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2:00—"Common Injuries of the Chest," by EDGAR W. DAVIS, Department of Chest Surgery, University Hospital, Ann Arbor, Mich. (illustrated with slides).

2:30—"Industry Turns to the Physician," DR. CAREY P. MCCORD, Director, Health Conservancy Laboratories, Cincinnati, (slides, gross specimens, autopsy material and special measuring instruments).

3:30—"Fractures of the Neck of the Femur, Acute and Ununited; Methods of Treatment for the Acute Fractures and Various Types of Treatment Available for the Ununited Fractures," DR. CARL E. BADGLEY. Professor of Orthopedic Surgery, University of Michigan Medical School, Ann Arbor.

4:30—Business Meeting.

5:00—"Vascular Dilatation and Cerebral Stimulation," general discussion.

6:30—Annual Banquet.

8:00—"Clinical Aspects of Silicosis," by DR. ROSS K. CHILDERHOSE, of Allenwood, Penna., (slides and exhibit).

DR. HORACE W. PORTER summarizes the program as follows:

"It was the aim of the Program Committee to get away from the usual symposium on fractures as much as possible, and deal more with the medical side of our specialty of industrial medicine and surgery.

"The address of Dr. McCord was of unusual interest; the various occupational diseases had never been discussed so completely before at any meeting of this organization. Dr. McCord had just returned from Yucatan where he was sent to investigate an occupational disease which suddenly became prevalent among the natives who gather chicle for the chewing gum manufacturers. The disease consisted of an necrosis of the left ear and chin, with the right side of the face uninvolved.

"In addition to discussing occupational diseases in general, Dr. McCord demonstrated, by autopsy of three guinea pigs, one of which was normal, the results of injecting dust containing silica into the peritoneal cavity of these animals.

"Dr. McCord also demonstrated various instruments for the proper and accurate measuring of dust and carbon monoxide.

"The movie on 'Burns' was a clinical masterpiece; the result of considerable work on the part of Dr. Grover Penberthy and his Assistant, Dr. C. N. Weller, of the Children's Hospital, Detroit.

"Another outstanding feature of the day's program was the evening address by Dr. Childerhose, who is associated with Devitt's Camp for Tuberculosis at Allenwood, Penna.

"Situated as he is in the anthracite district, Dr. Childerhose has made the study of silicosis a hobby, and is well able to discuss the subject authoritatively.

"At the business meeting the following officers were elected for 1936-1937:

President—A. H. WHITTAKER, M.D., F.A.C.S., Detroit;

Vice-President — LEON SEVEY, M.D., Grand Rapids:

Secretary-Treasurer — DON F. KUDNER, M.D., F.A.C.S., Jackson;

Directors — L. H. CHILDS, M.D., Flint; CARL E. BADGELY, M.D., F.A.C.S., Ann Arbor; and HENRY J. PYLE, M.D., Grand Rapids."

Clinical Aspects of Silicosis

By ROSS K. CHILDERHOSE, M.D.,
Allenwood, Penna.

SILICOSIS is essentially an industrial disease and as such it has a vital interest for us. Within the past decade especially, the interest in this particular industrial hazard has become so widespread, and the knowledge of the disease so increased, that it has become necessary for us as physicians to have a thorough understanding of the condition. Text books of 10 years ago contain little or no information on the subject, and we must concede that most of our knowledge is but recently acquired. Not only has interest become widespread amongst the medical profession, but legislators also, aided by representatives of labor, are enacting or propose to enact laws of compensation for the disability caused by this disease.

Considerable confusion exists regarding the various names that have been applied to lung destruction caused by dust. For example, the term pneumonoconiosis is the one usually applied to dust diseases in general, and because of this, is the preferable term because most cases are not due to one particular dust alone, but are a mixture of two or more dusts. For instance, the anthracite miner of Pennsylvania is subjected to two dusts, i.e., silica from the rock and coal dust. Thus he has pneumonoconiosis rather than silicosis. The term anthracosis applies to the pigmentation of the lung caused by the carbon. Siderosis is considered as the damage to the lung by the inhalation of iron dust, but I doubt whether any actual damage to the pulmonary tissues is done. As we shall see, the actual destruction is caused by silica, and the term silicosis is now generally applied for all these conditions although, properly speaking, pneumonoconiosis is correct.

Dusts are classified into two groups, i.e., organic and inorganic. We find the first type of dust in many industries, more especially in the textile trades. Carpet and rug weaving factories are noted for their heavy dusts. Tobacco factories also have some degree of dust of this type. These dusts may cause harm to the workers by: (1) carrying inorganic dust particles along the lung, (2) by acting as a vehicle for the entrance of bacteria to the lung and thus causing an inflammatory reaction or (3) by setting up a protein hypersensitiveness in the body to this particular dust. This latter condition appears in the form of bronchitis, rhinitis, and headache. In the textile industry it is known as "Shoddy Fever," and generally affects the new worker only. While these various disturbances by organic dusts may be serious or trivial, yet at no time do they cause a definite pulmonary tissue destruction, and for our discussion may be dismissed.

We must, therefore, look to the inorganic dusts as the cause of pneumonoconiosis. Any inorganic dust to do this must contain silica, either alone or in combination. It is amazing the number of industries in which silica may be the main constituent of the dust arising in the process of work. May I mention some of them?

Nearly all forms of mining render a silicosis

* Presented at the Spring Meeting of the Michigan Association of Industrial Physicians and Surgeons, Jackson, Mich., May 6, 1936.

hazard to the workmen, such as gold, silver, lead, zinc, copper, iron and anthracite. There is a high degree of hazard in the quarrying and dressing of granite, sandstone and flint. In iron foundries, particularly in the preparation of moulds for casting and the shaking out of these casts, we find a high silica content in the dust. Sand blasting, pottery manufacture, glass making, sand rock drilling for excavating work, all contribute a silicosis hazard at some stage in the work. I have only to remind you that silica is the chief ingredient in most of our abrasives, and this brings to mind scouring and polishing soaps and powders, sandpaper, sandstone and whetstones, sand blasting, and even some forms of tooth pastes and powder contain silica in a very finely ground state. In recent years the asbestos industry has been included, but this will be discussed later.

In order to have a clear understanding of the disease, we should remember several factors upon which its development depends. The *first* concerns the *size* of the dust particle inhaled. To be effective in its destructive action, all dust particles containing silica must be less than 10 u. in diameter, and the vast majority are certainly under 4 u. This means that the dust which is harmful is invisible. Particles larger than 10 u. cannot be phagocyted and are therefore carried off in the bronchial secretions. The *second factor* is the *density or concentration of dust.* Most authorities report this in the number of dust particles less than 10 u. in the cubic foot. Usually the number runs in the hundreds of thousands or millions depending on the clarity of the atmosphere. The standards of safety set up by various official bodies consider that a concentration of dust less than 10 million particles per cubic foot in which the free silica content is 35% or less to be the maximum limits of safety. Later, I shall discuss several industries and give the comparative dust concentrations that may be found when no prophylactic equipment is used. A *third factor* that is equally important in the development of the disease is the *length of time* to which the workman has been exposed. The duration of time necessary to produce silicosis is naturally affected by the other factors already mentioned, and will therefore vary greatly depending on the industry. Approximate time limits will also be mentioned later for some of the large industries.

In some industries the silica dust is contaminated by the presence of other inorganic dusts which seems to have either a stimulating or a retarding effect on the corrosive action of silica on the pulmonary tissues. Typical of these is that of coal dust which has apparently the effect of retarding silica, and that of alkaline soap in the abrasive soap industry where silicosis frequently assumes the acute form. Thus the problem of silicosis is different in different industries. The individual resistance of the laborer must also be taken into account. Many times I have been puzzled by the apparently small degree of fibrosis in a case where I would normally expect an advanced stage. These occasional cases have been explained by an extraordinary ability on the part of nature to excrete the silica before it can be ingested by the phagocytes and carried into the pulmonary tissues.

The action of silica on the pulmonary tissue is that of a foreign body having a corrosive action. The sequence of changes that occur in the lung following the inhalation of silica dust is readily understood by realizing that silica essentially causes an over-production of fibrous tissue and this in turn causes an entire derangement of the lymphatic drainage of the lung. Silicosis is therefore a disease or disorder of the lymphatic system of the lung. The pulmonary lymphatic drainage is slightly different from the venous drainage. Roughly speaking, we may say that the inner ¾ of the lung drainage is toward the hilum through the peri-vascular and peri-bronchial lymphatics, while the outer ¼ is drained through the subpleural cistern which in turn empties into the larger lymphatics along the inter lobar septa and from thence to the hilum. We shall see the importance of this in a few moments. At the bifurcation of each blood vessel lies a minute patch of lymphoid tissue through which pass these peri-vascular and peri-bronchial lymphtics. These lymphoid deposits act as an important filter to the inward passage of the dust particle.

Pathology of Silicosis

WHEN the dust particle reaches the alveolus it is phagocytosed by large mono-nuclear cells of endothelial origin, which for want of a better name have been called "Dust Cells". These cells are able at first to transport the particle to the neighboring tracheo-bronchial lymph nodes where their further progress is arrested, and the first destructive effect of the dust is noted. The present conception of its action is that silica is acted upon by the alkaline body fluids and slowly dissolves to form a complex colloidal silica hydroxide, which in turn has a highly corrosive action on the nearby cells. Thus we have a minute area of necrosis surrounding the dust particle. Because the conversion of the silica into the hydroxide is very slow we have, for practical purposes, a continuous irritant, and this explains why the simple removal of the worker from the dust exposure does not prevent a continued advance of the disease in the formation of scar tissue. This irritating and corrosive action of silica stimulates the formation of scar tissue in its neighborhood to an over abundance. Naturally, this distribution of scar tissue will depend on the lymphatic drainage. Thus, early in the disease we will see an intensification of the trunk markings and a nodular appearance on the x-ray film from fibrous tissue formation along these vascular channels. These small areas of fibrosis will be seen particularly in those minute areas of lymphoid tissue. With continued dusting these nodules become larger and give a "snow-storm" effect on the x-ray film. Should, however, the exposure to silica be so intense or carried out for a long time the lymphatic drainage becomes so disorganized that dust accumulates in the interstitial tissue and massive areas of fibrosis are formed. That portion of the lymphatic drainage that runs peripherally also carries dust particles, and in the course of time a pleuritis results and dense pleural adhesions are formed. The density of these adhesions is often astounding, and at autopsy they make the removal of the lung very difficult. These adhesions, especially at the diaphragm, interfere with respiration and a characteristic finding of the third stage is the marked irregularity and fixity of the diaphragm. With the continued formation of scar tissue, a marked degree of emphysema develops and, especially

along the mediastinal margins, the alveoli enlarges so greatly that emphysematous blebs are often found at autopsy. In the later stage of the disease there is a partial obliteration of the vascular bed and increased pressure results on the right heart. The whole picture is one of excessive proliferation of fibrous tissue with nature making valiant efforts to compensate by emphysema. Naturally the vital lung capacity is lowered and the extra burden put on the heart, bringing the final picture to one of cardiac decompensation.

Clinically, the disease is divided into three stages, but this classification depends more on the x-ray picture than on the actual clinical findings. Several classifications have been advanced, some of which are very complicated. None of them is entirely satisfactory but the one in general use is as follows.

STAGE 1: Physical signs and clinical symptoms are absent, but the x-ray shows a slight increase in the hilum shadows and perhaps there is a slight restriction of the right half of the diaphragm under fluoroscopic examination. Some claim that a slight haze in the mid-zone of the right lung is indicative, but I doubt whether this is entirely reliable. The x-ray diagnosis at this stage is most difficult.

STAGE 2: Physical signs and clinical symptoms are usually absent although there may be a slight bronchitis and the vital lung capacity is moderately lowered. The x-ray appearance is, however, quite characteristic—nodular shadows distributed equally on both sides in the inner ⅔ of each lung field.

STAGE 3: Physical signs are those of emphysema and moderate bronchitis. There may be a low grade fever but the most prominent sign is that of dyspnea on mild exertion, and some cyanosis. The x-ray picture discloses large patches of fibrous tissue in both lungs, distortion of the diaphragm by numerous pleural adhesions and areas of emphysema, particularly in the apex and at the base.

In acute silicosis, the first and second stages are not often seen because the process is so rapid that large masses of fibrous tissue form directly.

Complications

THE only complication of importance is *pulmonary tuberculosis*. It is a recognized fact that if there has been a pre-existing latent tuberculous lesion the inhalation of silica dust will undoubtedly re-activate it. If, however, the silicosis has been well established, then the secondary infection of tuberculosis will be somewhat limited and tends to be chronic. There is a certain similarity between the two diseases in that both have a predilection for the lymphatic system. If the lymphatics are obliterated or badly disrupted by areas of fibrous tissue, then the tuberculous lesion tends to be hemmed in, so to speak, and we have a fibroid type of tuberculosis. Just to emphasize the importance of this complication let me quote the figures of the Massachusetts Special Industrial Commission. Their findings among industrial workers were that the tuberculosis death rate among granite workers was 33%, and among tool sharpeners was 15%, while the proportionate mortality of males over 20 in the general population was only 7%. These figures apply, of course, to cases of pure silicosis, but sometimes the tuberculosis is modified by a contaminating dust. In the anthracite industry tuberculosis assumes a very chronic form with very few symptoms. This has been attributed to the "protective" action of the coal dust, but the exact explanation is still forthcoming. Where an alkaline dust is found in combination with the silica, as in the abrasive soap industry, not only is the action of silica enhanced, but also, the susceptibility of the worker to tuberculosis is increased.

Diagnosis

THE diagnosis of silicosis, like all other diseases, depends upon a well taken history. It is not sufficient merely to inquire whether the man was working in a dusty occupation. One should know the particular type of work, how long he has been at it, what precautions the company has taken to prevent dust, and what his former occupations have been. Frequently, with a well taken history, a diagnosis can be made with a fair degree of accuracy, and the diagnosis can then be clinched by an x-ray. The roentgenological picture is no doubt familiar to you. A combination of the history and the film is in most cases, sufficient for the diagnosis. Unfortunately, the physical examination of the chest is not of much value. The fibrosis, even when in large masses, gives very few signs because of the overlying areas of emphysema. It is this emphysema that acts as an effectual blanket, and hides the underlying pathology.

About the only problem in differential diagnosis is that of tuberculosis, and usually it is a question of whether tuberculosis is a complication or not. Generally, one can say that the distributions of the shadows on the film are those of silicosis because they have an equal and bilateral distribution, and are found chiefly in the mid-zone of the lung. In distinction, the shadows of tuberculosis have a predilection for the apex and are often unequally distributed. The presence of a cavity is a definite sign of tuberculosis, since we never get excavation in a pure case of silicosis. Just a word of caution in the interpretation of cavities in advanced stages of silicosis:—I have often been misled by the presence of areas of emphysema which are surrounded by patches of fibrosis. The appearance is most deceptive, and frequently the only means of definitely determining the presence of the suspected cavity is by autopsy. However, nearly all patients with cavity formation will show tubercle bacilli in the sputum. In all suspected cases of tuberculosis it is necessary to examine the sputum thoroughly. Just to rely on one or two negative sputum examinations is to have a false sense of security. I have seen many cases of silicosis with cough and expectoration, no fever, and normal weight and working, who are open cases of tuberculosis, and who were, therefore, continually infecting their families. It has been my practice to require at least 10 negative sputum examinations and two 24-hour collection specimens which were examined either by concentration methods or by culture or guinea pig inoculation. Every case of silicosis should be considered as a probable tuberculosis patient until proven otherwise, and even then he is a candidate for an acid fast infection.

Individual Industries

THE hazard of silicosis naturally varies in degree among the different industries in which silica is used. Some industries have more dust than others. Probably the greatest concentration

dust is to be found in hard coal mining where ers and miners helpers may be exposed to as as 232 million particles per cubic foot. Fortately, this dust has only 1½% of silica in it, wever, the rock drillers in the anthracite mines an average exposure of 82 million particles per bic foot, and the silica percentage is up to 31. is is explained by the fact that the rock in that ion contains about 31% free silica. Usually, it es about 20 years for the regular miner to deop a second or third stage silicosis, but the rock iller, on the other hand, will show a third stage as short a time as three years. For some reason other silicosis is not seen to nearly the same ent among the soft coal workers as we see it in anthracite region. There are probably 100,000 ners in the anthracite area, and you can underd that silicosis is quite a problem with us.

The asbestos industry has come in for much ention in recent years. The action of asbestos the lungs is similar to that of silica and in comition asbestos is a magnesium aluminum silicwith traces of iron. The destructive effect is as serious as pure silica, but it is supposed that estos undergoes changes in the lung quite like ose of silica, only slower and that asbestosis, as it called, is a true silicosis. The most dusty astos occupations are to be found in the factories, pecially where the mineral is broken up and finly woven into the numerous materials with ich we are familiar. The disease rarely adnces to the third stage. The one unique feature its pathology is the presence of the so-called sbestos bodies". These are highly refractive odies about 20 u. to 100 u. long. They are yellowbrown in color and do not take any stain. In ape the bodies may be clubbed, and they give a ussian Blue reaction for iron. In fact, it is beved that they are partly caused by the iron that found with the asbestos mineral. The finding these bodies either in the sputum or at autopsy, nfirms the diagnosis of asbestosis. There is the me frequency of tuberculosis in asbestosis as in licosis.

One of the great uses of silica is as an abrasive. ecause of its hardness, it lends itself particularly ell for this purpose. One of the chief offenders is sandstone wheel, and under its use there is med a constant stream of silica dust. Metal st is also formed, but it does no damage. Tool inders may be subjected to a dust concentration as high as 20-30 million particles per cubic foot. ome grinders have shown a third stage silicosis ter five to eight years' occupation. The use of ater on the stone in tool sharpening is of very ttle value. In a British study of 500 grinders who d worked at this occupation for 21 years there ere 73% who had silicosis. It is now advised at the carborundum be substituted for the sandone wheel because it is not nearly so dangerous, is, because it does not wear away so quickly and very much less free silica.

Silica is also used in the form of sand blasting. eedless to say, this exposes the worker to a real licosis hazard. In the iron foundry, the use of d in casting causes quite a high concentration silica in the atmosphere, particularly in the aking out of the casting and cleaning the casting. Massachusetts, a study of these conditions reealed a dust count as high as 200 million for the d chippers and 84 for the sand slingers. In lass manufacture, the hazard of silicosis may be high and also in certain parts of the pottery works. Each industry is a study in itself.

The more one studies the subject of silicosis the more one is amazed at the innumerable uses that modern science has developed for this seemingly inert substance. All the way from astronomical telescopes to tombstones do we find silica.

Treatment

I THINK we all realize the seriousness of silicosis, and naturally our problem is how to prevent it. This can be done in one way only. That is to remove the dust at its source. Vacuum dust removal equipment is the only way to insure against this danger. Standards of safety require that dust counts be kept under 10 million per cubic foot. The use of face masks, except where a definite supply of fresh air is given continually, is of no use. Those small face masks that cover the nose and mouth and contain a wet sponge are dangerous because they do not prevent the dust from entering the lung, and give a false sense of security.

Once the disease has gained a foothold, the man should change his occupation, but even this will not stop the gradual formation of fibrosis in the future. In the advanced stage, the patient should be put to bed, and if possible, given sanatorium care. In this way the cardiac reserve can be improved, and the patient made more comfortable. The seriousness of silicosis as an industrial hazard requires our careful consideration and study just as much as any other major problem.

Traumatic Derangements About the Ankle Joint

By M. T. KOVEN, M.D., F.A.C.S.,
and
H. E. BELLER, M.D.
Brooklyn, N. Y.

MANY traumatic mechanisms about the ankle joint are incompletely understood and often are inadequately treated. We shall attempt to classify the most important derangements while integrating where possible the manner of their causation and the lesions produced and methods of treatment.

The three chief factors which determine the type of injury in and about the ankle joint are the force of the impact, the obstacle, and the position of the foot when striking the obstacle. Thus, running or jumping proportionately increases the force, while the relation of the position of the foot impacting against an obstacle to a large measure determines whether the foot inverts or everts.

In the discussion to follow, one notes that the astragalus appears to play the most important role in the anatomic consideration. From its position it is the pivoting bone buttressed between the ankle joint and the foot. Hence, it is essential to review some features of the astragalus. Superiorly, its articulating surface narrows from the anterior to the posterior portion and articulates with the tibia. In complete dorsiflexion the wider sides are wedged firmly against both malleoli. In plantar flexion this mortice is insecure because of the space between its posterior lateral walls and the malleoli.

* Read before the Association of Traumatic Surgeons, New York City.