Trasys Center for Env. Hlth - Progress report re. Center Grant ES00002 to NIEHS
HSPH
10/15/72

M-4088

9. ASBESTOSIS PREVALENCE

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In June 1972, we re-examined 150 of the 195 shipyard workers whom we had initially studied in 1965 and 1966. We are continuing to trace the remaining workers and to attempt to obtain death certificates and autopsy data of those who have died.

Our investigations in June consisted of repetition of the standard respiratory disease questionnaire and forced vital capacity (Dr. Ferris), physical examination of the chest including flow rates and ultrasonic probing of the chest to determine pleural abnormalities, and measurement of the diffusing capacity. We have just recently (9/30/72) completed an examination of the roentgenograms on these workers by a panel of three observers (Dr. Weber of the Mass. General Hospital, Dr. Gaensler of Boston University, and Dr. Murphy). The data are currently being analyzed. Preliminary results suggest that our assessment of the prevalence of the disease in 1966 underestimated the seriousness of the problem. Five of twelve workers diagnosed as asbestotic have died of cardiopulmonary disease in the interim. Four others have shown obvious signs of progression. In addition, there are probably ten new cases of asbestosis.

Further analysis of the specific parameters we have obtained in this longitudinal study will hopefully allow us to diagnose this disease earlier.

The inadequacy of the available tools for diagnosing asbestosis has stimulated work in two areas: breath sound analysis and ultrasonic examination of the pleura. These are summarized below.

9a. AUSCULTATION OF THE CHEST

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The role of chest auscultation in diagnosing asbestosis was studied in the pipe coverers previously referred to. Two methods for making auscultation more objective were examined. The first was a simple system for accurate recording of auscultatory findings. It allowed the computation of arbitrary scores which were comparable to other parameters for assessing pulmonary fibrosis. For example, duration of exposure in pipe coverers exposed to asbestos was as closely associated with a rales score ($r = .388$) as with more complex studies such as single breath diffusing capacity ($r = -.395$); steady state diffusing capacity at rest ($r = -.175$) and vital capacity ($r = -.386$).

The second method, sound spectral analysis, promises to make auscultation more accurate. Analysis of numerous rales from asbestos workers revealed that most had frequency peaks between 500 and 800 K. These are readily distinguished from "coarse" or "moist" rales in pneumonia and congestive heart failure wherein the rales had lower frequency spectra. We were also able to clearly distinguish between rales ("discontinuous" noises) and rhonchi ("continuous" noises) via this method. We believe that further study of such methods may be useful for early detection of diffuse lung disorders.

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9b. A-SCAN ULTRASOUND ASSESSMENT OF PLEURAL THICKNESS

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Dusts which cause Pneumoconiosis are often associated with concomitant production of abnormalities of the pleura. Silica, asbestos and talc are particularly recognized as minerals which lead to pleural disease. Indeed, persons who die with advanced asbestosis have quite markedly thickened pleura. This thickening is associated with reduced pulmonary function even in the absence of parenchymal disease. Although marked pleural thickening can readily be seen on a posterior-anterior chest roentgenogram, minor degrees of pleural abnormalities are not well characterized roentgenologically. The prominent appearance of pleural disease at post mortem in cases of asbestosis can be compared to the relatively subtle shadows cast on the roentgenogram in such cases.

Ultrasonic methods have been employed to study the pleural surface. Indeed, Finnish authors have described abnormalities of the pleura in asbestos workers using both A and B-Scan ultrasound. Unfortunately insufficient experience with the A-scan has led to a reasonable degree of confusion in identifying echo patterns from the chest. When used in conjunction with B-Scan ultrasound the A-Scan is far easier to interpret than when it is used by itself. B-Scan ultrasound, however, is relatively complex to perform from a technical standpoint. Consequently, when indicated for the study of pleural abnormalities it certainly only could be used in specific individuals in whom there is a reasonable suspicion that the pleura is abnormal as determined by the more conventional techniques. In contrast, A-Scan is technically simpler and thus can be employed to screen populations with industrial exposure. With these thoughts in mind we attempted to study the specific echoes obtained while

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using A-Scan ultrasound on the chest wall.

Our preliminary results indicate that reflections from the lung surface are, indeed, markedly different than that found from the tissues situated immediately peripheral to the lung. The pulsatile nature of the reflection from the lung itself is, indeed, the most striking phenomenon seen with this ultrasonic method. By studying the costophrenic gutters both in inspiration and in deep expiration it is possible to indirectly demonstrate the presence and absence of lung tissue immediately adjacent to the chest wall. We currently believe that we can characterize with a reasonable degree of certainty various tissues from the skin to the surface of the lung. We have utilized this technique in a number of selected normals as well as in a group of people with an asbestos exposure who have roentgenologically evident diffuse pleural thickening as well as pleural plaque formation. There appears to be a clear difference between the normals and exposed persons but further work in this area is necessary. We believe that this work could lead to a method useful in monitoring workers with exposure to pneumoconiosis-producing dusts.

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Asbestos Prevalence (1972)
ACL Doc 25481 · Page 5 of 6
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