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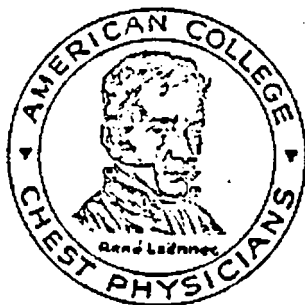
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El carcinoma del pulmón también ocurrió con tal prominencia que no se debe abandonar la posibilidad de que sea producible en un sujeto sensible por la asbestosis grave, a menos de que sea refutado esto por investigaciones ulteriores.

Aunque la tuberculosis pulmonar ocurrió con más frecuencia que en la serie general de autopsias, el análisis cuidadoso de los datos pertinentes no apoya la idea de que exista alguna relación entre estas dos enfermedades, mientras que, por lo menos en un caso, hubo datos que indicaron que la tuberculosis puede seguir mejorando aún cuando la asbestosis continúa avanzando.

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Discussion

LEOPOLD BRAHDY, M.D., F.C.C.P.
New York, New York

Prior to 1930 there was almost no mention in American medical literature of any specific disease due to asbestos. In the British and French literature, authors recorded high frequency of chest

symptoms among asbestos workers and speculated on the relation of symptoms to the inhalation of asbestos or of silica or of iron or to other conditions associated with asbestos work. There were reports of a high tuberculosis morbidity among those exposed, and general high mortality. Some authors gave fair descriptions of the pathology or of the clinical picture of what we now call asbestosis but correlation of the pathology, roentgen appearance and the essential factor in etiology was lacking. There was consensus, however, that somehow asbestos work was harmful. The International encyclopedia on industrial diseases, entitled "Health and Occupation," published in 1930, deplors the lack of more accurate and detailed data on the asbestos hazard, and goes on to say that the increasing utilization of asbestos urgently calls for study of the condition.

In 1930, Lynch and Smith published the first necropsy protocol together with the detailed occupational history which left no doubt that the disease is a lung fibrosis and is directly due to the inhalation of the asbestos in the dust. R. S. Mills, in the same year, and later, Lanza, Gardner, McConnell, Pendergrass, Shull, Sparks, Stone and others made important contributions. In the seventeen years since that confession of confusion in the encyclopedia and Dr. Lynch's pioneer publication, American medicine has progressed with speed in the knowledge of this condition.

Today, we have had the privilege of hearing the latest report of the continuing observations by Dr. Lynch. Some problems, he tells us, need further elucidation, among them, the relation to carcinoma. Although we must keep an open mind, the evidence warrants the tentative conclusion that asbestos workers do have a higher incidence of lung cancer. Now that early lung cancer often is amenable to surgery, this gives added value to the periodic chest x-ray survey among these workers and calls for more meticulous film interpretation than is essential in most survey work.

From the viewpoint of public health, of diagnosis, and of workmen's compensation adjudication, observation is needed to establish the etiologic relation or the absence of any relationship between tuberculosis and asbestosis. Many of the older clinical observations (though not all) favored the conclusion that there is a relationship similar to that between silicosis and tuberculosis. The report we have heard today does not confirm this view. One statement, or rather the implication of one statement, of Dr. Lynch must be interpreted in the light of other clinical experience. Dr. Lynch reported one case in which tuberculosis improved in the face of advancing asbestosis. However, we occasionally find tuberculosis improving in spite of the presence of silicosis and we do know that silicosis adversely affects tuberculosis. Tuberculosis sometimes

heals under the most unfavorable conditions of the infected organ. That tuberculosis may heal in the face of asbestosis, therefore, should not add any weight to *either* side in evaluating the effect of asbestosis on tuberculosis.

Dr. Lynch may not be conscious of a sermon to the clinician contained in his post-mortem reports. He said that in most of the well-developed, though not in advanced cases, the history of asbestos exposure was traced *after* necropsy diagnosis. To me, that means that the clinicians neglected the occupational histories of their patients, probably because there was another serious disease present. That the diagnosis was made post-mortem suggests there were not enough routine chest films made of these workers when they were well and certainly not sufficient chest examination when they became ill. We should take to heart this failure to recognize the occupational disease during life. Many new substances were introduced in industry in the last decade; new compounds known only to the chemist today will be used in industry tomorrow. Some of these substances may produce chest symptoms. The *clinician*, practising in the locality of the industry, and not the pathologist should be the first to clearly state that an industrial process is a health hazard. The *clinician* must be the first to know, what is equally important from a health viewpoint, that some suspected industrial process is *not* a health hazard. Let us reflect on the significance of the fact that twenty years ago hardly one of us would have suspected that the lung symptoms presented by one of our patients working on refrigerator insulation were directly due to asbestos. Perhaps today some of us are missing the etiologic factor in cases that now are under our observation because we neglect the occupational history. Dr. Lynch's studies on the pathology would be of only passing interest if he had not combined them with investigations into the occupational history in each individual case. It is the correlation which makes them a major contribution to medicine.

There are other conclusions of Dr. Lynch's applicable to problems of the clinician. Dr. Lynch finds indications that when exposure ends the disease develops but little further. The prognosis, therefore, is definite, and that must be a comfort to the victim that makes it easier to get him readjusted to a new occupation and makes Workmen's Compensation adjudication much simpler. Dr. Lynch is speaking, of course, of the fibrosis; his statement must not be applied to the secondary cardiac embarrassment once that has begun. That may progress even without exposure. If I misinterpret Dr. Lynch's conclusion I trust he will emphatically correct me. The pathologic finding that fibrosis persists and ages into scar tissue explains the outstanding physical sign of the disease,

limitation of chest expansion and also means that no recession of symptoms or signs directly related to the fibrosis is to be expected.

Another finding is that length of exposure and severity of the disease are correlated. This sounds self-evident but in silicosis we cannot make that statement. This characteristic should enable us to concentrate and strengthen asbestosis case-finding among those workers who need it most, i.e., those with long exposure, and to be alert when examining a patient who declares: "Oh no, that dust does not bother me, I'm used to it, worked there without trouble for twenty years." Furthermore, this correlation of exposure-time with severity enables a workmen's compensation board to equitably allocate liability when two or more employees with asbestos hazards are involved in the same case. Then, too, Dr. Lynch's conclusions mean that when we have negative medical findings on a patient exposed for only a few months we can reassure him with confidence on the absence of dust disease.

As if to balance these conclusions which make our jobs easier, the newer observations indicating that nodular fibrosis may occur in asbestosis makes matters more complicated. Now, knowing that nodulation occurs in asbestosis not only the pathologic but also the roentgenologic distinction from silicosis becomes more blurred. We must be more cautious or perhaps, give up trying to reason from a film or a pathologic finding as to the type of dust exposure.

This has been an informative presentation. It necessarily is an unusual privilege to be first to hear reports of pioneer investigation from men whose publications have their place in the history of diseases of the chest. I want to express my thanks to Dr. Lynch for that privilege today.

Discussion

W. BERNARD YEGGE, M.D., F.C.C.P.
Denver, Colorado

I wish to compliment Dr. Lynch on the contribution he has made to our knowledge of asbestosis.

Pulmonary asbestosis differs from silicosis by size, etc., of the particles inhaled and by chemical composition. Asbestosis is caused by an alkali which is magnesium silicate, along with calcium and iron. In silicosis the offending dust particle is silicon dioxide. In asbestosis the shape of the particle causes them to be arrested more often in the bronchioles and the alveoli and they are not so