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before

U. S. Dept. of Labor, Occupational Safety and Health Hearing on Proposed Occupational Asbestos Standard

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My name is George W. Wright. I am the Head of Medical Research in the Department of Medicine of St. Luke's Hospital, Cleveland, Ohio. In 1932 I received the MD degree from Indiana University School of Medicine. After five years of Post Graduate Training in Internal Medicine with special training in pulmonary disease I spent two additional years in research training in the Department of Physiology at Case-Western Reserve University. From 1939 to 1953 I was a member of the Saranac Laboratories of the Trudeau Foundation engaged in studies of the pneumoconiosis, including asbestosis. Since 1953, I have continued this general field of study in my current position.

My statement will be confined to consideration of a single primary question. This question is: What is the quantity or concentration of asbestos, in the air breathed by those engaged in the production, manufacture or use of asbestos products, which can be tolerated for a normally expected work life without risk that such exposure will cause disease? The answer to this question goes to the heart of these hearings. While the question is rather easy to formulate it is difficult though not impossible to answer.

The obligatory information required for developing an answer to the primary question on an acceptable scientific basis is of two categories. One of these essential categories concerns the dose or quantity of asbestos inhaled and the other essential category concerns the biological response. What we need to learn is the dose-risk relationship.

The dose is estimated in terms of concentration of respirable asbestos in the air multiplied by the duration of the exposure. It is obligatory that we establish a dose, in measurable terms, at which the biological manifestation of disease occurs and then, by observing populations exposed to progressively smaller and smaller doses, determine the level at which the biological reaction to asbestos no longer occurs.

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To make observations with regard to the various dose levels of different working populations without knowledge of the biological response of these same populations is of no use in answering the primary question. To make observations of the biological response in various working populations without valid knowledge of the different levels of asbestos exposure experienced by these same populations is equally of no use for finding an answer to the primary question. Standards for a safe level of asbestos exposure proposed or set in the absence of reliable and valid data in both of these two categories must be considered empirical and to some degree arbitrary and should be recognized as such.

I wish to indicate some of the difficulties encountered in efforts to obtain the information essential for determining what the safe level of asbestos in the occupational environment truly is.

Asbestos is a generic term embracing four varieties that are in common commercial use. Of these, chrysotile is used in largest quantities followed by crocidolite, amosite and anthophylite. Each of these has different chemical and physical attributes. Some working populations are exposed to only one variety of asbestos while others are exposed to two or more simultaneously and in varying proportions. Thus there are several kinds of populations each exposed in quite different ways to one or more kinds of asbestos, each of which might react biologically in a different manner. Moreover, workers exposed to asbestos are further exposed simultaneously to other airborne agents. In production, manufacturing and utilization processes these may differ greatly. For example, the asbestos producer is exposed to asbestos with minimal coexisting agents while, in contrast, the insulation manufacturer inhales asbestos plus silica plus other pneumoconiosis producing dusts. Insulation applicators inhale these materials plus a variety of

additional agents present in whatever the environment is in the place where he is working at that time. Attention to such coexisting agents has been ignored in studies of insulation workers. Variations of type of fiber and the circumstances surrounding their use must be taken into account in answering the primary question and would suggest the possibility that different standards might be rational in order to meet different conditions of use of asbestos.

There are four, totally different biological phenomena or diseases thought to be related in some way to the inhalation of asbestos fiber. These are pulmonary fibrosis, bronchogenic cancer, thickening of the pleura, and mesothelioma. Not a single one of these diseases is peculiarly related to or caused solely by the inhalation of asbestos fiber. Each of these four diseases occurs "do no'vo" or for other reasons in persons who have never been exposed occupationally to asbestos. The fact is, the biological reaction attributable to asbestos is not a specific kind of disease induced solely by asbestos. Instead, the biological reaction to asbestos manifests itself by the fact that in some populations occupationally exposed to asbestos, there is, in contrast to nonexposed populations, an excess occurrence of one or more of the four previously mentioned diseases. To determine the safe level for the use of asbestos, we are required to demonstrate the level of exposure to asbestos at which no excess of disease develops when the exposed population is compared to a nonexposed population. In order to establish a safe level we must examine suitable control populations not exposed to asbestos, but age matched and residence matched, from which population one can learn the frequency of occurrence of the specific diseases in question. In addition, we must examine an exposed population, which can be ranked in various levels of exposure, in order to learn at what level an excess of these diseases occur in the exposed population.

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The observation of a biological abnormality in only one or two persons who have been exposed to the inhalation of asbestos fibers does not permit a comparison to suitable control groups or afford contrasting exposure values. Such observations are of no use for determining a safe level standard. Isolated case reports of mesothelioma, brochogenic cancer or pulmonary fibrosis in individuals who may also have had an exposure to the inhalation of asbestos fiber are of no use in setting a safe standard for asbestos based upon sound scientific principles. Larger populations in which only the frequency of occurrence of disease is reported, without the necessary data indicating ranges of exposure and in which no attempt to relate variations of exposure to disease is made, fall into the same nonusable category for setting a valid safe level. This is not to say that observations in such groups are of no value for other purposes, but the issue should not be confused by introducing data from such inadequate studies into considerations of safe levels of asbestos exposure. Suitable control populations made up of individuals in sufficient number who were never exposed to the occupational inhalation of asbestos fiber and occupational groups whose exposures are demonstrated to have wide variation are difficult to obtain but absolutely essential for our purpose. The absence of this kind of control data, excused or overlooked on the basis that it cannot be obtained, is an unacceptable condition if the safe level standard is alleged to be set on the basis of universally accepted scientific principles.

There is difficulty also with assessing the frequency of occurrence of the diseases thought to be related under some circumstances to the inhalation of asbestos fiber. There is disagreement as to the criteria for a valid diagnosis of mesothelioma. The criteria for a diagnosis of "asbestosis," especially in its least severe manifestation, are not generally agreed upon. The use of the chest X-ray for diagnosing the early manifestation of asbestosis is a case in point. A recent report by Murphy and his co-workers of a study of employees in a shipyard demonstrated that

twenty percent of the control, or nonexposed population gave evidence of X-ray abnormalities which, if observed in the exposed population in the absence of controls, in all probability would have been interpreted as being evidence of the effect of the inhalation of asbestos fibers. In a population which has been exposed to asbestos inhalation, it is very tempting to ascribe any departure from a perfect appearance in the chest roentgenogram to the occupational exposure, and indeed, in some studies this has been done. It is imperative to recognize that some persons who have never had an occupational exposure to asbestos will show the same X-ray shadows that have been interpreted by some as evidences of asbestosis in populations exposed to the inhalations of asbestos fiber. Studies such as those by Murphy et al exemplify the necessity of having suitable controls included in epidemiological studies bearing on the determination of a safe standard for asbestos.

There are still other difficulties having to do with establishing the dose-risk relationship. The health effects of asbestos inhalation are both dose and lapse-time related. Overt evidences of asbestosis do not appear until years after the initiation of the exposure. The interval between onset of exposure and its effect is even greater for the development of bronchogenic cancer or mesothelioma. This interval ranges between twenty and forty years. Because of the long time lapse between the initiation of exposure and the manifestation of injury, the incidence of disease occurring in populations now under study is the result of, and must be related to, exposures which took place twenty to forty years or more ago. To relate exposure measured only in the past five or so years to the current frequency of development of disease which actually was induced by exposure occurring years earlier, is a serious error. During the past twenty-five to thirty years there have been many technicological changes in the production, manufacture and use of asbestos

containing material. In many situations there is much less asbestos now being incorporated in the material than was true twenty or more years ago. In addition, there has been a progressive dust control effort to reduce the concentration of airborne asbestos in mines, mills and manufacturing establishments in a deliberate attempt to reduce and abolish asbestos related disease. So much has been accomplished in this direction by technicological change and systematic asbestos control efforts, that contemporary measurements of occupational environments cannot be accepted as representative of conditions twenty to forty years ago.

To summarize this part of my statement I strongly urge that when data offered in support of setting a safe level standard is being judged, one should ask the following questions:

1. What were the different kinds and proportions of asbestos used and what were the coexisting agents to which workers were exposed in the different occupations?
2. What were the specific criteria and methods used, and were they adequate for making a diagnosis of the biological reaction attributed to the inhalation of asbestos?
3. Were suitable controls in the sense of nonexposed populations and exposed populations whose exposures varied in intensity and duration utilized?
4. Was the exposure which was actually responsible for the disease properly determined or were contemporary dust estimates inappropriately applied to disease which was in fact the result of exposure years ago?

The demands inherent in these questions pose formidable obstacles to arriving at a scientifically valid single number which will indicate the concentration of occupational airborne asbestos that can be tolerated with safety for the customary duration of employment. In spite of this, as I will show later, there are substantial data which will satisfy the requirements posed by these questions and which can be used for the purpose of determining at least a first approximation to the safe level of occupational exposure to airborne asbestos.

We must now examine the various established and proposed standards for a safe level of asbestos in the occupational environment, in the light of the just discussed requirements as to data needed for setting a standard on a sound scientific basis. Systematic efforts to reduce the amount of asbestos in the air of working places by governmental directive was made in the factories of Great Britain beginning in 1931. No numerical standard for this purpose was set, but Statuatory Rules and Orders were promulgated. Later, on the basis of limited data relating impinger dust counts to X-ray abnormalities in several asbestos textile factories of the USA, Sayers and Dreeson suggested a numerical standard of five mp/ft^3 as a tentative safe level. This was the limit adopted by the American Congress of Governmental Industrial Hygienists in 1946. Utilizing the conversion data for textile mills published by Lynch and Ayers of the U. S. Public Health Service, five mp/ft^3 is the equivalent of approximately thirty fibers/cc.

In 1968 the British Occupational Hygiene Society suggested numerical guides aimed at reducing the risk of developing asbestosis. The suggested guides indicate that those concentrations, averaged over a three month period below 2 fiber/cc ought to be considered low, and that over the course of 50 years of employment, 100 fiber years is the level below which there would be less than a 1% chance of developing asbestosis as defined chiefly by the presence of rales.

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If asbestosis is defined by X-ray abnormality, the level becomes 135 fiber years. In 1968 the ACGIH changed its recommended standard to a TWA of 2 mp/ft^3, or twelve fibers/cc. In 1970 this Committee decided to change the standard to 5 fibers/cc, which is equivalent to less than 1.0 mp/ft^3. Several months ago OSHA set an emergency standard of 5 fibers/cc, and within the past few days NIOSH now proposes a standard of 2 fibers/cc.

What is the scientific basis for these various standards? The standard proposed by Sayers and Dreeson did attempt to relate dose of dust containing asbestos as measured by the midget impinger to the risk of developing pulmonary abnormalities in terms of abnormal X-ray patterns. Their study showed a higher frequency of abnormal X-ray patterns in those most heavily exposed as contrasted to those less exposed. They found no excess of abnormal chest X-ray patterns in those workers exposed to less than 5 mp/ft^3. Hence their suggested standard of 5 mp/ft^3. We now know, however, that the population studied was not observed long enough for the full range of disease to develop. The authors appreciated this possibility and proposed the standard as being a tentative one. In retrospect we know that the standard, although it led to marked lessening of the dustiness in some occupations, was not adequately based and the passage of time has demonstrated it to be an inadequate standard.

The numbers proposed by the British Occupational Hygiene Society are based on a study of 290 male employees from a single asbestos textile mill. Their employment began after 1933, all had worked for at least ten years, and the exposure period extended from 1933 until 1966. The indicator of disease was basal rales and X-ray changes. No measurements of dustiness in this mill were available prior to 1950. Membrane fiber counts done in the modern manner were used only in the last year of the study and these served as the factor for converting previous counts using