PROCEEDINGS

OF THE

THIRTIETH ANNUAL MEETING

OF THE

MEDICAL SECTION

OF THE

AMERICAN LIFE CONVENTION

1940 OFFICERS
MEDICAL SECTION

Chairman—Dr. A. J. Robinson, Medical Director, Connecticut General Life Insurance Company, Hartford, Connecticut.

Vice-Chairman—Dr. W. F. Blackford, Medical Director, Commonwealth Life Insurance Company, Louisville, Kentucky.

Secretary—Dr. B. F. Byrd, Medical Director, National Life & Accident Insurance Company, Nashville, Tennessee.

Member of Board of Managers—Dr. T. H. Dickson, Medical Director, Minnesota Mutual Life Insurance Company, St. Paul, Minnesota.

Program Chairman—Dr. [illegible] Livingston, Medical Director, Mutual Life Assurance Co. of Canada, Hamilton, Ontario.

dust particles of one to two and three microns in their largest diameter, that those particles had no physical characteristics in the ordinary sense in which we apply them. Artificial abrasives are among the hardest substances known. Neither silicon carbide nor aluminum oxide produced any reaction resembling silicosis in experimental animals nor do workmen exposed to these dusts show any clinical symptoms or X-ray appearances suggestive of silicosis.

SILICATES

A considerable number of silicate dusts have been studied and their effects upon experimental animals, both by inhalation and injection, noted. They produce little or no reaction in the body tissues and what little reaction some of them do produce does not tend to progress. An exception should be noted here regarding asbestos, which is considered separately.

Nor is occupational exposure to silicate dusts accompanied by any such clinical picture of disability and death as is found in silica dust exposures. The Public Health Service studied some talc mines in 1935. Only 66 men were exposed. Tuberculosis morbidity and mortality rates were very high in the county where the mines were located and the dust exposure was exceedingly severe, running into hundreds of millions of particles. Eight men showed advanced pneumoconiosis but tuberculosis was also present in most of them. It is conceivable that exposure to tremendous quantities of any mineral dust, regardless of silica-content, might so overwhelm the lungs that they could not deal with the invading substance. Irritation, inflammation, and probably infection would follow with disability in proportion. Such isolated instances do not invalidate the conclusions stated in the previous paragraph.

ASBESTOS

Asbestos is the only dust of combined silica which is known to cause a definite pulmonary fibrosis which may result in disability and death. Let me make that point clear. When we talk of silicosis and the types of dust that produce silicosis, we mean dusts that contain silica in a free or uncombined state, SiO_2. When the silica is in the form of silicon carbide or magnesium silicate or iron silicate that is what we mean by combined silica. The combined silicate dusts, as I have just mentioned, do not produce any pulmonary disease, with one exception, and that one exception is asbestos, which is a hydrated magnesium silicate mixture, with a little iron in it. Most of the asbestos fabricated in the United States comes from the mines in

Quebec. Asbestosis, however, is not found among asbestos miners, most of whom work in open quarries or pits, nor among men engaged in milling operations whereby the asbestos is prepared for shipment and bagged, but among workers in fabricating plants. Fabrication, you will understand, being in the nature of a textile process. Only recently has an explanation been offered for this anomaly. It would appear that not only is asbestos an exception in that it differs from other silicate dusts in its ability to cause structural changes in the lungs but its action appears to be mechanical and not chemical like the action of silica. If asbestos is crushed to fine particles, it will not cause asbestosis in experimental animals nor will asbestos produce any reaction elsewhere than in the lungs. If its action were in the nature of silica, the effect would be more pronounced with smaller particle sizes. In other words, Gardner established that the intensity of a silica reaction upon animals depended upon the smallness of the silica particles, because, to sum it up briefly, the finer the silica particles, the greater is the surface exposure which acts upon the pulmonary tissue.

Asbestos particles, below a certain size, do not produce any effect in experimental animals. Furthermore, you can produce a silicotic reaction, the typical silicotic nodule, in any organ by injecting prepared silica. You can get silicosis of the liver or of the spleen or elsewhere. But you cannot produce any reaction in animals with asbestos anywhere except in the lungs, and then only if your particles are sufficiently large. Consequently, Gardner concludes that the relatively long spicules of asbestos produce their effect because the lungs are never at rest and the constant movement provokes the fibrous reaction.

Like silicosis, asbestosis develops slowly, presenting the characteristic symptom of shortness of breath. The number of cases of asbestosis that have come to autopsy is small but there are several cases on record in which death was due entirely to the asbestos and not to any associated infection or other condition. There does not seem to be any pronounced tendency to tuberculosis in asbestosis, but most of the fatal cases have been complicated by other disease such as cancer and diabetes so that the extent to which the asbestosis was responsible for death is not clear.

I made a rather exhaustive study a few years ago of all the insurance certificates that I could find in which asbestos was given as the cause of death and then followed up the previous history of these people and got their medical certificates when they were on disability. The diagnosis given on the disability certificates was usually so at variance with the diagnosis given on the death certificates, often by

the same physician, that it was impossible to come to any definite conclusion and say, "this man died of asbestosis," when his previous disability certificates showed that he had myocarditis or high blood pressure or cancer or diabetes or pulmonary tuberculosis.

The pathology and X-ray appearance of asbestosis are entirely different from silicosis and in the absence of a definite history of exposure over a considerable period of time to asbestos dust, the diagnosis is difficult. A great deal of success has been attained in controlling dusts in the plants where asbestos is fabricated so that it is not likely that asbestosis will ever become an important industrial disease, as far as the number of cases is concerned.

The scarcity of clinical data on asbestosis makes the prognosis doubtful. We have had a small group of cases under observation for about ten years — there were originally, I think, some sixty odd men in this group and we still have fifty of them where we can get at them — and some of these show a definite but slow progression as far as can be judged from the X-ray films. However, what the outcome may be remains to be seen. These men are all working and show no signs of any disability.

In the meantime the plants have been cleaned up so that these men are not exposed now to inordinate quantities of asbestos dust, so it is difficult to say what would have been the progress of their disease had their working conditions remained the same.

SILICOSIS

It may be said that there is a very general accord among the leading investigators and research authorities in the opinions and conclusions presented at the international conferences on silicosis (Johannesburg, August, 1930; Geneva, August and September, 1938.) Such differences of opinion as may be expressed from time to time do not seriously affect the general agreement on the basic factors affecting the cause, incidence, effects, and prognosis of silicosis.

The effect of silica upon the lungs depends on the nature and extent of exposure. That is to say, upon the quantity of dust in the air breathed by workmen, the amount of free or uncombined silica in the dust, the size of the dust particles, the presence of other substances in the dust which may modify the action of silica, and the length of time or duration of exposure. The individual himself must also be considered. Previously existing disease of the lungs, especially a tubercle infection, will influence his reaction to inhaled silica. There may be other phases of susceptibility about which we know little or