis, although the last is disputed.

The size of harmful quartz particles is believed regularly to lie below 10 microns, and probably below 5. Obviously, the size must be such as to pass through the smallest entryway into the lung sacs. Also, the size must be such as to permit phagocytosis, although this last statement may be open to question.

Long hours of labor, particularly when associated with hard physical tasks, may increase both the depth and rate of breathing and remarkably increase the amount of dust entering the lung. Due to fatigue, the last hour of any work day may at times lead to the inhalation of as much dust as all the preceding hours during that period of work.

The probability of acquiring silicosis increases inversely with the age at which work was begun in any dusty trade. It follows that a youth beginning employment at sixteen is far more likely to acquire silicosis, other things being equal, than a workman who enters the same industry at forty. This statement is so true that if all of us were to live a lifetime of 150 years or thereabouts, probably most of us would acquire silicosis from ordinary exposures — from streets, schools, homes, farms, dust storms, et cetera.

Sex appears to play no part in susceptibility, although silicosis in the female is rare because of failure to enter the dusty trades. The negro is perhaps *more* susceptible than other races, while the northern European is by some regarded as *less* susceptible than other racial types. This is disputed.

Of more importance possibly than any other factor except exposure to silica dust itself is previous exposure to less harmful dust. When a workman in a silica-using industry earlier has been employed as a coal miner, glass worker, gypsum worker, or as a worker in almost any other dusty trade he appears prone to acquire silicosis in a shorter time and under conditions of less exposure than is true for a man without such history. This is not in conflict with the prior note that the simultaneous breathing of mixed dust apparently tends to retard the action of silica under some circumstances.

The very interesting chain of events following the arrival of silica in the lung sac is a matter for special presentation by those who follow in this symposium, particularly the pathologist and roentgenologist.

At the outset of a limited discussion of the clinical manifestations of silicosis, it should be emphasized that much less reliance is to be placed on such features than upon work history, x-ray examinations and autopsy. Well-established typical silicosis as demonstrated by the x-ray may be found in a workman without any complaint of discomfort and who, from physical examination, presents only meager evidences of abnormality. On the other hand, symptomatology and clinical examination may be of great worth in differentiating between simple silicosis and silicosis complicated by other affections, usually tuberculosis or other infections. The cardinal sympton of silicosis is shortness of breath. The cardinal sign is diminished chest expansion. In the early stages of this disease the silicotic tends to put on weight, which possibly is due to self-limitation of work exertion. Should he fail to put on weight, secondary infections should be suspected. The demonstration of silica in the urine on a qualitative basis is of no value. All persons present silica in the urine from the ingestion of this substance in food. Quantitative determinations may be of some worth.

Pain in the chest or history that pain

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has been present is common. Pain when present is more often localized in the mid-portion of the anterior chest, but may be anywhere. In those patients suffering from dyspnea, dizziness may be encountered. When marked fatigue or weakness is described by the silicotic, at once suspicion should be developed that secondary infection is present. Occasionally fatigue may arise from profound dyspnea and may not be present when dyspnea is not marked. Night sweats point to infection, as does also elevated temperature, which significantly is absent in simple silicosis. When sleeplessness arises it is usually associated with unfavorable progress. Sleeplessness may be associated with coughing, but, on the other hand, coughing is not of universal occurrence in silicosis.

On physical examination the respiratory rate is likely to be noted as increased, but not markedly so. The chest appears to be fixed and on palpation is lacking in resonance and elasticity. The information obtained through auscultation, percussion, and other determinations usually fails to provide any true concept of the pulmonary condition. In part this is due to the uniformity of change throughout the lungs, and, in part, possibly is due to a periphery of emphysematous tissue, which tends to mask evidences of deeper-seated conditions. The possibilities for confusion are but emphasized by the observation that a marked degree of simple silicosis may evince practically no evidences of deviation from the normal, while in the very early stages some cases may present rather marked departures.

Silicosis is prone to be associated with tuberculosis, and, in the absence of tuberculosis or other infection, silicosis readily demonstrated by the x-ray may be quite free from disabling capacity. Silicosis may activate antecedent tuberculosis, giving rise to the term tuberculo-silicosis, or may be followed by tuberculosis, leading to common usage of the term silico-tuberculosis. It has been observed that silicosis associated with tuberculosis is of low infectivity, and that the families of silicotics may not show a higher frequency of this disease than those of workers in non-dusty trades. However, this matter might well be the objective of further investigation.

Thus far in our discussion we have considered silicosis as a precise entity likely to present the same features in victims for all trades where exposure may be provided. This is not exactly true. For some unknown reason, the silicosis appearing in different trades is slightly different, particularly in its capacity to produce disablement. The foundry-worker at 45, with a moderate degree of silicosis, probably may finish out his work span without his silicosis reaching a disabling stage. This is less likely to be true of the gold-miner in Canada or the sand-quarrier or the sand-blaster. In general, however, it must be recognized that silicosis, once inaugurated, tends to progress, and practically never recedes. The differences mentioned as characteristic of certain trades are largely ones of rate of progress.

Years after the cessation of exposure, silicosis may put in its first recognizable appearance. An interval of seven years is not remarkable.

The average case of well-established silicosis, such as represented by the onset of disability, rarely lives more than five or seven years, and in the great majority of instances death may be attributed to tuberculosis. Cardiac involvement is not prominent as a direct result of silicosis, but pneumonia superimposed on silicosis is of common occurrence.

Medicine is confronted with unusual difficulties in establishing the degree of disability in silicotics. Accordingly, difficult problems center about the management of the silicotic as a worker. A moderate degree of silicosis is no bar to continued employment, provided further exposure is eliminated. However, in every instance, the victim of simple silicosis is a potential victim of tuberculosis. Upon the advent of tuberculosis naturally the medical management is closely related to that of tuberculosis unassociated with silicosis.

Mindful of manifest shortcomings of a discussion of silicosis so limited as this has been, an attempt at amends is made by directing the attention of those who may be interested to two recent publications. First, a book entitled "Industrial Dusts," by Drinker and Hatch, published by the McGraw Hill Book Company during the past few weeks; and, second, a smaller book entitled "A Second Symposium on Silicosis," growing out of an institute conducted in 1935 at the Trudeau School of Tuberculosis at Saranac Lake.

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