FILE NAME: General Electric (GE)

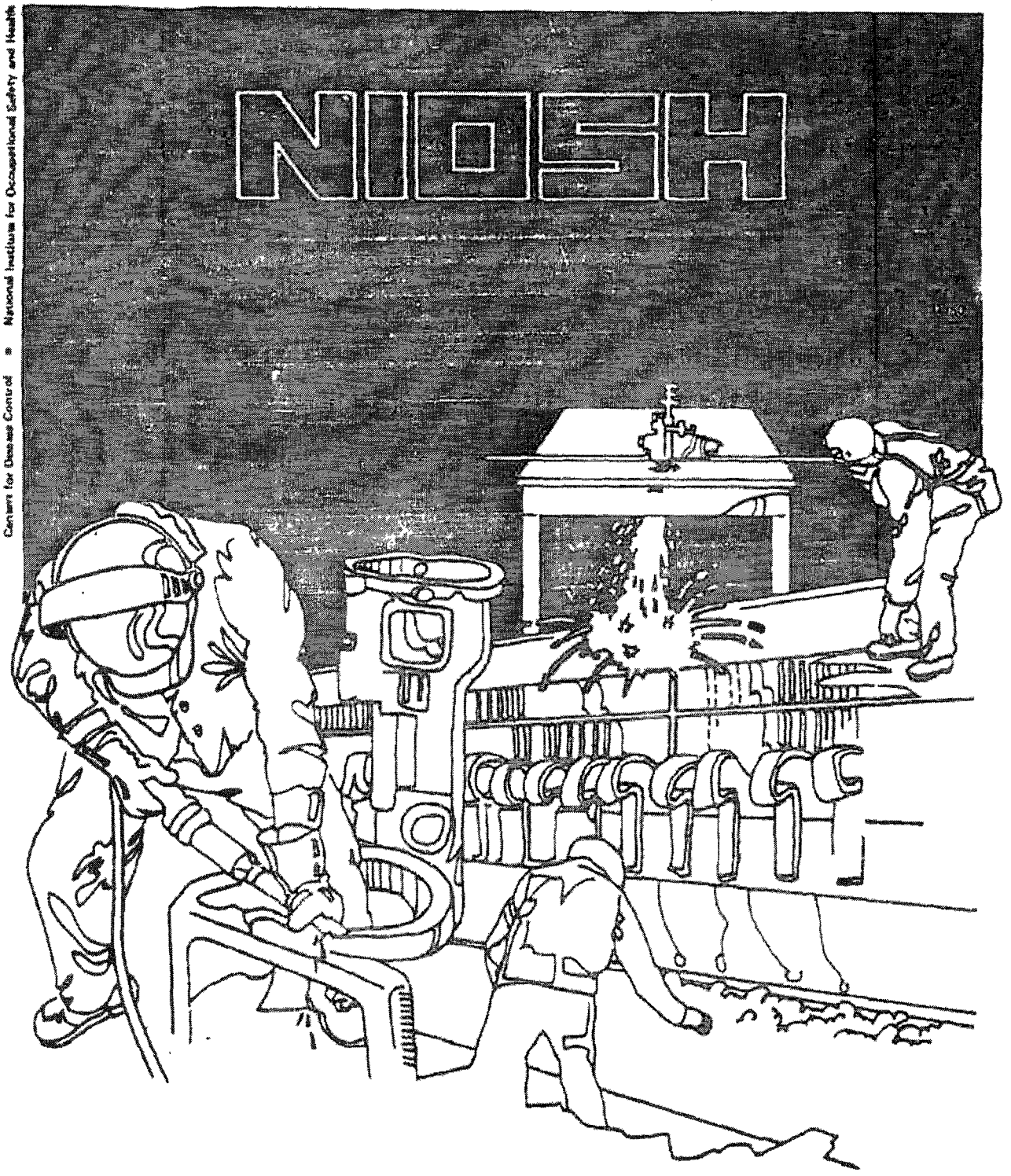
DATE: 1985

DOC#: GE204

DOCUMENT DESCRIPTION: NIOSH Health Hazard Evaluation Report - GE Plant, OH

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES • Public Health Service
Centers for Disease Control • National Institute for Occupational Safety and Health

NIOSH



Health Hazard Evaluation Report

HETA 83-044-1596
GENERAL ELECTRIC PLANT
EVENDALE, OHIO

PREFACE

The Hazard Evaluations and Technical Assistance Branch of NIOSH conducts field investigations of possible health hazards in the workplace. These investigations are conducted under the authority of Section 20(a)(6) of the Occupational Safety and Health Act of 1970, 29 U.S.C. 669(a)(6) which authorizes the Secretary of Health and Human Services, following a written request from any employer or authorized representative of employees, to determine whether any substance normally found in the place of employment has potentially toxic effects in such concentrations as used or found.

The Hazard Evaluations and Technical Assistance Branch also provides, upon request, medical, nursing, and industrial hygiene technical and consultative assistance (TA) to Federal, state, and local agencies; labor; industry and other groups or individuals to control occupational health hazards and to prevent related trauma and disease.

Mention of company names or products does not constitute endorsement by the National Institute for Occupational Safety and Health.

HETA 83-044-1596
JUNE 1985
GENERAL ELECTRIC PLANT
EVENDALE, OHIO

NIOSH INVESTIGATORS:
Roy G. Parrish, M.D.
Richard Hartle, C.I.H.
David Groth, M.D.

I. SUMMARY

On November 9, 1982, the National Institute for Occupational Safety and Health (NIOSH) was requested to evaluate the "local age" or heat treatment area of building 700 at the General Electric Plant, Evendale, Ohio. The requestor was concerned about four electricians who had worked in this area and were thought to have suffered adverse health effects from their exposure to an insulating material, possibly asbestos.

Thirty-three electricians have worked in the heat treatment process since its inception in 1957. The purpose of the process is to relieve metal stress which develops during manufacture of jet engines. The process has changed dramatically through the years, but in general, has consisted of heating either small, localized areas or large sections of a jet engine to a high temperature and then slowly cooling them. Between 1957 and 1965 and possibly later, the process utilized various insulation materials, including some containing asbestos.

General Electric electricians, industrial hygienists, and the NIOSH staff constructed a process history for heat treatment from interviews of 11 current or former General Electric electricians on January 13 and July 28-29, 1983. No official records of materials or the process prior to 1965 remain.

NIOSH obtained autopsy reports and post-mortem lung tissue from two deceased electricians who had worked in the heat treatment process between 1957 and 1965. One of these men died of metastatic colon cancer; the other died of necrotizing pneumonia superimposed on pre-existing pulmonary emphysema and fibrosis, the latter associated with pigmented material (probably containing asbestos). Lung tissue from both men was subjected to electron probe analysis for mineral fibers using a transmission electron microscope. Analysis showed 23 million fibers/gram of freeze dried tissue in one man and 15.5 million fibers in the other. The tissue contained 4.4 and 1.1 million amphibole asbestos fibers per gram dry tissue, respectively, representing significant occupational exposures. Mean amphibole fiber content in the lungs of non-occupationally exposed men, as reported in the medical literature, is 140,000 fibers/gram dry lung tissue (standard deviation of 100,000).

Based on these results, NIOSH determined that a health hazard existed, but no longer exists, from exposure to asbestos in the heat treatment process at General Electric. Both deceased electricians who spent time in the heat treatment process and whose autopsy findings were reviewed by NIOSH, had diseases associated with asbestos exposure (asbestosis in both and colon cancer in one). Recommendations for identifying and medical monitoring individuals potentially exposed to asbestos at the General Electric facility are contained in Section VIII of this report.

KEYWORDS: SIC 3724 (Aircraft Engines and Engine Parts, including Jet Propulsion), Electricians.

II. INTRODUCTION

On November 9, 1982, the National Institute for Occupational Safety and Health (NIOSH) received a confidential request to evaluate the "local age" area of Building 700 at the General Electric Plant, Evendale, Ohio. The request identified four electricians who had worked in this area who were thought to have suffered adverse health effects from their exposure to an insulating material, possibly asbestos, over the course of several years. In addition, others who had worked in this process were concerned about adverse effects on their own health in light of their exposure to the insulating materials. NIOSH visited the General Electric Plant on January 13, 1983, and distributed a letter about this visit on March 1, 1983. Preliminary results of electron probe analysis were described in a letter dated June 17, 1983. NIOSH conducted a follow-up medical survey on July 28-29, 1983, and sent out another letter on November 7, 1983, with the final results of the electron probe analysis.

III. BACKGROUND

The General Electric Company, Evendale, Ohio, is a large manufacturing facility which produces jet engines. It currently employs approximately 13,000 hourly workers.

In 1956 or 1957 a process called the "local age heat treatment process" was introduced to relieve stress that developed in metal parts during the manufacture of jet engines. The process consisted then, as now, of heating either small, localized areas or large sections of a jet engine to a high temperature and then slowly cooling them.

The local age heat treatment process was first located in Building 800 across from the nitride area and was used on the X-211 engine. On this engine the combustor, collector, and tubing were heat treated. The process involved cutting fire brick and shaping it to conform to the part(s) to be heated. Nichrome wire was used as the heating element and was inserted in the contoured section of the fire brick. This "furnace" was held to the part being heat treated by a tack-welded nichrome wire. Next, a number 20 chromalloy thermacouple with asbestos coating was tack-welded to the part to monitor the temperature during heating and cooling. After being cut to size, back-up insulation material was placed so as to cover the furnace. This insulation, which came in peach-colored sheets, was manufactured by the Johns Manville Company, and was thought to be composed of asbestos. Next, loose bulk insulation material, greyish-white in color, and also thought to be asbestos, was used to fill in around the furnace and back-up insulation. The bulk insulation was stored in cardboard boxes and was placed by hand or with shovels on top of the back-up insulation material to a depth of several inches. Finally, an asbestos blanket was used to cover the loose bulk insulation.

These materials were used from 1956 until mid 1958 when an olive-green colored material was added to the loose bulk insulation. This came in bulk containers also and was thought to have been fibrous glass. In 1959, the heat treatment area was moved to the southeast area of Building 700. Large X-211 parts were treated there. In about 1961, the heat treatment area was moved again, this time to the J6 area of Building 700.

From 1959 until 1966, the materials used for heat treatment were identical to those used in Building 800, as described above. About 1965 or 1966, vermiculite became the major loose, bulk-insulating filler. It was stored in garbage cans. Fiberfrax was introduced in 1965 or 1966 as the blanket covering the bulk insulation. After 1975, the use of loose insulation was discontinued with the replacement of nichrome wire heating coils with a quartz lamp.

No records remain that give the composition, trade name, or manufacturer of the insulation products used prior to 1965. To the best of their knowledge, neither the electricians, industrial hygienists, nor medical staff at General Electric felt that the electricians would have had any potential for routine asbestos exposure at General Electric other than in the local aging heat treatment area.

Since the inception of the heat treatment process, approximately 30 to 35 electricians have worked on the process. This job was traditionally defined as an electrician's job. The amount of time spent by any one electrician on the process varied considerably, depending on the work-load. During the early years of heat treating, electricians frequently worked overtime on this process and several worked in heat treatment at the same time because of the large size of the engines. The electricians said that the insulation material was loose and very dusty, and in the course of covering and uncovering the parts to be heat treated, large amounts of the dust became suspended in the air and were inhaled. The electricians moved the insulation material with their hands or shovels when packing it around the engine parts.

IV. MATERIALS AND METHODS

A. Environmental

Settled dust samples were collected from relatively inaccesible locations at all areas within the facility where the heat treatment process had been located. These samples were analyzed for asbestos by polarized light microscopy and dispersion staining techniques in hopes of gaining information on fiber types and size. Samples of the current materials were also obtained and analyzed for asbestos.

B. Medical

The NIOSH medical officer obtained and reviewed the autopsy reports that were available on two of the four electricians from the heat treatment area who were known to have died. Also, the remaining autopsy material, including paraffin blocks, slides, and wet tissue were obtained from the pathologists who had autopsied these two electricians. This material was reviewed by the NIOSH medical officer and a NIOSH pathologist. The NIOSH pathologist performed electron microscopic analysis of lung tissue from both men for fibers. Due to the small amount of tissue available from each worker, the wet tissue and tissue from the paraffin blocks were pooled for analysis. Given the variation of fiber concentration that can be seen in different parts of the same lung[1], this pooling would be expected to produce a result more representative of the fiber concentration in the lung as a whole than would tissue from a single site. The tissue was freeze dried, ashed in a low-temperature asher, suspended in water, placed on a filter, and examined by transmission electron microscopy.

During the follow-up medical survey, the medical officer administered a questionnaire to 12 of the General Electric electricians who had worked in the heat treatment process. It consisted of work history, (concentrating on time spent in the heat treatment process) and a limited medical history. Through interviews with electricians, company and union officials, we identified 33 electricians as having worked in the heat treatment area between 1956 and 1983. Of these, 11 completed questionnaires. Nine others still worked at General Electric but did not complete the questionnaire. Twelve of the remaining 13 electricians were either dead or retired, and one could not be located. The purpose of the questionnaire was to obtain exposure information and any recollections that the electricians had of the process or the materials that they had used in the process. It was not intended to be a detailed medical history.

V. EVALUATION CRITERIA

The Occupational Safety and Health Administration (OSHA) standard for asbestos limits exposure to 2 fibers/cc > 5um in length averaged over an 8-hour workday with a ceiling concentration of 10 fibers/cc not to be exceeded over a 15-minute period. There is also a provision for the medical monitoring of workers routinely exposed to levels in excess of 0.1 fibers/cc. This exposure standard was devised to minimize the risk of developing asbestosis. OSHA is presently going through the rule-making process to lower this standard. The American Conference of Governmental Industrial Hygienists, and independent scientific body, also recommends chrysotile asbestos exposures be limited to 2 fibers/cc over an 8-hour workday (with the notation that asbestos is a human carcinogen).

NIOSH currently recommends that exposure to asbestos (of all fiber types) in occupational environmental environments be kept to the lowest feasible level that can be reliably determined. This corresponds to 0.1 fibers/cc > 5um in length, which is the concentration that can be quantified using the PCM method. This recommendation is based on the absence of a threshold dose for asbestos below which no cancers occur. Therefore, NIOSH believes there is no "safe" level of exposure to asbestos for the elimination of all cancer risk. Thus, any exposure results in some increased health risk.

Asbestos is the name applied to a family of naturally occurring, flexible, fibrous hydrous silicate minerals that are relatively indestructible and heat resistant. Although fibrous minerals are found everywhere in the earth's crust, only a few are of commercial importance: (1) chrysotile (from serpentine rock), and (2) crocidolite, amosite, and anthophyllite (classified as amphiboles).

Asbestos is a well-known human carcinogen. In addition, it produces a non-malignant lung disease known as asbestosis. Many studies have documented the association of cancer and asbestosis with asbestos exposure (5-11).

The type of cancer most commonly associated with asbestos exposure is lung cancer. The relative risk of developing this cancer is approximately five times greater in those exposed to asbestos than in non-exposed individuals. Asbestos and cigarette smoking have a synergistic effect in producing lung cancer. In addition, a rare tumor known as malignant mesothelioma occurs almost exclusively in people exposed to asbestos.

Colon cancer has also been associated with asbestos exposure. In the most thorough and widely respected epidemiological studies of asbestos exposed workers and their causes of death, Selikoff et al.[6,8,9,10] showed that asbestos exposed workers had an excess of cancer of the colon with a relative risk ranging from 1.6 to 3.0 as compared to non-exposed workers. Other studies[5,7] have shown a two-to three-fold increased risk of colon cancer in asbestos exposed-workers. Selikoff et al.[10] have noted a latency (the time from first asbestos exposure to the diagnosis of clinical disease) of approximately 20 years before the appearance of excess risk of colon cancer.

Asbestosis is a form of pulmonary fibrosis secondary to the accumulation of airborne asbestos in the lungs. The severity of the disease varies. It depends in large part on the type and duration of exposure, the concentration of dust in the ambient air, and the fiber "burden" of the lung. Factors unique to the host also are believed to be important, although their relative significance in the evolution of the disease is uncertain. Asbestosis is a chronic and often progressive disease. Although decades usually elapse before it becomes clinically evident, shorter latency periods have been noted in asbestos workers experiencing exceptionally heavy exposure.[11]

In 1980, Churg and Warnock[2] reported on their electron microscopic study of asbestos fiber content in the lungs of 21 non-occupationally exposed urban dwellers. Eleven of these were men; they had chrysotile fiber contents of 120,000 fibers/gram wet lung tissue with a standard deviation (SD) of 110,000. (Ten grams of wet lung tissue corresponds to approximately one gram of dry lung tissue.) Their amphibole fiber content was 14,000/gram wet lung tissue (SD of 10,000). Eighty percent of the asbestos fibers were chrysotile and > 90% of the chrysotile was < 5 microns long. Ninety-five percent of the amphiboles were non-commercial, and two-thirds were < 5 microns long. However, 20 percent of the commercial fibers, amosite/crocidilite, and 20 percent of the anthophyllite fibers were > 10 microns long.

In a 1982 paper, Churg[3] commented on fiber content in patients with various types of asbestos-related disease. He noted that the number of asbestos bodies markedly underestimates the total number of fibers in the lung. In the general population the mean number of asbestos fibers is about 1,000,000/gram of dry lung tissue. Of these, > 80% are chrysotile fibers < 5microns long. Patients (without occupational asbestos exposure) in the general population who have pleural plaques have approximately the same number of total fibers, but their lungs contain about 50 times as many thin amphibole fibers of commercial origin. In addition, patients who have asbestosis and most patients with mesothelioma, have 100 to 200 million asbestos fibers/gram dry lung tissue. Churg noted that the grade of asbestosis appears to be related to the total fiber content. However, occasional patients may develop mesothelioma with much smaller fiber burdens. Both benign and malignant pleural disease appear to be closely related to the presence of long, thin amphibole fibers. Finally, based on the above observations, he concluded that asbestos-related disease depends not only on the total number of fibers present, but also on such factors as fiber type and size.

In a recent paper, Churg and Wood[1] described their findings that there may be a marked variation in the asbestos and non-asbestos fiber concentration from area to area within a given lung. They determined this by taking small adjacent samples from the peripheral regions of three normal and six asbestotic lungs and analyzing the asbestos and non-asbestos fiber content. In both types of lungs, they found a marked variation in fiber concentration from site to site. For example, within the same lung adjacent pieces of tissue yielded as much as a seven-fold difference in fiber concentraton. They also found that both the different fiber types, as well as the asbestos bodies and fibers, appear to vary independently in concentration throughout the lung.

VI. RESULTS

A. Environmental

Of 11 settled dust and material samples collected and analyzed for asbestos, only one contained asbestos (20-25% chrysotile). This sample was obtained from an overhead cold water insulation jacket located at the current heat treatment area to assess the possibility that settled dust samples contained asbestos from sources other than process materials.

B. Medical

The work histories provided by the electricians formed the basis of the previous description of the local age heat treatment process (Section III). From these work histories, the period with the greatest potential for exposure to dust that probably contained asbestos was 1957 through 1965.

Based on the available General Electric records and the electricians' accounts, both of the deceased electricians, whose tissue was analyzed, worked in local age between 1956 and 1965. It is not possible to determine the exact period nor the duration of work (i.e., hours per day, days per week) in local age as neither exposure records nor specific work histories in local age were kept by General Electric.

Review of the medical and autopsy records that were available to NIOSH showed that one of the deceased electricians had cancer of the colon metastatic to lung and several other sites. In addition, he had acute bronchial pneumonia, emphysema most pronounced in the left upper lobe, and several areas of interstitial fibrosis associated with pigmented materials. Based on the electron microscopy study, the pigmented material probably contained asbestos and other mineral fibers. Thus, the interstitial fibrosis would be asbestosis. This worker's exposure to the heat treatment process appears to have preceeded his death by at least 20 years. Electron microscopy showed 23.0 million fibers per gram of freeze dried lung tissue. Included in that count were 3.3 million amosite fibers; 1.1 million other amphibole asbestos fibers; 1.6 million chrysotile fibers, 4.3 million titanium and titanium containing fibers, 7.6 million silicate fibers with very high aluminum content (Al/Si > 1.0) and smaller amounts of magnesium, iron, and calcium; 2.7 million silicate fibers with a moderate aluminum content (Al/Si = 0.2 - 1.0) and smaller amounts of magnesium and iron; and 3.3 million other silicate fibers.

The second worker died of acute necrotizing, hemorrhagic pneumonia with secondary recent thromboemboli. In addition, he had pulmonary fibrosis associated with anthracotic pigment (probably containing asbestos based on results of electron microscopy), chronic pleuritis, and acute and chronic bronchitis. His medical records showed that he had been severely disabled by lung disease for several years prior to his death; several pulmonary function tests showed severe obstructive disease. Due to lung-related medical disability, he retired from work at General Electric in 1977. The necrotizing pneumonia, which was the cause of his death, probably was a complication of his underlying lung disease. His freezed-dried lung tissue had 15.5 million fibers per freeze-dried gram. Included in that count were 2.1 million amphibole asbestos fibers, 4.1 million titanium containing fibers; 4.7 million fibers with very high aluminum content (Al/Si > 1.0) and smaller amounts of iron and calcium; 2.6 million fibers with a moderate aluminum content (Al/Si = 0.2 - 1.0) and smaller amounts if iron, calcium, and magnesium; and 1.6 million other silicate fibers.

The available pathology data are limited to those two former electricians whose lungs were analyzed by both light and electron microscopy. Both men's lungs showed areas of interstitial fibrosis associated with pigmented material probably containing asbestos. Regrettably, the tissue samples obtained at the time of autopsy, were not sufficient to allow diagnosis and grading of asbestosis using the criteria of the College of American Pathologist's.[11]

VII. DISCUSSION AND CONCLUSIONS

Although no asbestos was detected in the settled dust samples, this does not rule out the historical use of asbestos in the heat treatment process. The sites of sample acquisition may have been cleaned during the time since the heat treatment process had moved, or the fiber concentrations may have been too low to detect.

The electron probe analysis supports the following conclusions:

1. Both men had significant occupational exposure to asbestos, as well as other natural and synthetic mineral fibers. These exposures resulted in an accumulation of fibers in their lungs to levels approximately ten times higher than those found in non-occupationally exposed urban men. In addition, amosite fibers are frequently found in occupational and not non-occupational asbestos exposure settings.

2. The types and distribution of fibers in both men were similar. Since the distribution and types of fibers were unusual, both men probably received their exposure from a common source, i.e., during work at General Electric in the heat treatment process area and not from pre- or post-General Electric employment. The process history and work histories obtained from other General Electric electricians in July 1983 identified the heat treatment process area as the work area associated with the most significant potential exposure to asbestos for General Electric electricians.

3. The positive analysis for asbestos confirms that asbestos was used in the heat treatment process area and suggests that any electrician who may have worked in that area probably had a significant exposure to asbestos. Clearly the extent of exposure depends upon the time spent in the heat treatment area and the years during which this exposure occurred. The years 1957-1965 appear to be the ones with the greatest potential for exposure to asbestos. It may have been used from 1965 to 1975, but there is less supportive evidence for this period. Nevertheless, in the absence of environmental or product information concerning the materials used, the possibility of exposure to asbestos subsequent to 1965 cannot be excluded.

Transmission electron microscopy was essential in determining whether occupational exposure to asbestos and various mineral fibers occurred since it was not possible to sample the process as carried out in past years and since there were no purchase orders for materials or product information for the early years of the process. It provided the evidence to confirm the impression of electricians that they had worked with asbestos at General Electric during the early years of the heat treatment process.

Since other General Electric electricians may also have had significant exposure to asbestos 20 or more years ago, this group should be carefully followed for development of asbestos related disease.

Based on discussions with General Electric industrial hygienists, electricians, and union representatives, it appears that other employees at General Electric may also have been exposed to asbestos. These employees include those in the following jobs:

1. Plumbers and pipe fitters. The finding of asbestos in insulation surrounding a cold water pipe during the initial survey confirms that asbestos insulation has been used in pipe insulation at General Electric.

2. Air conditioning repair and maintenance workers.

3. Office workers in the basement of Building 500.

4. Millwrights.

5. Sheetmetal workers.

6. Maintenance workers.

7. Masons and carpenters.

8. Some machinists.

VIII. RECOMMENDATIONS

Efforts should be made to identify occupational groups with potential asbestos exposure, determine the extent of their exposure, notify them of their exposure, and institute appropriate medical screening for asbestos related disease, where indicated.

1. General Electric and employee representatives should work together to survey job categories to determine which employees at General Electric have had asbestos exposure.

2. These exposed employees, along with the electricians identified during the course of this health hazard evaluation, should be notified of their exposure.

3. A periodic screening program for asbestos exposed employees should be instituted to detect asbestos-related disease present now or appearing in the future. The screening program should include medical history and physical examination, periodic chest roentgenograms, and pulmonary function tests, including diffusion assessment. Stool guiac, sigmoidoscopy, and sputum cytology may also be useful.

4. An education program to notify asbestos exposed employees of their exposure and their increased disease risk should be provided.

5. A smoking cessation program should be targeted at those employees with confirmed exposures.

IX. REFERENCES

1. Churg A., Wood P., Observations on the Distribution of Asbestos Fibers in Human Lungs. Environ Res 1983;31:374-80.

2. Churg A, Warnock M L. Asbestos Fibers in the General Population. Am Rev Respir Dis 1980;122:669-78.

3. Churg A. Fiber Counting and Analysis in the Diagnosis of Asbestos-related Disease. Human Pathol 1982;13(4):381-92.

4. Churg A, Warnock M L. Asbestos and other Ferruginous Bodies: Their Formation and Clinical Significance. Am J Pathol 1981;102:447-56.

5. Miller AB. Asbestos Fiber Dust and Gastro-intestinal Malignancies. Review of Literature with Regard to a Cause/effect Relationship. J Chronic Dis 1978;31:23-33.

6. National Institute for Occupational Safety and Health. Workplace Exposure to Asbestos. Cincinnati: National Institute for Occupational Safety and Health. DHHS (NIOSH) Publication No. 81-103.

7. Schneiderman, MA. Digestive System Cancer Among Persons Subjected to Occupational Inhalation of Asbestos Particles: A literature Review with Emphasis on Dose Response. Environmental Health Perspectives 1974;9:307-11.

8. Selikoff IJ. Cancer Risk of Asbestos Exposure. In: Hiatt HH, et. al. eds. Origins of Human Cancer. Vol 4. Cold Spring Harbor Laboratory: Book C, 1977:1765.

9. Selikoff IJ, Hammond EC, Seidman H., Mortality Experience of Insulation Workers in the United States and Canada. 1943-1976. Ann NY Acad Sci 1979;330:91-116.

10. Selikoff IJ, Hammond EC, Seidman H., Latency of Asbestos Disease Among Insulation Workers in the United States and Canada. Cancer 1980;46:2736-740.

11. Pneumoconiosis Committee of the College of American Pathologists and the National Institute for Occupational Safety and Health. Asbestos-Associated Disease. Arch Pathol Lab Med 1982;106(11):541-96.

12. Executive Office of the President. Standard Industrial Classification Manual. Washington, D.C.: Office of Management and Budget, 1972.

13. Office of Federal Statistical Policy and Standards. Standard Occupational Classification Manual. Washington, D.C.: Department of Commerce, 1980.

14. Employment and Training Administration. Dictionary of Occupational Titles, Fourth Edition. Washington, D.C.: Department of Labor, 1977.

IX. AUTHORSHIP AND ACKNOWLEDGEMENTS

Report Prepared by: Roy G. Parrish, M.D.
Medical Officer
Medical Section, HETAB, DSHEFS

Richard Hartle, C.I.H.
Industrial Hygienist
Industrial Hygiene Section, HETAB, DSHEFS

David Groth, M.D.
Chief, Pathology Section
Technical Support Branch
DBBS

Originating Office: Hazard Evaluations and Technical Assistance Branch
Division of Surveillance, Hazard Evaluations, and Field Studies

Report Typed By: Stephanie M. Harris
Secretary
Medical Section

X. DISTRIBUTION AND AVAILABILITY OF REPORT

Copies of this report are currently available upon request from NIOSH, Division of Standards Development and Technology Transfer, Publications Dissemination Section, 4676 Columbia Parkway, Cincinnati, Ohio 45226. After 90 days, the report will be available through the National Technical Information Service (NTIS), 5285 Port Royal, Springfield, Virginia 22161. Information regarding its availability through NTIS can be obtained from NIOSH Publications Office at the Cincinnati address. Copies of this report have been sent to:

1. General Electric Company
2. Union, IAUAW
3. Confidential Requestor
4. NIOSH, Region V
5. OSHA, Region V

For the purpose of informing affected employees, copies of this report shall be posted by the employer in a prominent place accessible to the employees for a period of 30 calendar days.