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REPORT TO U. S. RUBBER COMPANY, ASBESTON PLANT, HOGANSVILLE, GEORGIA, REGARDING ASBESTOSIS IN EMPLOYEES AT THE ASBESTON PLANT, FOR THE ASBESTOS TEXTILE INSTITUTE MEETING

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The Asbeston Plant of the U. S. Rubber Company started operations in 1940. From its inception, there was cognizance of the potential medico-industrial hazard in working with asbestos fibers. Several factors militated against the possession of satisfactory knowledge regarding the nature, degree and extent of this danger.

Communications between different companies working with this fiber were handicapped by the requisite need for secrecy inherent in a highly competitive system of free enterprise. Adequate studies regarding the epidemiology, radiographic, clinicopathology changes and functional or physiologic had not yet in many instances been undertaken; marked the means by which particularly the latter could be accomplished even being developed. Moreover, as will be indicated later, and as has been suggested by others in the United States and England, there is reason to believe that not only has the nature of asbestos disease changed in the past eighty years; but also is probably different in varying places at present, because of such factors as type with location of operation within a given plant and the type of asbestos fiber predominantly used.

Awareness that there was a potential problem motivated the U. S. Rubber Co. in Hogansville, Georgia, to start routine annual chest X-rays on all personnel working in the Asbeston plant at the inception of its operations in 1940.

Additionally, detailed dust count studies at many stations within the plant have been continually performed over its twenty-one years of operation.

Information adduced from these data and films have subsequently proven invaluable in yielding some estimate of the approximate accumulative exposure to asbestos dust and its relation to the production of asbestos lung disease. Examination of serial roentgenograms of individuals has moreover enabled us to describe the radiographic picture of asbestos lung disease as it occurs at our plant and places us in a position of greater confidence in detecting early changes at a time when we hope removal of the worker from risk will prevent development of a pulmonary cripple and keep a trained worker on the labor market.

The present program was inaugurated in the Asbeston plant in Hogansville in 1957. Its goal was to develop all the information possible regarding the general nature of the asbestosis problem as it occurs at our plant and to detail, insofar as possible, the specific alterations in the individuals at risk. To this end all workers receive comprehensive annual examinations which include a detailed history and physical examination. (Male workers are advised to consult their family physicians for rectal examinations, and female workers are requested to consult their personal doctors for breast and pelvic examinations); a hemogram which includes white with differential count;

hemoglobin, hematocrit and red blood cell count. A sedimentation rate and urinalysis are also performed. Vital capacity has been measured by the McKesson-Scott apparatus and yearly skin test for tuberculosis and histoplasmosis have been done. In selected cases, sputa are collected for tubercle bacillus culture and about six patients have received bilateral bronchograms - with negative findings. Chest measurements at inspiration and expiration, height and weight and the routine vital signs are recorded.

Our effort then, is to see the patient as an individual and a whole, with emphasis upon the changes - if any - wrought on his physical and emotional being by occupational exposure to asbestos dust; to quantitate that exposure in dust count dosage and resultant radiographic alterations; and to more recently measure pulmonary physiologic performance.

Numerous factors seemed apparent in an initial report in 1958, and the passage of time has done little to alter initial tentative conclusions.

Dust exposure in our plant lessened more-or-less steadily with the passage of the years, following continuing efforts by responsible plant personnel to entrap, divert and exhaust the asbestos dust from the work area atmosphere.

Review and study of serial chest X-rays of workers who were beginning to develop pulmonary asbestosis in Hogansville demonstrated a more-or-less orderly progression of radiographic maturation from normal lung to diseased lung. This comparative study was particularly valuable in that it rendered more easily apparent the detection of the earliest radiographic changes of pulmonary asbestosis in this plant. The logical sequel of course, is early discovery of affected workers in order that they may be removed from risk before advanced and irreversible pulmonary changes have occurred.

In this connection, it seems pertinent to note that the progression of radiologic changes seems so far to be unique for this plant. We were privileged to consult with Dr. Kenneth E. Smith, Medical Director, Johns-Manville Company and Dr. John F. Knox, Medical Director of Turner Brothers Asbestos, Ltd., Rochdale, England who showed me X-rays of their workers with pulmonary asbestosis. The radiographic patterns of their asbestosis patients seemed to be of a far coarser and further advanced nature than our patients at comparable clinical levels of disease. The type and source of the involved asbestos fibers, dust size, and possibly dust velocity at time of inhalation may be some of the factors involved in the difference.

As a local convenience and method of utilizing available information relating dust count data and accumulative exposure, we developed the term MPPCFY meaning million particles (of asbestos dust) per cubic foot-year. We found the greatest exposure density of workers affected by pulmonary asbestosis to be around 50-60 MPPCFY's. A curve plotting asbestosis against MPPCFY shows a slow rise prior to this point and a tendency for asbestosis to become more predictably certain with increasing exposure. In order to keep a trained worker on the job, the conclusion seems inescapable then that at least one vital step involves as near as complete eradication of the dust as is possible.

Certain stations, such as weaving and winding, seem more at risk in our plant, but the changing work population and insufficient number of workers have prevented, so far, any valid statistical and predictive evaluations.

Furthermore, certain types of workers; namely supervisory personnel, have seemed more susceptible to the disease and the attrition among these valuable people has been relatively high. Speculation leads one to wonder whether the very attributes which drive certain workers to excell and become supervisors may have at the same time encouraged them to take greater risks, show more disregard of the dust hazard, and ultimately become casualities.

In regard to hazard and risk we have adapted the policy that knowledge of the risk and awareness that we are doing everything possible to minimize it and remove the worker from danger should he become an incipient victim is the most prudent course to follow.

From a laboratory standpoint, patients who develop the disease have tended generally to show rising values of hemoglobin, hematocrit and red blood counts. There have been no significant alterations in white blood count or differential values unless there has been a complicating infection. Vital capacities, as measured by the McKesson-Scott vital capacity apparatus, have shown universal decrease.

From a clinical standpoint the cardinal features have been onset of chronic low-grade non-productive cough, occasional sub-sternal and epigastric indigestion, and an insidious onset with progressive lessening of strength, pep and energy and a stealthily increasing shortness of breath on exertion. No one has complained of orthopnea or paroxysmal nocturnal smothering. Chest pain, except for fleeting and hard to describe almost neuritic symptoms, has been largely absent. Some patients with fairly well advanced pulmonary disease complain occasionally of difficulty in getting air into their chests. This symptom may be part of the hyperventilation syndrome, and subsequent respiratory studies have indicated that these patients almost uniformly have an increased minute volume of respiration.

Those with pulmonary asbestosis in our plant have invariably shown diminished chest and diaphragmatic excursion, fairly normal respiratory murmurs except for a tendency for the development of bronchovesicular sounds over the bases posteriorly and laterally; the typical finger changes of hypertrophic pulmonary osteoarthropathy without tenderness along the shafts of the lung bones; and increasing intensity of the second pulmonic sound suggesting rising tension in the pulmonary circuit. So far, none who show even moderately advanced pulmonary asbestosis (with one possible exception who was not observed by me) have demonstrated cor pulmonale manifested by right heart failure, radiologic change in hilar vessels or electrocardiographic changes of right sided strain. This seems somewhat strange but perhaps time will throw more light on this particular problem.

In order to learn more regarding the physiologic alterations of pulmonary asbestosis and to provide a more-or-less quantitative method of following these alterations we started about two years ago to study the volumes of the various physiologic lung compartments; their ventilation; the distribution of inspired gas as measured by the pulmonary nitrogen emptying rate

(7min test) and pulmonary nitrogen washout curves; and the character of the recorded spirogram.

More lately we have begun to study in these patients of ventilation-perfusion relationships and the diffusing capacity of the lungs for oxygen by the method of Riley and Cournand using two different levels of inspired oxygen concentration. The partial pressures of oxygen and carbon dioxide in arterial blood at these two levels are determined by the direct bubble method of Riley, Proemmel and Franke. The arterial blood content of oxygen and carbon dioxide and various gas analysis are measured in the Van Slyke manometric apparatus and arterial PH's are determined with the Beckman PH meter.

There are appended to this report three pages in tabular form showing in some detail the results of our studies on several patients with pulmonary asbestosis.

All have radiographic changes of varying degree, uniformly involving the lower lung fields and, as the disease progresses, the serosal surfaces of heart and lung. All have evidence of decreased chest excursion and many show decreased descent of lung bases to percussion. Breath sounds over the inferior portions of the lungs, usually posteriorly and often laterally and anteriorly are normal to bronchovesicular and frequently reveal fine to medium end-of-inspiratory sticky rales and occasional faint rhonchii.

Where the second aortic heart sound is louder than the second pulmonic, serial observations over several years have tended to reveal a changing relationship with decreasing aortic second and increasing pulmonic second sounds.

The majority have clubbing of varying degree—some pronounced, and some minimal, and there appears to be little correlation with the severity of the process.

Uniformly, there is some dyspnea on mild to moderate exertion and a general loss in sense of well being and usual strength and energy.

Vital capacities are almost uniformly decreased. Residual volumes tend to be slightly increased and total lung capacities, so far have tended to remain close to predicted normals. This may be because of increases in residual volumes, for the directly measurable inspiratory capacities and expiratory reserve volumes are uniformly decreased.

Minute volumes are almost uniformly increased and arterial CO_2 pressures tend to be low. Almost all have increased ventilatory equivalents for oxygen.

Maximal breathing capacities are uniformly decreased, although the figures given may be somewhat lower than the patients' full ability because the test has been performed in the semirecumbent position.

There is little evidence of airway obstruction and the pulmonary nitrogen emptying rates have been almost uniformly normal. Timed vital capacities have tended to show some lag at one second but normal emptying at two and three seconds.

Arterial oxygen saturations has been normal as have arterial partial pressures of oxygen at 21 percent oxygen in the ambient air.

Diffusing capacities for oxygen at rest have varied in four patients measured from 10.8 to 16.0 cc/mm/mm.

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