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This magazine is published to promote sound thought upon and concerning industrial medicine and traumatic surgery. To that end it will contain articles, news items, reports, digests, and other presentations, together with editors' comments. The editorial policy is to encourage frank discussion. On this basis contributions are invited.

Industrial Medicine

Reg. U. S. Pat. Off.

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The Journal of Occupational Diseases and Traumatic Surgery

With which are consolidated "The Industrial Doctor" and "International Journal of Medicine and Surgery."

The Science, the Law and the Economics of Industrial Health

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General Motors Corporation Medical Conference

—Dayton, Ohio, November 2 and 3, 1939—

THE recent MEDICAL CONFERENCE OF GENERAL MOTORS PHYSICIANS, held at the Biltmore Hotel, Dayton, Ohio, November 2 and 3, 1939, comprised five meetings and 18 presentations.

The Program was as follows:

Thursday, November 2:

MORNING SESSION: Chairman, M. M. SHAFER, M.D., Medical Director, Frigidaire Division, Dayton.

1. "Recent Developments in Relation to Silicosis," LEROY U. GARDNER, M.D., Saranac Lake, New York.
2. "A Plan for the Control of Tuberculosis in Industry," MAX BURNELL, M.D., Medical Director, AC Spark Plug Division, Flint, Michigan.
3. "X-Ray in Industry," M. WILLIAM CLIFT, M.D., Hurley Hospital, Flint, Michigan.
4. "Preliminary Report on Hygiene Studies of Welding," L. B. CASE and V. J. CASTROP, Industrial Hygiene Laboratory, General Motors, Detroit.

AFTERNOON SESSION: Chairman, A. L. BROOKS, M.D., Medical Director Fisher Body Division, Detroit.

5. "Recent Developments in the Occupational Dermatoses," MARION B. SULZBERGER, M.D., New York City.
6. "The Course of Disabling Morbidity among Industrial Workers, 1921-1938," WILLIAM M. GAFAFER, D.Sc., U. S. Public Health Service, Washington, D. C.
7. "The Industrial Nurse," HAROLD M. JAMES, M.D., Medical Director, Delco Products Division, Dayton.
8. "The Doctor's Part in the Personnel Program," GEORGE A. COBURN, Personnel Director, Delco-Remy Division, Anderson, Indiana.

EVENING SESSION: Chairman, WALTER M. SIMPSON, M. D., Director, Kettering Institute for Medical Research, The Miami Valley Hospital, Dayton.

9. "Relationships of Industrial Medicine to Private Practice," STANLEY J. SEEGER, M.D., Chairman Council on Industrial Health, American Medical Association.
10. "Unfinished Business," C. F. KETTERING, Director General Motors Research Laboratories, Detroit.

Friday, November 3:

MORNING SESSION: Chairman, G. L. BIRD, M.D., Medical Director General Motors of Canada, Ltd., Oshawa, Ontario, Canada.

11. "A Practical Approach to Supervision of Mental Health in Industry," F. S. PARNEY, M.D., Chief, Division of Industrial Hygiene, Dept. of Pensions and National Health, Ottawa, Ontario, Canada.
12. "An Example of Industry's Participation in the Anti-Syphilis Campaign," P. J. OCHSNER, M.D., Medical Director, Fisher Body Lansing Division, Lansing, Michigan.
13. "Study of the Factors of Employability, Especially Disabilities and Infirmities of the Elderly, and Problems Arising from Employment of the Same," GEORGE A. PAUL, M.D., Medical Director, Hyatt Bearings Division, Harrison, New Jersey.
14. "Early Diagnosis of Acute Appendicitis," C. RUSH MCADAM, M.D., Medical Director, Fisher Body St. Louis Division, St. Louis, Missouri.

AFTERNOON SESSION: Chairman, F. B. WISHARD, M.D., Medical Director, Delco-Remy Division, Anderson, Indiana.

15. "Relationship of the Length of the Inguinal Ligament to the Occurrence of Inguinal Hernia," C. M. HILLENBRAND, M.D., Medical Director, Harrison Radiator Division, Lockport, New York.
16. "Hernia in Industry; Review of the Cases During the Past Ten Years," R. L. JOHNSTON, M.D., Medical Director, Inland Mfg. Division, Dayton.
17. "Antiseptics and Their Action," R. M. FREEMAN, M.D., Medical Director, Linden Division, Linden, New Jersey.
18. "Management of Fractures of the Spine," R. W. POBORSKY, M.D., Medical Director, Electro-Motive Division, La Grange, Illinois.

Except for DR. SULZBERGER'S address on "Recent Developments in the Occupational Dermatoses" all of the papers given at this Conference are included here in full text, in the order of their places on the Program:

Recent Developments in Relation to Silicosis

LEROY U. GARDNER, M.D.,
Director, The Saranac Laboratory for the Study of Tuberculosis,
Saranac Lake, New York

WHEN your Chairman asked me to speak of new developments in the field of silicosis I was perplexed because all but a few of the pathological, clinical and diagnostic aspects of this disease are now well established. I realized

that you had become better acquainted than I with the practical problems of diagnosis because you were dealing with them every day. After some deliberation I decided to tell you of some of the experimental work in progress at the Saranac Laboratory and to permit myself to speculate as to its significance. These experiments are incomplete and no conclusions are possible now, but perhaps they may ultimately lead to a better understanding of the action of irritant dusts upon the lungs.

As far as I have been able to discover, it is still true that only certain forms of free silica and the group of fibrous silicates known as asbestos, are capable of producing fibrosis in a normal lung. Many other silicates have been incriminated by roentgenologists, but when autopsy specimens have been examined it is discovered that there is associated chronic infection or that the inhaled dust contains significant quantities of free silica.

I shall first compare the action of chrysotile asbestos with that of quartz upon experimental animals and review considerable evidence bearing on their respective modes of action. A few of these observations now have practical significance; others may prove to be quite unrelated to the pneumoconioses found in human beings.

FOR the last year or two we have been coming to the conclusion that inhaled *asbestos* fibres are irritating not because they are silicates but because they are stiff fibres which mechanically irritate the lungs. Unlike the free silicas, these minerals will not stimulate fibroblasts in any part of the body; only those in the lungs are affected. It was inferred that these organs were affected because the movements of respiration are so much more rapid and continuous than those of other viscera. Then it was discovered that if asbestos was ground very finely so that few of the fibres were longer than 2μ in length the irritating property of the asbestos was practically destroyed. Inhalation experiments with such fine chrysotile asbestos have now been continued for three years. No fibrosis has developed in spite of the fact that an average atmospheric concentration of 125 million particles per cubic foot of air has been maintained. In contrast I would point out that in a previously reported experiment[1] one third this concentration of long fibre asbestos dust produced well marked fibrosis after about two years.

If the effect of asbestos were chemical, one would expect that a decrease in size would accelerate tissue response. With free silica large particles have little effect, but as their size decreases cellular reaction becomes more vigorous and even constitutional symptoms may ensue.[2] With fibrous asbestos the reverse is true.

The histology of early asbestosis does not suggest a chemical injury. Even under the most extreme conditions that can be created by artificial injection there is no preliminary phase of tissue necrosis with infiltration of leucocytes as occurs with high concentrations of very fine quartz. The connective tissue cells merely multiply very slowly in areas where the asbestos fibres are caught in the bronchioles. As collagen forms and contracts, the air spaces are obliterated by scar tissue. In experimental animals, at least, this change is not a progressive one after cessation of exposure to the dust as is the case in the response to quartz. Perhaps the reason is the deposition of the peculiar iron-containing coating on the surface of the inhaled fibres giving rise to the characteristic "asbestosis bodies." Dr. Timothy Leary considers[3] that the resultant smooth rounded ends of these bodies are no longer capable of scratching the delicate surfaces of the air spaces.

Finally it is most suggestive that among dozens of different silicate minerals only the five known as asbestos, which are unique because they are fibrous in structure, should be commonly recognized as pulmonary irritants. The variation in chemical composition within this group is greater than that between them and many other silicates. In fact chrysotile asbestos has the same chemical formula as a non-fibrous silicate, serpentine, which is physiologically inert. Obviously irritation would seem to be associated with the physical, rather than the chemical, composition of these minerals.

One point of practical significance may be indicated by these observations, namely, that very finely ground asbestos is not dangerous. This conclusion has support in clinical observation, for it has long been known that at the Thetford mills there was no clinical asbestosis even though in former years the atmosphere was very dusty and the dust was extremely fine. The fact that fabrication of fibres of the same mineral in American plants could produce disease was one of the puzzling features of this disease. But these experiments offer a plausible explanation. The fine dust in the mills is composed of serpentine and extremely short chrysotile fibres; that in the spinning and weaving mills contains many more long fibres.

IN THE case of *free silica* everything points to a chemical form of irritation although the solubility hypothesis as now conceived is not compatible with all the observations that have been made. In experimental animals inhaling or injected with excessive quantities of pure quartz or flint in exceedingly fine state of subdivision, it can be observed that tissue reaction takes place in two stages. First there is an acute inflammation resulting in necrosis. Following it there is healing with fibrosis in the form of a granuloma whose end product is the silicotic nodule. The collagen laid down by the connective tissue cells is modified in a peculiar way analagous to that in the tanning of leather, and this is responsible for the characteristic hyalinization of silicotic fibrosis. Incidentally, leather has been tanned with silica and sections of such leather reveal that the collagen has undergone a similar hyaline transformation. In human subjects only cases of rapidly developing silicosis show evidence of a two-stage reaction. In those with ordinary chronic disease, degenerative changes are slight and overshadowed by the formation of connective tissue.

The solubility hypothesis is based solely upon indirect evidence. It is known that colloidal silica is toxic and that quartz will dissolve to some extent in slightly alkaline fluids of the same pH as those of the body. But it has never been possible to prove that silica has dissolved within the body, because the soluble material is immediately combined with local tissue elements. It is now generally accepted that the soluble silica detected in the urine and blood probably comes from ingested food and drink rather than from inhaled dust.

WE have made a good many experiments whose results are difficult to reconcile with the solubility hypothesis. They do not necessarily invalidate it because we may not have all the facts and do not know how to interpret them. For example, we have found that cellular reactions begin within 15 to 30 minutes after injecting fine particulate silica. In the test-tube traces of dissolved silica are only discoverable by an exceedingly sensitive colorimetric reaction after 12 to 24 hours. Possibly "vital" factors may accelerate the process, but no means of evaluating them has yet been discovered. No one has ever produced fibrosis by single or repeated injections of the various forms of soluble silica. The effect is apparently not due to the quantity of silica dissolved, for many of the inert silicates in vitro are more soluble than quartz or flint. Evidences of alteration of the surface of quartz particles long in contact with living tissue have thus far escaped detection.

In years of experimenting to discover the mechanisms by which free silica exerts its peculiar effects upon living tissues we have repeatedly observed evidences of a specific toxicity. Guinea pigs, injected intraperitoneally with large doses of fine quartz particles are often obviously sick for some hours. When Mr. Cummings and Mr. Redlin[2] were seeking an avenue for the quantitative introduction of silica they tried intravenous injections into rabbits and discovered the method which we now use as a standard for comparing reactions to different dusts. But incidentally they found that these animals would die very promptly if the unit dose of quartz particles were too large. These deaths were not embolic because they did not occur with large particles but only when the quartz grains were 3μ or less in diameter, and they did not develop on injecting suspensions of other minerals ground to the same size.

Mr. Dworski and Mr. Delahant have extended these early observations in a long series of experiments that have yielded interesting though puzzling information. They have shown that fine silica injected into the left side of a guinea pig's heart acts like a strong poison.

Symptoms of shock followed by death within a few minutes to one hour occur when a 400-gram guinea pig receives an intracardiac injection of 3 or 4 cc. of a 1% suspension of 1 to 3μ quartz particles in physiological salt solution. The reaction is specific in that it is not produced by quartz particles of larger size and it has not been observed on injecting suspensions of several other minerals ground to the same particle size.

Symptoms are initiated even before completing the injection by the onset of rapid, labored respiration. The skin of the face and extremities becomes pale. The animal displays uncoordinated muscular movements and sporadic convulsions terminating in death if the dose is sufficient. When smaller doses are administered complete recovery takes place.

Autopsy of fatal cases discloses an intense engorgement of the spleen, omentum and subperitoneal tissues which are deep purple-red in color. The liver, and in fact practically all the remainder of the body, is almost bloodless. Counts of ear-vein blood show only about half the number of leucocytes present just before the injection and a corresponding reduction in red blood cells. The blood no longer clots. There is no hemolysis.

Animals that have recovered from one reaction seem to acquire a tolerance so that in some instances at least twice the former dose may be injected without symptoms. The degree of tolerance, however, varies widely in different individuals.

These symptoms are not influenced by the injection of adrenalin. They are not reproduced by transfusing the blood of an animal dying in shock into another one of the same species. They are only exhibited on injecting suspensions of quartz particles less than 3μ in diameter. They are not produced by injecting the supernatant salt solution from which the quartz particles have been removed by centrifugation. The severity of the reaction does not vary with the age of the particle suspension, although the amount of soluble silica increases on standing.

The toxic effects of quartz can be more or less effectively neutralized by adding small amounts of different substances. Colloidal aluminum hydroxide gives complete protection in a dilution of 0.03%; colloidal ferric hydroxide is much less effective. Animal charcoal cannot be injected, because it flocculates with the quartz and produces emboli. The finely ground metallic aluminum, used by Denny, Robson and Irwin[4] in the prevention of silicosis has no influence upon acute intoxication. Further experiments are in progress to verify the conclusion of these authors that aluminum coats the surface of the quartz particles with insoluble material.*

Recent observations indicate that all species of animals are not equally susceptible to intravenous injections of quartz. Dogs, which develop silicotic fibrosis very slowly and under conditions not yet defined, tolerate intravenous injections of comparatively large doses of fine quartz without symptoms of any kind. Rabbits, guinea pigs and white rats all die of shock.

Soluble colloidal silica in dispersed phase is also

* Subsequent experiments have shown that sterilizing suspensions of metallic aluminum by heat destroys their power of neutralizing the action of silica. Unheated sus-

toxic, and intravenous injections produce the reactions just described with extremely small doses. With larger ones death occurs promptly with symptoms referable to the respiratory tract. The lesions are then confined to over-distention of the lungs and subpleural petechial hemorrhages.

THE results produced by injecting particulate silica into the circulation all suggest a relationship to a large amount of mineral surface suddenly brought into contact with the blood or tissues. This is borne out both by the absence of reaction to large particles in excess of 3μ in diameter and possibly by the observations on neutralization. Any effect due to preformed soluble silica liberated in the suspension before injection has been eliminated, and no active soluble silica could be demonstrated in the blood of an animal dying in shock by transfusion into a second host. Everything now suggests an effect taking place very rapidly at the interface between the quartz and some element in the blood or tissues. Whether this is due to the very rapid production of colloidal silica in this location or to unique electrical properties of quartz is still a fascinating subject for speculation. In the same category is the demonstration of the tolerance conferred by previous injections.

The manifestation of acute toxic symptoms by those species of animals which develop silicotic fibrosis most readily and their absence in the dog, which appears to be much more resistant, suggests that these immediate reactions may have some bearing on the etiology of silicosis. Until more is learned about the reactions in the dog no inference is possible.

The influence of particle size is just as significant in the production of advanced silicotic fibrosis as it is in eliciting acute symptoms of intoxication. Many injection experiments have convinced us that quartz particles over 3μ in diameter are of little or no physiological significance. The fact that the lungs of persons exposed to most dusts contain few particles larger than 10μ in diameter has been responsible for the establishment of this size as the maximum important in dust counting. As a matter of fact we know that with some dusts there may be many larger ones. While it would be just as well that any large fragments should be excluded from the lungs, it is felt that only ones 3μ or less in diameter are responsible for causing silicotic fibrosis.

We have long maintained that particles of this size are irritating because they present a sufficient surface of silica to the fluids. Recently Mr. Dworski decided to test this reasoning further by injecting graded doses of different sized quartz particles which would all present the same total surface area. He found, however, that regardless of the total surface, silicotic reaction only developed when the size of the particles was 3μ or less. On examining his material it was discovered that with particles 4 to 5μ in diameter or larger there were only two to four visible in a thin section of each phagocyte. These cells appeared to be quite uninfluenced by the ingested material. In the case of particles 3μ or under the number inside each phagocyte was countless, and there was marked evidence of degeneration of the cytoplasm of these cells. In other words, the effect of surface area had to be exerted inside of the cell and not upon the body fluids as a whole. Extracellular quartz seemed to have little influence, and even intracellular particles of a large size produced no visible evidence of irritation.

THIS observation brings us back to the cause of these progressive changes. Apparently they are initiated with degeneration in the primary phagocytes, an effect which is exactly paralleled in the histogenesis of tuberculosis. Some years ago I published a paper calling attention to the similarities between silicosis and tuberculosis.[5] In both of these diseases the primary irritants, quartz grains or tubercule bacilli, as the case may be, intoxicate the phagocytes. In tuberculosis the cells thus poisoned are commonly designated as "epithelioid cells;" in silicosis the morphological effect is similar. Visible fat droplets accumulate in the cytoplasm and the elements stainable by supravital dyes undergo rearrangements in characteristic patterns. Giant cells of the Langhan's type are produced in each disease by stimulation and repeated multiplication of the nucleus. In each disease many cells die and their degenerated products are liberated in the tissue spaces. Incidentally it is the pabulum of necrotic cells in silicotic lesions on which tubercle thrive so well, an effect which is responsible for the high frequency of complicating tuberculous infection in silicotics. In the tubercle it is in the necrotic or caseous tissue that tubercle bacilli are most frequent. Perhaps they not only cause such caseation but they themselves thrive on it. Coincidentally more cells migrate to the area and proliferate to form the granulomatous nodule that is characteristic of each disease.

Finally the production of necrotic tissue, high in lipoids, by two very dissimilar primary irritants recalls the experiments of Fallon[6] and his hypothesis that it is these lipoids which are directly responsible for the formation of the granuloma. He showed that the injection of such lipoids, freed at least partially of contaminating silica particles, was capable of producing a granulomatous nodule.

Such mechanisms may be involved in the production of both silicotic nodules and tubercles. It still remains to demonstrate the nature of the primary injury by each of these irritants, and it is this problem that will probably engage our attention for some time to come.

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A Plan for the Control of Tuberculosis in Industry

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IT IS a proud day when it can be stated that one of the safest places to be in this country is at work. Statistics from the National Safety Council seem to bear this out. The reduction in the frequency and severity accident rates in our industrial institutions has been nothing short of remarkable. Prevention has been the watchword.

With such improvements, the consideration of the medical departments in industry has more recently been drawn to the general health of its employees. Attention to the control of tuberculosis has been a very major issue. We believe that in no adult group throughout the country can so much be accomplished in prevention and early recognition of this disease as in our industrial institutions.

Legislation varies in the different States regarding occupational disease but the ultimate objective of this legislation is clear: The general health of the employee must increasingly become the responsibility of the employer. While the status of tuberculosis has been debated by many legislative bodies, silico-tuberculosis is definitely compensable. In the state of Illinois the new occupational disease disability compensation law went into effect on October 1, 1936. Dr. C. O. Sappington states that "during the first year of the operation of this law, 10.9% of disability claims filed were for pulmonary tuberculosis while silicosis claims were 15% of the total."[1]

The corporation that is looking toward the future with the best interests of its employees in mind certainly should be actively engaged in the problem of the control of tuberculosis. Many of our industrial institutions have already shown, by marked reduction of the incidence of pulmonary tuberculosis, just how this can be accomplished. Our own corporation has a very definite program concerning tuberculosis.

THE PROGRAM: 1. THE PRE-EMPLOYMENT PHYSICAL EXAMINATION. Every prospective employee is given a thorough physical examination. This is done with the intent of placing that individual in industry where he or she is best physically as well as mentally fitted. This examination is not done for the purpose of either passing or rejecting that person for employment. This pre-employment examination gives the examining physician the best possible opportunity to detect pulmonary tuberculosis.

Dr. Robinson Bosworth, President of the Illinois Tuberculosis Association, states:[2] "85% of adult pulmonary tuberculosis has its beginning in the age group 18 to 28. In the vast majority of cases there are no symptoms and no physical signs."

It is exactly this age group that concerns us most in our pre-employment physical examination, for it is during this age period that most employees first enter industry. We therefore feel that such an examination is not complete without an x-ray of the chest.

We believe, with Dr. Andrew R. Riddell, of the Industrial Hygiene Department, Ontario Department of Health, that the wholesale tuberculin testing in industry has many disadvantages. He states that[3] "the individuals must present themselves at least three times—there is considerable disruption of work—even after most careful explanation as to the meaning of the test, the employees who react positively are greatly disturbed."

Applicants in whom active pulmonary tuberculosis is discovered are sent to their family physician. Our roentgenogram is at his disposal, but must be returned to our files so that we can later compare the progress of the disease and determine at what future time those persons may be pronounced employable.

Applicants with arrested or quiescent tuberculosis are placed at work after being informed of their condition and instructed to return for periodic examinations. These examinations for new employees, in this classification, are conducted every three months.

It has been increasingly evident that our responsibility does not end there. We explain to the employee that while he now has an arrested tuberculosis, it was once active. We encourage him to have the members of his family examined by the family physician. We feel that not until then has industry wholly discharged its duty to the community.

2. PERIODIC EXAMINATIONS FOR OLD EMPLOYEES. These examinations are made at different times and for varied reasons:

(a) Upon returning to work after vacations.
(b) After absence caused by an illness.
(c) Re-employment following seasonal lay-off.
(d) Transfer from one type of work to another.
(e) Medical department requesting the examination for some definite reason.
(f) Volunteer requests by the employee for such an examination.

Little comment is necessary concerning these examinations except to state that every employee comes into our medical department for a general physical check up at least once each year.