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The Asbestos Industry—
Certain Health Experiences Among Asbestos Workers

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In this presentation I shall try to outline certain health experiences which we have seen in asbestos workers, not only in my own company but in many other associated companies.

While asbestosis is a well recognized occupational disease, there are relatively few people in the working populations of the United States and Canada who are exposed to asbestos dust, and, actually, very few ever develop the disease. It will be shown that the lives of the asbestos workers are not shortened by their occupation, and also that work in these trades does not predispose the individual to other pulmonary diseases of nonoccupational nature.

The word "asbestos" is generally used to describe several fibrous magnesium silicates which are entirely different in their chemical composition and in their physical properties. There are several types of these fibers. The most important are the chrysotile, amosite, and crocidolite. Deposits of these various types of fibers are found throughout the world. The largest deposits are in eastern Canada and Africa.

These asbestos fibers are highly resistant to heat and some are resistant to acids. They have great tensile strength and large surface areas. Because of these properties, as well as their filamented structure, the industrial use of these fibers throughout the world is increasing. The textile industry has used these fibers for generations to produce blankets, clothing, threads, ropes, tapes, braided tubing, and filters.

In recent years, however, there has been an increasing use of asbestos in the insulation, building, and friction material trades. In addition, the fiber can be found in wallboard, shingles, pipe covering, floor tiles, brake linings and brake blocks, cements, putties, and extensively in plastics.

Historically the material has been used for many years. Those who are biblical students will recall that in the third chapter of Daniel the three men, Shadrach, Meshach and Abednego were thrown into the fiery furnace by Nebuchadnez-

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zar, the king of Babylon. They emerged from the fire completely unharmed, and archeologists today lead us to believe that they were protected from the flame by a blanket of woven asbestos fiber. Thus we can see that the mineral has been used for many centuries, but it was not until 1927—thirty years ago—that a definite occupational pulmonary disease was associated with the inhalation of this fiber.

As previously indicated, the industry is finding many new uses for the fiber when mixed with other materials. It is an established fact that when asbestos fiber is mixed with silica or diatomaceous earth or other potentially toxic dusts, the pulmonary changes resulting from the inhalation of these mixtures are not typical of asbestosis. The X-ray pattern is different; the clinical course of the patient is changed, or the susceptibility to intercurrent infection is increased or decreased.

Thus, in making a diagnosis of occupational pulmonary disease, it is highly important to obtain a detailed occupational history; not only the job title, but also a complete list of raw materials used in the particular job, so that asbestosis, siliosis, or the mixed pneumonoconioses can be differentiated.

We believe that there are fewer than 15,000 people in the United States and Canada who are employed today in mines and mills where their potential exposure is only to asbestos fiber. In this industry the various mining, milling, and manufacturing operations create some dusts which are capable of being inhaled. If asbestos fibers from ten to fifty microns in length are inhaled continuously and in sufficient quantities over a period of many years, a typical pulmonary fibrosis will develop. It has been demonstrated that this fibrosis is not due to the chemical, but rather to the mechanical action of the fibers.

The fibers are deposited in the terminal bronchioles of the lungs. Then the tissue reacts, coating the fiber and forming what is known as the asbestos body. This appears to be a defense mechanism of the lung.

Many individuals who have these asbestos bodies in their sputum have had only intermittent exposures to the dust and have absolutely no evidence of the disease. So it seems more appropriate to use the term "asbestos bodies" rather

than "asbestosis bodies," signifying an exposure to the fiber but not necessarily indicating disease.

If increasing quantities of the fiber are continually inhaled, the tissue reaction progresses and a generalized, diffuse fibrosis or scarring appears throughout the lower lobes of the lungs. With additional exposure this fibrosis may spread to the other lobes of the lungs, resulting in respiratory embarrassment, and eventually cardiac failure.

The pulmonary fibrosis resulting from prolonged inhalation of the fiber will produce a very typical X-ray pattern which does not resemble that produced by any of the other known toxic dusts. The X-ray picture should never be used to estimate the presence or extent of an impaired pulmonary function or disability. Many cases with X-ray evidence of advanced asbestosis have been known to carry on their work and live very comfortable lives for several years.

There's no typical clinical picture of asbestosis. The disease is very slow and insidious in its onset, and very slowly progressive with continued inhalation of the fiber. The textbook signs of cyanosis and clubbing of the fingers are virtually nonexistent in the cases of asbestosis we see today.

An X-ray survey was made of 708 employees working in an asbestos mill where the ore was dried, crushed, separated and graded, packed and then shipped. Operations in this plant required the employees to rotate through various jobs, so that it was impossible to relate any X-ray changes to a particular job or to a specified dust concentration. At the same time, it could be assumed that all members of the group had been exposed to varying concentrations of the dust.

The X-rays of these employees were divided into three groups: essentially normal; those with exaggerated markings, but not typical of any disease; and those with definite asbestosis. Of the 708 employees studied, 649 or 91 per cent had normal X-rays. This is of interest, because 204 of the employees, or 29 per cent of the total, had more than ten years of service. Two men actually worked over forty years in varying dust concentrations with absolutely no evidence of any increased markings in their X-rays.

Fifty-two of the 708 employees showed some increase of all peribronchial markings although none had any evidence

of asbestosis. Sixty-nine per cent of this grou had more than ten years of service.

Of the original group of 708, only 7 had developed X-ray evidence which could be diagnosed positively as asbestosis. These men exhibited various stages of pulmonary involvement. All were working steadily at their jobs with no signs of disability. It is of interest to note here that none of the men developed X-ray evidence of asbestosis with under twenty years of exposure.

Medical literature has given considerable attention to occupational pulmonary disease, but very little has been reported on the occurrence of nonoccupational respiratory disease among employees in the dusty trades.

In order to determine the incidence of nonoccupational disease in a group of 1,561 men and women working in the asbestos industry, it was decided to study the claims submitted for sickness and accident insurance. All employees in the survey group participated in the plan operated by an independent insurance company. Indemnification was made only after the nature of the illness had been certified by the treating physician.

The study included claims submitted over a five-year period for such illnesses as the common cold, sinusitis, pharyngitis, grippe, bronchitis, pneumonia, asthma, and pleurisy. Occupational pulmonary diseases such as asbestosis were excluded from the report.

Of the 1,561 employees in the survey group, there were approximately equal numbers in the dusty and nondusty occupations. Of all claims filed for respiratory diseases, only 45 per cent were for employees who had a dust exposure. Not only did this survey show that the incidence of disease over the five-year period was approximately the same in each group; but it also established that there was no appreciable difference in the duration of illness in either group. Also clinical observations for many years had given the impression that a dusty occupation in itself would not predispose a person to more nonoccupational respiratory disease than would a dust-free job.

Animal experiments and clinical observations have shown that asbestosis does not predispose a person to the develop-

ment of pulmonary tuberculosis, nor does it aggravate an apparently healed tuberculous lesion. In two isolated one-industry towns in the Province of Quebec where asbestos was mined and processed, the incidence of tuberculosis over a period of many years was no greater than that of other isolated towns with comparable population, but without a dusty trade. In addition, the incidence of tuberculosis among asbestos workers was lower than that in the general population in these mining towns.

Conflicting opinions and different reports make it extremely difficult to confirm or deny conclusively the causal relationship of asbestosis and carcinoma of the lungs. Too often a common conclusion is drawn from observations and experiences with different racial groups living in different parts of the world, under variable socio-economic conditions and working in diverse occupational exposures. To these variables I'd like to add the fact that there are the various types of asbestos fibers, which may have different actions on the lungs.

The Canadian experience with asbestosis has been limited to the chrysotile fiber. The majority of industrial processes in the United States use this fiber, but recently there has been some increase in the use of amosite and crocidolite. On the other hand, the British and other European industries use greater quantities of the harsher fibers, amosite and crocidolite. Therefore, in trying to clarify the causal relationship of asbestosis in cancer, many variable facts should be clearly identified, especially the type of fiber used and whether or not other dusts, such as silica or diatomite, were present in the industrial or environmental atmosphere.

To amplify this point I'd like to take a few moments to describe the three types of asbestos fibers most commonly used today.

An electron micrograph of the South African blue or crocidolite fiber would show that it is composed of many short, sharp spicules which are rather brittle.

Amosite fiber is brownish yellow in appearance and found in Africa and Australia. Seen under the electron microscope at the same magnification as above, the fibers would appear longer, less sharp, and they actually are less brittle than the crocidolite.

Crude chrysotile fiber found chiefly in Canada is greenish white in appearance. Electron micrographs show that this fiber is long, soft and silky.

I think that it is reasonable to assume that the short, sharp and brittle fibers such as crocidolite and amosite could cause much more extensive damage to the lung than could the long, soft, silky chrysotile fiber. Therefore, I think it is unfair to compare the experience of some of the European operations with our North American operations, using different types of fibers with different actions.

To amplify my statement that there are different occupational diseases having different X-ray pictures and different clinical patterns, I would like to describe the X-ray appearances of some of these diseases.

The X-ray in asbestosis shows a fine, diffuse, infiltration, bilateral in both lower lung fields. The heart outline is shaggy or completely obliterated. The nodular or conglomerate patterns of other pneumonoconioses are not seen in asbestosis.

In silicosis the X-ray reveals a nodular infiltration which is bilateral and appears in all lung fields from apex to base. Conglomerate shadows can appear in any of the various lung fields. The homogeneous or ground glass appearance of asbestosis is not seen here.

The X-ray of diatomite pneumonoconiosis can show an exaggeration of all linear markings, a very fine nodular or granular pattern, and frequent large coalescent shadows usually in the apices.

The X-ray pictures resulting from the inhalation of a mixture of dusts never follow the same pattern and certainly are not typical of either of the inhaled dusts. In present-day working environments a man might be exposed to mixtures of silica and coal, asbestos and silica, or diatomite and silica. Thus you could have many bizarre X-ray shadows changing from fine granular infiltrations to homogeneous and later to coalescent shadows in the same individual.

Several years ago, anyone who installed insulation materials was called an asbestos worker, because asbestos was practically the only material used for this purpose. Today, however, there are mineral fibers, synthetic fibers, clays, cement, magnesia, and a host of other substances used in the insulating

trades. The majority of these materials are relatively non-toxic, and it is obviously incorrect to label the insulation installer today as an asbestos worker.

There are numerous articles in the medical literature today reporting the incidence of asbestosis or asbestosis and lung cancer among asbestos workers. The conclusions of the authors should not be accepted implicitly unless the report clearly indicates that the so-called asbestos worker was exposed only to asbestos fibers and not to a mixture of dusts.

One company in the group I am reporting has a retirement plan which has been in effect for sixteen years. They have a total enrollment of slightly over 21,000 people; but, of course, this figure has varied slightly through the years. Since they started to use asbestos fibers just about 100 years ago, the following review of the pension plan will include employees who have had an exposure to the fiber over a long period of time.

In the past sixteen years there were 1,185 employees who retired under the provisions of the pension plan. The average length of employment for this group was 24 years. Although we are unable to determine specifically how many of these had extensive exposures to asbestos fiber, it is reasonable to assume that the majority had, since asbestos is used in nearly all of the product lines. Of the 1,185 there were 186 who retired for disability before reaching the age of 65. Only 6 of these had asbestosis.

This low incidence based on long employment with potential exposure would seem to indicate that the asbestos trade is not as hazardous as may have been indicated in the past.

Conclusions

1. Asbestosis is a recognized occupational pulmonary disease, slow and insidious in its onset. It takes many years of exposure to develop, it does not progress after exposure ceases, and there is very little disability until it is far advanced.
2. There are relatively few workers exposed only to this dust in industry today.

3. Conflicting medical opinions exist concerning asbestosis. Frequently adequate occupational histories are not obtained so that other pneumonoconioses are incorrectly called asbestosis.
4. Exposure to asbestos fibers does not shorten the life of the worker.
5. Inhalation of asbestos does not predispose the worker to other pulmonary diseases which will shorten his life.
6. The asbestos industry has controlled the exposure so that the incidence of asbestosis is very low.

CHAIRMAN LAWSON: Thank you very much, Dr. Smith, for being with us. It was a very interesting paper.

Do any of you gentlemen have any questions that you would like to ask of Dr. Smith while you have him available? If not, we will proceed to close.

Dave Cartwright, I think, gave us a splendid report on taconite this morning. The printed paper, of which you presumably have a copy, covers it in much more detail and has an excellent bibliography if you care to investigate further. We greatly appreciate what Dave has done for us.

Morris Piller—thanks ever so much for moderating the panel. And thanks to you, Chet Barney, Leon Hovey, Paul Shea, and Jack Wilson.

If there is nothing further to come before the meeting, we stand adjourned until 1953. I have been asked by the secretary if you will please—please!—leave your badges on the table as you go out.

Thank you all very much!

[The meeting adjourned at 12:10 o'clock.]