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UNIVERSITY OF MARYLAND
SCHOOL OF MEDICINE
DIVISION FOR PULMONARY DISEASES
BALTIMORE 1, MARYLAND

January 27, 1965

Robert F. Chenowith, M. D.
Medical Director
Maryland Casualty Company
Baltimore, Maryland 21203

Dear Doctor Chenowith:

We have completed our review of the group of records of the employees of the Grace Company who are associated with the ZONOLITE operation in northwestern Montana.

I have chosen to approach this from the point of view that the Grace Company is desirous of maintaining this operation while assuring both themselves and their employees of the smallest possible risk of health hazard from the development of pulmonary asbestosis.

You will find enclosed the original records plus:

1. A brief summary of the literature.
2. An analysis of the health records.
3. An outline for an epidemiological study of the effects of this industrial hazard.

I would summarize our thoughts and recommendations in the following way. I have nothing but the highest respect for Dr. Nelson in undertaking this study while carrying on a busy practice. Howevem he suffers from a lack of equipment and personnel needed to perform an adequate evaluation. While many of the records are unsatisfactory, a plus 20% x-ray incidence of pulmonary infiltrate plus some confirmatory physiologic evidence would suggest that this plant has an important health problem. This is particularly so, as respiratory function tests of the type he performed are aimed primarily at eliciting obstructive airway disease while pulmonary asbestosis is primarily a fibrosis or restrictive disease. In other words, should more sensitive tests, i. e., diffusing capacity, lung compliance, work of breathing, arterial blood gases and ventilation at rest and exercise, and total lung volumes and airway conductance be applied I would not be surprised by a doubling of this incidence of disease. A second point to note is that in any group of workers such as this where a majority are cigarette smokers one can expect to find a prevalence of 20% or greater of obstructive

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airway disease on the basis of smoking when sensitive tests and questionnaires are utilized. The company cannot be held responsible for this disease. In addition a 5-15% prevalence of asthma can be expected in the adult population. Therefore, if the company plans to evaluate this health problem, it shouldn't be done without the assurance of a complete epidemiologic and physiologic work-up and some care must be taken that in the selection of respiratory function tests one is not caught in the frequent trap of assuming that a few sophisticated tests offer an assurance that in the future individual workers won't be studied, possibly for legal reasons, with complete testing which will reveal defects not previously shown. This exact circumstance has occurred in Maryland and I can assure you that the defendants lost such cases, no doubt in part from their unwitting failure to obtain complete testing in a complete study plan at the outset.

I would recommend to you that a complete epidemiologic and physiologic study be done in Kalispell in the near future, followed by the institution of whatever dust control measures are deemed necessary and a repeat of the testing procedure in two - three years. In addition, a matched, control group of workers not exposed to asbestos be studied, possibly from another Grace Co. operation, eg. Davidson Chemical, etc.

Such a study would be costly as it would involve moving a team of investigators and equipment to Kalispell on two occasions plus the study of a control group. However, it has been my experience that the cost of such a study very seldom exceeds the size of one lawsuit resulting from industrially acquired disability. It is possible that the Div. of Occupational Health, U. S. P. H. S. would support such a study.

It is undoubtedly anticlimatic to note that we have the personnel and experience to do such a study and I will be speaking to the Montana Academy of General Practice this June.

Should you have any further questions, I would be happy to attempt to answer them.

Sincerely,

William S. Spicer

WILLIAM S. SPICER, JR., M. D.
Head, Division for Pulmonary Diseases

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Enclosure

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ASBESTOSIS STUDY

The following is a cursory analysis of 140 subjects, 136 of which work in an asbestosis plant. Four subjects are city employees.

The information available consists of name, age, height, weight, vitalor spirometry, chest film report, and brief work history.

Methods:

Spirometry was obtained with the vitalor. Tracings were examined for technical accuracy and for subjects cooperation and exerted effort. The forced vital capacities (FVC) were expressed as % of predicted normal vital capacity. These are classified: S = satisfactor E = exceeded 5.0 liters which is the maximum capacity of the vitalor, and U = unsatisfactory.

The forced expired volumes in 1 second (FEV1) are expressed as % of predicted normal for 1 second. These and the maximum expiratory flow rates (MEFR) were categorized: 1 = good, 2 = fair, and 3 = completely unsatisfactory record.

The chest film reports were graded according to the asbestosis classification used by Williams and Hugh-Jones (1). Each lung was divided into thirds and each area graded 0-3 for no, slight, medium, and heavy mottling (grading 0-18 for no to maximum involvement from asbestosis). Pleural reaction was also recorded. [redacted] subjective in that it was accomplished from the x-ray reports and not from the x-rays directly

Results:

Radiological Changes compatible with asbestosis were present in 20.6% (28/136) of the plant workers: 39% (18 subjects) of these had pleural reaction, presumably from asbestosis as no case of pleural reaction was reported in the absence of evidence of asbestosis on x-ray. The severity of asbestosis appears to be related to the total duration of work and to the duration of work in the mill (table 2). The x-ray grading system of 0-18 would classify 26 of the 28 workers with asbestosis as having

minimal disease (grade 1-6), 2 as having moderately advanced disease (grade 7-12), and none as having far advanced disease (grade 13-18).

Spirometry. Vital capacities were considered satisfactory for analysis in 35% of cases (49/140). 18% (25 cases) of the records exceeded 5.0 liters while 47% (66 records) were considered submaximal for various reasons. Therefore, 65% of the vital capacities could not be used in the analysis.

60% (85 records) of the $FEV_{1.0}$" were either good or fair and considered satisfactory for analysis. The other 40% were excluded from the study.

62% (87 records) of the MEFR's were either good or fair and considered satisfactory for analysis. The other 38% were excluded from the study.

The relationships among spirometry, x-ray evidence of asbestosis and work history are seen in Tables 1 and 2. It appears that the FVC and MEFR values in those without asbestosis on x-ray are slightly below normal. This is probably a result of submaximal effort on the part of the subjects. The FEV_1 in this same group is within normal limits. Relative differences between the different groups suggest mild decreases in the vital capacity, FEV1 and MEFR in those with asbestosis The FEV1 may be worse in the mill workers. No definite changes in spirometry are seen with increasing grade of asbestosis however, the number of cases in each group is small.



Discussion:

This study indicates a 20% incidence of asbestosis in workers studied. Crude spirometry suggests that this group of affected workers have mild restrictive and obstructive ventilatory insufficiency. The 4 city employees studied revealed no definite trend. No evidence is present to indicate that they have been affected by asbestosis.

1. Williams, R. and P. Hugh-Jones. The Radiological Diagnosis of Asbestosis. Thorax, 15:103, 1960.

1	7	82.0 (2)*	70.7 (7)	232 (3)	134.1
2	8	76.0 (1)	66.5 (4)	275 (3)	160.2
3	5	85.7 (3)	83.0 (3)	190 (3)	156.8
4	3	84.0 (2)	92.5 (2)	225 (3)	150.3
5	2	75.0 (1)	--- (0)	--- (0)	213.0
6	1	86.0 (1)	77.0 (1)	140 (1)	189.0
7	1	64.0 (1)	48.0 (1)	--- (0)	189.0
8	1	79.0 (1)	88.0 (1)	200 (1)	81.0
Total 28 means		80.7 (12)	74.1 (19)	222 (14)	155.0
				Mean all workers	101.5

* Values in parenthesis indicate no. of records.

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STUDY OF THE EFFECTS OF AN INDUSTRIAL HAZARD

IN INDUSTRY:

I. Evaluation of Current and Past Hazards

A. Review of causes of death of:

employees who died while employed
employees who died after retirement
employees who died after changed jobs

Ideally, would like to know:

age at death
length and type of occupation in this particular industry
previous or later occupations and their duration
known medical conditions prior to death (hospital & medical records)
cause and contributing causes of death (death certificate)

B. Review of reasons for:

premature retirement)
job changes) in the industry
as a whole and by divisions within the industry related to exposure

C. Evaluation of present health status of current employees and comparison of their health status with that of similar men not exposed to asbestos. The control group would have to be carefully chosen to allow similar probability of illness and/or degeneration for all other reasons except exposure to asbestos. Demographic, historical, personal habits, socio-economic and medical care factors would have to be considered. Measurements of present health status used for comparison would include: medical history of morbidity, results of standardized physical examination, and objective measures of body function.

II. Evaluation of Effects of Control

1. Change in incidence
2. Reduction in progresssion of disease.

(a) An objective definition of abnormality would have to be defined.
(b) The prevalence of this abnormality would be determined.
(c) An estimate would be made of the median time of exposure in those who show this abnormality.
(d) The prevalence would be re-determined after a time approximately equal to the median time for development of the abnormality used as the criterion of change.

The incidence would be calculated.

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LITERATURE SUMMARY

Although we appreciate that the industrial physician is well aware of the medical problems associated with pulmonary asbestosis, it would seem proper to informally summarize some of the medical literature particularly as it relates to the case in question.

A. Pathology

Pathologic studies fall into two categories. The first, which make up the majority, are those which are concerned with the autopsy reports from humans who have pulmonary asbestosis and/or who have been exposed to asbestos inhalation. The second group are experimental animal studies. Both types of studies have certain inadequacies. The human studies suffer from the fact that they represent only the end stage of the disease and they do not give insight into the early developmental processes. The animal studies, on the other hand, do not necessarily parallel the human findings while they do give some insight into the development of the disease. Summarizing both together we can point firstly to the obvious discrepancy between the actual weight of silicates found in the lungs of asbestos workers at autopsy and the severity of the disease This simply illustrates that not all individuals respond to the same degree nor on the other hand are all asbestos fibers of equal potential toxicity. The primary pathologic lesions are of two types: 1) a nodular development in the small bronchioloes; and 2) a fibrosing lesion which can lead to the scarring and obliteration of alveolo-capillary areas. Even in the British populations where the incidence of centrilobular emphysema and chronic bronchitis are high the asbestosis workers demonstrate very little evidence of emphysema although occasional bullae and centrilobular changes are found. In at least one report, far advanced cases will tend to show a high incidence of bronchiectasis. The other classical characterisitics of the disease are the marked pleural changes and the development of both pleural and lung malignancies.

There is no simple way to measure progression of disease as a result of the momentum from initial disease development. As there is no ethical way to study exposed and unexposed defined cases to evaluate the variation in disease development, this method of evaluation will be mostly subjective and not as valuable as calculation of incidence rates at stated intervals after control is initiated.

IN THE COMMUNITY:

I. Mortality

Comparison of age and sex specific death rates from all and specific causes with those of the United States as a whole and with other similar communities not exposed to asbestos.

II. Morbidity

(a) Analysis of diagnosis for which social security is given before age of retirement. Examination of a sample of recipients to verify or add diagnoses.

(b) Search for morbidity data collected by the National Health Survey.

(c) Execution of a morbidity survey among a sample of the population to compare either with the results of the National Morbidity Survey or a survey among a selected control population.

(d) Comparison of diagnoses of age specific hospital admissions with published data or with a specially collected control sample.

To evaluate the effects of control or removal of any hazard--some or all--analyses would have to be repeated after a stated interval of time (probably 5 - 10 years).

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In summary, the primary pathologic lesion is a fibrosis at the level of the small lung radicals and alveolo-capillary surfaces, usually with minimal secondary obstructive and/or emphysematous changes. The fibrosing process seems to be directly related to the takeup of the asbestos fibers in the less than 10 microns in length size, by the alveolo macrophages and fibrocytes. There are some thoughts that the fibrosis can occur intermittently following the ~~succession~~ cessation of exposure due to the lamination and breakdown of asbestos fibers which have been enclosed in a proteinaceous material.

B. Clinical and ~~Radiologic~~ Radiologic Picture in Relation to Pathologic and Functional Change.

The clinical picture of these patients is usually that of "alveolo-capillary block" with the additional element that any respiratory disease which would be common to the community, i.e., chronic bronchitis, may cause a significant overlay. The classical clinical picture of alveolo-capillary block is that of a small lung with minimal physical signs, dyspnea at first on exercise, eventually at rest, accompanied by clubbing and cyanosis with right ventricular strain and eventual failure. It may be ~~important to~~ point out that such patients do not appear to be short of breath to the examiner as early in the disease as do patients with obstructive airway disease where the work of breathing utilizing accessory muscles of respiration is obvious. There are considerable discrepancies between the x-ray picture and the clinical symptoms on the ~~other~~ one hand and/or the respiratory function tests on the other hand. In general, one may say that the presence of an x-ray lesion strongly suggests a more severe disease. However, when one comes to the important question of progression of the disease either in the presence of continuing exposure or after cessation of exposure the x-ray becomes a less valuable tool. Clinical and physiological progression is not uncommon in the absence of x-ray change. Physiologic evidence of disease has been described in the presence of negative x-rays and would be anticipated. Again, x-ray, clinical, and physiologic severity are related to the length of exposure however, there are many examples in the literature of cases

of short term exposure, i.e., 18-24 months with severe disease.

The physiologic testing results are what one might expect from the pathologic results. These would be small total lung volume, an increase in compliance, a reduction in diffusing capacity, with either normal or borderline arterial oxygen tensions at rest which fall on exercise at least during the acute stage of the disease, and alveolar hyperventilation. On the other hand, obstructive airway disease plays a very small part and in this particular case one should note that tests such as the forced vital capacity and FEV-1 sec. are aimed primarily at determining obstructive airway disease. It is equally important to bear in mind that with the high prevalence of chronic obstructive airway disease which is present in the population of this country, probably secondary to cigarette smoking, that the identification of such disease by objective methods such as the measurement of airway resistance or conductance and residual volumes would be important in spparating its effect from that of asbestos.

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