

FILE NAME: DuPont (DUP)

DATE: 1966 Aug

DOC#: DUP162

DOCUMENT DESCRIPTION: Journal Article - Occupational Hazards of Pipe Insulators

Occupational Hazards of Pipe Insulators

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THE DEATH of a member of the International Association of Heat and Frost Insulators and Asbestos Workers, resulting from a *Bacillus anthracis* infection, focused attention on the potential occupational hazards of this trade. An investigation of the work which these men perform and the materials with which they come in contact was undertaken in order to determine the nature of the hazard, if any, of this trade. To initiate the study, all materials used by union members in the Cincinnati area were first reviewed and evaluated. These materials are divided into five major categories (A to E) according to their chemical composition. Each of these will be dealt with individually.

An analysis of the job indicated that the handling of fibrous glass, magnesium carbonate and calcium silicate materials containing asbestos fibers, adhesives, and cements occupies 80% to 90% of the average insulator's working time. Accordingly, these materials were of particular interest and have been reviewed in detail within the framework of the major category in which they reside.

Glass Insulating Materials

The term glass has traditionally been linked, in the minds of men, with lacerations and other forms of physical injury. In addition to such physical insults, there is reason to be concerned with the possible effects from the ingestion and inhalation of airborne particles of glass, since glass insulating materials are produced in forms that may give rise to airborne fibers and flakes of glass.

Review of Literature Concerning Glass Fibers.—Glass fiber materials were first developed by Owens-Corning Fiberglas Corporation in 1932, and in 1938 this company opened the first plant in the world for the production of fibrous glass.

Glass fibers are silicates and have the following general composition:

Silicon dioxide (SiO ₂)	54-65%
Oxides of aluminum	1-15%
Calcium (Ca)	0-22%
Magnesium (Mg)	0-6%
Boron	0-10%

Others additives: sodium (Na), phosphorus (P), iron (Fe), barium (Ba), titanium (Ti).

A phenol formaldehyde resin is applied to the glass fibers to bond and hold them in boards, mats, or wool-like masses. The resin is bonded securely to the glass fiber by heating to temperatures ranging from 450 F to 550 F, in order to prevent its removal from the glass and vice versa. Thus, the resin is not an airborne hazard in itself. Glass fiber insulating materials have the following properties: they are incombustible and resistant to high temperatures (600 to 700 C); they are nonhygroscopic, impuscescible, and do not deteriorate; they are stable, not attacked by oils, corrosive fumes and acids (except hydrofluoric); they are not attacked by parasites, mites, termites, or rodents; they are resistant to traction, but are not elastic and have little resistance to friction and impact. Glass fibers commercially serve as thermal, electrical, and acoustical insulators.

Under the influence of sufficient friction or impact, the insulating material shatters into small glass fibers of varying lengths with jagged ends. Some of these fibers are of sufficiently small size to remain airborne for appreciable periods of time, thus

Submitted for publication Aug 20, 1965; accepted Nov 11.
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Reprint requests to Kettering Laboratory, College of Medicine, Eden and Bethesda Avenues, Cincinnati 45219 (Dr Zavon).

Malignancy as the Cause of Death From Jan 1, 1939 to Jan 1, 1964

Insulator	Cause of Death	Years in Trade
1.	Bronchiogenic carcinoma	28
2.	Bronchiogenic carcinoma	17
3.	Carcinoma of lung	38
4.	Metastatic carcinoma of brain	42
5.	Generalized carcinomatosis	29
6.	Carcinoma of tongue	41
7.	Carcinoma of caecum	24
8.	Carcinoma of sigmoid	42

providing a possible source of particles for ingestion and inhalation.

Glass fibers in various insulating materials will have the following average diameters:

Insulating Material	Diameter (μ)
Building insulation	7.11
Pipe insulation	10.7
Duct insulation	5.84
Acoustical products	13.21

The fibers listed above range from approximately 5μ to 14μ in diameter, and under normal conditions their minimal lengths will range from 8μ - 12μ to 60μ - 90μ . Most fractured fibers do not reach these minimal lengths, and in numerous atmospheric surveys of fiberglass manufacturing plants in areas involving the production and handling of various products made from finished glass fibers, only 6% of all airborne fibers were less than 20μ in length and only 40% were less than 100μ in length.¹

Airborne glass fibers are too large to enter the alveoli. Their tendency, unlike the fibers of asbestos, is to form felt-like masses on any object with which they come in contact, and this probably aids in preventing their inhalation. Thus, it is the consensus of opinion of many investigators that airborne glass fibers present no lower respiratory hazard.¹⁻⁸

As stated previously, glass fibers are silicates, but contain no free silica (SiO_2) or free sodium silicate, thus do not give rise to silicosis. No harmful vapors or fumes can be given off, since no substances are present which might volatilize within the range of temperature recommended for service.⁵

There have been numerous investigations of plants and of employees engaged in producing and handling glass fibers. The majority of investigators agree that glass

fibers give rise to a moderate amount of irritation to the upper respiratory system, but have no adverse effects on the lower respiratory system.^{2,11,12} One of the most recent and thorough studies took place in 1963 at the world's oldest production plant for glass fibers, located in Newark, Ohio, in which active commercial production began in 1938. In 1963 this plant produced 250,000,000 pounds of glass fibers and employed 1,352 men and 113 women with more than ten years of company service. Clinical surveillance of these workmen throughout the period in which the manufacturing processes have been in operation, in conjunction with the examination of serial x-ray films of these workmen, have failed to reveal the existence of any form of respiratory disease which can be related to glass fibers.¹

It appears from the data thus far assembled that the airborne spicules of glass fiber to which insulators would be exposed in their work present no pulmonic hazard.

A workman's initial exposure to glass fibers gives rise to a temporary cutaneous irritation and pruritus, but does not induce sensitivity. Punctiform erythema appears promptly after contact with glass fibers, and usually disappears after the workmen discontinue such exposure. Occasionally the erythema persists after the itching has ceased, and may not disappear completely until several days later.¹³ Many experiments with animal and human subjects have revealed that these microtraumas are due to mechanical irritation and are very superficial, localized strictly in the corneal layer of the skin.^{2,4,12-18} In biopsies taken from irritated sites, examinations with both incandescent and polarized light have not enabled the investigators to locate glass spicules or foreign body reactions in the epidermis or dermis.¹⁶ The pruritus increases with perspiring and rubbing. Pruritus appears primarily on wrists, ankles, neck, and at other areas where clothing fits closely to the body and enables the little glass fibers to accumulate. They penetrate the fabric of clothing and irritate the skin causing slightly painful stimuli which are interpreted as an itching sensation. Further experimentation has revealed that the severity of this super

ficial cutaneous reaction varies directly with the size (diameter) of the glass fibers being handled. This finding has been confirmed in all interviews conducted with insulators on plant visits.¹⁶ The reactions of workmen to glass fibers are highly variable; the degree of reaction may vary from day to day with changes in temperature, humidity, type of clothing worn, and other preexisting abnormalities of the skin.^{10,19}

Apparently, insulators may undergo superficial, transitory cutaneous irritation and pruritus, but do not develop a true dermatitis.

One additional testimony to the benign composition of glass fibers is the large number of medical and related uses of the material in intimate contact with both normal and severely damaged tissue. Glass fiber materials are used as bandages, skin graft material, wound dressings, tracer threads for surgical sponges, prosthetic appliances, blood plasma filters, extra-pleural packing, American Medical Association approved glass fiber filled pillows, and in dental work.^{1,20-24}

View of the Toxicity of Glass Flakes.—

The second type of airborne, particulate glass, is that which occurs in the form of flakes and arises from the handling of an insulating material known as foam glass. These flakes may be ingested, inhaled, and deposited on the skin and in the external ocular space. An experiment with animals was conducted to determine the effect of glass flakes, less than 50 μ in size, on any organ with which they might come in contact by one of the above mentioned means.¹⁰ It was found that these flakes were removed effectively from the eye by blinking and did not give rise to corneal injury. After the animals had ingested a diet containing 50% (by weight) of glass flakes, all of their organs were examined grossly and microscopically. No abnormalities were observed. There was no evidence of trauma, ulceration, or degeneration of the gastrointestinal mucous membrane. Both intratracheal injection and inhalation of flakes gave rise to a cellular reaction in the alveoli which was no more severe than that of finely divided glass which was being used as a control.

Not all glass flakes encountered by insulators will have such small dimensions as to

be respirable. However, the above experimental evidence is cited for completeness and to account for any unforeseen situation in which particles of such small dimensions may be generated.

It seems evident that glass insulating materials present no major health hazard to the members of the insulating trades in the normal performance of their occupation.

Asbestos-Containing Materials

History and Properties of Asbestos.—The second major category of insulating materials is the group containing asbestos. Asbestos is one of the oldest insulating materials in the world. Geologically, asbestos is a term applied to a number of different minerals of which the characteristic feature is a structure composed of long, parallel, flexible fibers. This structure is unique, because the fibers are capable of repeated longitudinal subdivision into units of molecular proportions. The name asbestos embraces some 30 minerals of fibrous crystalline structure, all resembling each other. These similar minerals are divided into two major groups; the amphibole group, containing the commercially important actinolite, amosite, amphibole, anthophyllite, crocidolite and tremolite, and the serpentine group, containing chrysotile.²⁵⁻²⁸

Chemically, asbestos is a magnesium silicate. The average composition is a combination of magnesium oxide (MgO), silicon dioxide (SiO₂), ferrous oxide (FeO), aluminum oxide (Al₂O₃), and water (H₂O). The bulk of the asbestos of commerce is chrysotile (3MgO · 2SiO₂ · 2H₂O), which is mined in Quebec, Canada. Other major sources are South America, Russia, and Rhodesia.

Asbestos is incombustible, separates into filaments of fibers, possesses unusual flexibility and high tensile strength, is resistant to heat, moisture, and to the corrosive action of acids.^{28,30}

In 1880, the annual world production of asbestos was only 500 tons, in 1942 it was 500,000 tons, in 1951 it was 1,300,000 tons, and by 1962 it had risen to 2,400,000 tons.²²

²¹ This tremendous increase in production has resulted from an equally large increase

in the variety of uses and the distribution of the material.

Review of the Literature Concerning Asbestos.—The first report of a death resulting from the inhalation of asbestos was recorded in 1900.²² In the next quarter century, many investigations have been performed and have definitely linked prolonged exposure to asbestos to the development of asbestosis, a diffuse pulmonary fibrosis. The risk varies directly with the duration of the exposure and is unaffected by the age of the worker. Following continued exposure to high concentrations of airborne asbestos, the disease may develop fully in seven to nine years, and may cause death after about 13 years. Pulmonary fibrosis, as it occurs in asbestos workers, differs considerably from that which occurs in workers exposed to free crystalline silica dust; it differs radiologically and also on postmortem examination of the affected lungs.^{29,32-34} Exposure to asbestos does not give rise to systemic poisoning, biochemical abnormalities, or allergic reactions such as asthma and skin rashes.²⁸ One of its results is a curious clinically observable particle in the pulmonary tissue, and in the sputum, called the "asbestos body." This body was first observed in the sputum in 1906, and later directly linked with exposure to asbestos.³⁵ Asbestos bodies may be found in the lung, pleura, lymph nodes, and spleen at necropsy.³⁶ They are variable in size, color, and form and have been produced experimentally in animals.^{37,38} The finding of asbestos bodies in the sputum is only indicative of the fact that previous inhalation of asbestos has occurred, and although they are usually more numerous during current exposure, they may be found for years after the cessation of such exposure. Conversely, even in advanced asbestosis of long duration, they may not be found at all or only in sparse numbers.^{32,35,36,38-41}

Animals have been subjected to the inhalation of air bearing dispersions of asbestos and have developed pulmonary lesions similar to those observed at autopsy in men suffering from asbestosis.^{26,28,42,43}

The initial pathological change, peribronchiolar fibrosis, was found to be produced primarily by fibers 20 μ to 50 μ in length,

but not by fibers less than 20 μ in length.^{28,41} These facts are accepted by the majority of investigators, although they are contrary to all other concepts of pathogenicity of other airborne solid particles, in that here the larger fibers produce the pulmonary damage. To the extent that there is disagreement, only further experimentation will resolve the question.⁴⁴ The flexible filamentous structure is believed to be an essential factor producing the irritating action. All other filamentous particulates (glass fibers) are solid and inflexible and do not produce fibrosis.²⁸ Once asbestosis was verified as a specific disease entity, surveys of the asbestos textile industry were performed to determine tolerable limits of atmospheric contamination.^{27,45,46} Here clear-cut cases of asbestosis were found in association with prolonged exposure to concentrations in excess of 5,000,000 particles per cubic foot (mppcf), but none were found in situations involving prevalent lower levels of contamination. Thus, the level of 5 mppcf has been recommended as tolerable without qualifications as to the length of asbestos fibers dispersed in the air.^{27,28}

During the last quarter century much epidemiological evidence has been accumulated to link exposure to asbestos with carcinoma of the lung.^{32,37,41,47-54} The reality of a relationship between carcinoma of the lung and such exposure was recognized formally at the International Symposium on the Endemiology of Lung Cancer (Council of the International Organizations of Medical Sciences, 1953). More recently additional surveys have been conducted to reveal any relationship which might exist between exposure to asbestos and the occurrence of mesothelioma (a primary malignant tumor arising from serous surfaces). The results of these surveys will appear later in this article.

Many surveys have been carried out in an attempt to determine the incidence of lung cancer in relation to asbestosis.^{26,49,55} The most reliable evidence indicates that from March 1924, until December 1955, the incidence of lung cancer in human asbestosis was 17.8%.⁵²

Even with the wealth of information that

has accumulated, a few investigators still question the association of between the inhalation of asbestos and lung cancer.²⁷⁻³¹

It has been mentioned previously that the term asbestos encompasses some 30 fibrous minerals, of which only seven have industrial importance. It is probable that the physical character of each fibrous form imparts to it a fibrogenic and carcinogenic ability which varies slightly from all other asbestos minerals. There is extensive experimentation in this area, but at the present time, however, no legal or conclusive toxicological differentiation is made between these seven industrial forms of asbestos and each is considered to be capable of producing asbestosis and carcinoma.

Ten to 15 years ago, asbestos board and cloth made wholly of asbestos were the best and most frequently used insulating materials. Thus, the average pipe insulator was exposed regularly to asbestos and must have been at considerable risk of developing asbestosis. If some of the older members of the union now suffer from this disease, they must have developed it years ago, for at this time (circa 1965), only one of the four insulation contracting firms that employ union men in this area (Cincinnati), regularly stock asbestos insulation in their warehouse, and this is utilized only rarely when specified. Today, local insulators are rarely, if ever, subjected to respiratory exposure to concentrations which approach the generally accepted tolerable limits.

Experimental Work With Magnesia and Calcium Silicate.—Insulators are exposed to asbestos, but in very small amounts and in an indirect manner.³² Two new insulating products developed in the last 10 to 15 years utilize 10% to 15% of asbestos, by weight, in combination with either magnesium carbonate or hydrous calcium silicate. Thus, insulators are exposed to asbestos only when they cut and install new insulation, or remove obsolete insulation of either these two compositions.

The dust of the product containing magnesium carbonate, known as magnesia in the insulating trades, is considered to be dangerous. It is reported to have caused pulmonary fibrosis which is radiologically

distinguishable from either typical silicosis or typical asbestosis, appearing to be intermediate between the two. It is impossible to explain the underlying lung lesion, but it may reflect a modification of the asbestos fibrosis affected by the introduction of a preponderance of a more readily soluble dust.³⁴

The dust of the product containing hydrous calcium silicate has been examined in extensive experimentation with animals.³³ The terminal syndrome observed of peribronchiolar fibrosis, atelectasis, and adenomatoid change was of the same kind as that which has been produced repeatedly in animal experiments by means of inhaled, particulate asbestos alone.

Thus, in both the magnesia and calcium silicate insulating materials, the 10% to 15% of asbestos has been indicated as the agent giving the dust its dangerous property of inducing fibrosis. Since about 40% of an insulator's normal work week is involved in handling these insulating materials containing low concentrations of asbestos, the health hazard of inducing fibrosis associated with exposure to such levels of concentration over prolonged periods of time becomes of critical importance.

Definition of Mesothelioma and Its Relation to Insulators.—Within the last ten years a disease entity, mesothelioma preceded by basal asbestosis, has been described and linked to a modest degree of respiratory exposure to asbestos.

A mesothelioma is a primary malignant tumor arising from serous surfaces such as the pleura, peritoneum, or pericardium.^{43,45} There are two kinds of pulmonary mesothelioma: (1) a primary form localized in one part of the pleura, and (2) a form diffuse in nature, involving the entire pleura and usually ensheathing the entire lung in tumor tissue.⁴⁴

Embryological investigations, tissue cultures, and autopsy reports indicate that there are gradations between the two forms. The diffuse tumor has been demonstrated to spread by: (1) growth over surfaces, (2) erosion into tissues and organs, (3) extension via lymphatics, (4) via vascular metastasis to distant sites, and (5) by various combinations of any or all of these

methods.⁶⁵ Both right and left pleurae are affected with equal frequency, and the tumor is twice as common in males as in females. The highest incidence occurs between 40 and 60 years of age. Diagnosis can be confirmed in most cases by cytologic examination of the abundant hemorrhagic pleural fluid which is usually present.⁶⁶

Many epidemiological surveys link exposure to asbestos with the formation of mesotheliomas.^{22,49,67,68} At present, no correlation is known to exist between the severity of pulmonary asbestosis and the frequency of occurrence of these tumors. In numerous cases the exposure to asbestos dust appears to be minimal or intermittent, and the only histologic evidence of such exposure is the presence of a few asbestos bodies and fibers in the lungs.^{69,70} In patients with peritoneal tumors, the pulmonary changes due to asbestos are below average in severity (disability has commonly been assessed at 10% to 30% by British standards). The shorter life span of the severely affected workman is thought to be the reason the syndrome is not more common. A cause of confusion is the frequent diagnosis of some other form of neoplasm such as carcinoma of the stomach, ovary, pancreas, and tonsil, as well as the unclassified cases of carcinoma involving the peritoneum.⁷¹

The handling of magnesium carbonate and calcium silicate insulating materials containing 10% to 15% asbestos fibers, by weight, may present a potential for the production of mesothelioma. If additional epidemiological investigations support the evidence available at this time, all insulators may have to be protected in an appropriate manner while at work.

Plastic Insulating Materials

Polyurethane.—The polyurethanes are a relatively new class of polymers. Like most synthetic polymers, the finished cured products or macromolecules based on polyisocyanates have proved to be physiologically inert.^{72,77}

Properly cured polyurethane foam plastics do not irritate the human skin.⁷⁸ As local insulators only handle the finished, cured,

foam product, this presents no health hazard in their occupation. Since this foam material need never be exposed to excessive amounts of heat during its handling, the slightly toxic pyrolysis products are usually unimportant.

Polystyrene.—The other plastic used in this field, at present, is polystyrene. Polystyrene is formed from styrene which is a colorless, odorless liquid. Upon heating, under carefully controlled conditions, it polymerises and changes to the solid polymer, polystyrene. It is probable that, in this change, several hundreds or even thousands of molecules of the original liquid styrene coalesce to form one molecule of the new solid polystyrene. Polystyrene comes on the market as a snow-white powder which then can be molded by compression to any desired form.^{79,80} Polystyrene in the molded form presents no hazard to health from its ingestion or contact with the skin, has no taste or odor, and is considered to be inert and nontoxic.^{77,81-84} When heated, the fumes from polystyrene are not significantly toxic, but may be disagreeable and for that reason should be controlled. Polystyrene has been approved by the Food and Drug Administration for use, commercially, in contact with food, and would appear to present no health hazard to insulators as they encounter this substance in the normal performance of their trade.

Cements and Adhesives

An insulator is in constant contact with cements, pastes, and adhesives since one of these materials is used regardless of the type of insulation being installed.

The Toxic Properties of Portland Cement, Asbestos Cement, and Wheat Paste.—Portland cement is composed of clay, lime, and silica in varying proportions. The silica is present as dicalcium and tricalcium silicate. Extensive clinical surveys of industrial workers subjected to respiratory exposure to finely divided portland cement in high concentrations have shown that the effect of the inhaled dust of that material on the lungs is insignificant.⁸⁵ Cement can act as a skin irritant by mechanical irritation of the silicates it contains and also by virtue of the lime it contains, which is highly alkaline and

hygroscopic. When cement with little moisture comes in contact with the skin, it absorbs moisture from it, and after some time causes the skin to become dry, hard, and thickened. Such skin may crack and result in fissures and indolent ulcers. Some cases of dermatitis produced by contact with cement were reported as possibly being due to the development of an allergic hypersensitivity to the hexavalent chromium contained within the cement.⁸⁶ Asbestos cement is composed of a portland cement along with a small amount of asbestos fibers. This material is always mixed in small quantities with water, and therefore, it is being handled, as a rule, in a wet, dustless form. The only possibility of producing a dusty atmosphere is in the pouring of the dry cement and during its mixing with water. During this brief period of time very few asbestos fibers are dispersed in the atmosphere. Asbestos cement should have no adverse effects on the health of those working with it. Wheat paste cement is composed of ground wheat, which when mixed with water acquires an adhesive quality. This material is inert and has no known toxic effect.

Review of the Health Hazards Associated With the Use of Adhesives.—In order to gather useful information, and still not omit a potentially hazardous substance, the number of adhesives analyzed was limited. A list was compiled of all the adhesives which are used most frequently. Of these adhesives those selected for analysis have been alleged or reported reliably to have caused illness, irritation of the skin, or other adverse effects. An additional adhesive was added as the only adhesive which is capable, at present, of meeting the requirements of the new local building ordinance.

The only health hazard related to the use of adhesives appears to be that associated with the organic solvents which they contain.

Since the solvents evaporate rapidly upon exposure to air it appeared that the majority of them have low boiling points. For this reason, a cold finger apparatus was utilized, at a temperature of 100 C, to extract the solvents from the adhesives. These solvents were then analyzed by gas chromatography to determine their exact composition.

Of the solvents determined, toluene is the most toxic. Commercial toluene may contain appreciable quantities of benzene, and so must be regarded with caution unless it is known to be of high purity. Pure toluene is completely eliminated from the body within 24 hours, and has no known irreversible or chronic effects within the body. Exposure to low concentrations of toluene causes fatigue, dizziness, and headache. Exposure to sufficient concentrations exerts a strong narcotic action (sleep-producing), but no chronic blood dyscrasias such as those induced by benzene are produced.⁸⁷⁻⁸⁹ The generally accepted tolerable concentration of toluene is 200 parts per million (ppm).

Ethanol is one of the least hazardous of the organic solvents. Inhalation of low concentrations of ethanol presents no health hazard, but it is capable of yielding an anesthetic effect when inhaled in high concentrations. The odor of ethanol is detectable at 2 ppm and only slight eye irritation results from exposure to concentrations barely in excess of 1,000 parts per million in the air.⁸⁷⁻⁸⁹

For the purposes of this survey, normal hexane is the most important of these solvents since it is contained in the only adhesive which meets the specifications of the new local building ordinance which went into effect on Oct 1, 1964. No systemic poisoning is associated with exposure to normal hexane under ordinary conditions, and it is rapidly eliminated from the body. Exposure to low concentrations of normal hexane may cause a moderate degree of irritation to eyes, nose, and throat. Headache, nausea, and narcosis may be induced by the inhalation of air containing 1,500 ppm for sufficient periods of time.⁸⁷⁻⁸⁹

Thus, on rare instances when insulators are exposed to these solvents in unusually high concentrations, they may develop symptoms of irritation, but after leaving the area they will recover rapidly without sustaining any permanent physical injury. Therefore, the handling of these particular adhesives appears to present no serious health hazards to members of the insulating trades.

Miscellaneous Insulating Materials

The fifth and last major category of insulating materials to be considered is composed of all materials which did not fit in previous categories. Hair felt, cork, and foam glass will be considered.

Hair Felt Insulation.—This survey was originally prompted by the death of one local insulator from anthrax. The goat hair felt he had been handling was contaminated with the spores of *B anthracis*. It appears that he contracted the disease through contact of this product with his skin, becoming thereby the first known victim in the United States, of this disease, acquired in this manner. This is a strange event since goat hair felt has been handled for decades by insulators. The spores of *B anthracis* are not exceedingly virulent, but are very resistant to unfavorable environmental influences. They will withstand dry heat and most chemical disinfectants for moderate periods of time and persist for years in dry earth.^{80,81} After the goat hair felt was identified as the contaminated material, it was traced to a distributor who had received the raw material from Africa. Further investigation revealed that both the goats from which the hair had been obtained and the soil of the goat farms were contaminated with spores of *B anthracis*. Goat hair felt is still used in other areas of the United States, but is no longer handled by any of the four insulating contractors in this area; a cattle hair felt has been substituted. Cattle hair felt has been known to be contaminated with anthrax spores. However, animal products contaminated with anthrax spores can be sterilized by autoclaving,⁸² and cattle hair felt now locally utilized is processed according to government specifications which include sterilization and washing.⁸² The insulators, therefore, incur no risk of contracting anthrax or other infectious diseases in handling such materials when so processed.

Cork Insulation.—Cork, which is handled infrequently by insulators, is derived from the cork oak, *Quercus suber* L.^{83,84} Cork dust is considered to be nontoxic, but it may constitute a nuisance.⁸⁵ The real danger, industrially, is that of explosion of this product

when stored in finely divided form. Insulators are not exposed to an environment which is likely to be dusty enough to produce an explosion. Recent articles by Cancelli^{84,85} indicate that cork dust may cause a pneumoconiosis known as Suberosis. The data presented by Cancelli appear to show that there is a chronic bronchitis produced, rather than a chronic, debilitating disease of the lung. In any event, the infrequent handling of cork by insulators dispels the likelihood of the occurrence among them of this questionable disease.

The Toxicity of Foam Glass Insulation as an Absorbent of Sulfur Dioxide.—The inherent toxicity of foam glass was explored in the first, major category of glass insulating materials. In addition to these considerations, the toxicity of sulfur dioxide must be examined in connection with foam glass, which is a porous material, for during its manufacture sulfur dioxide is produced and is captured in the pores. When the insulators manually cut foam glass to conform to the desired shape, the pores are fractured and sulfur dioxide escapes and contaminates the working atmosphere. Sulfur dioxide is an irritating gas, which exerts its primary effects on the respiratory system. The odor of sulfur dioxide in the concentration of 0.3 ppm is easily detected by the average individual, and that of 20 ppm is the least that is irritating to the eyes.^{86,88} It appears that the small amount of sulfur dioxide encapsulated in foam glass is incapable of contaminating the working environment to a significant degree. Therefore, this is largely a potential hazard which will rarely, if ever, become an actual hazard.

This fifth and final major category has contained a variety of materials, but none of these has appeared to present a hazard to the health of members of the insulating trade.

Next a literature review was made to find all prior papers in which the health hazards of insulators had been examined. Only four such papers were found:

Siebert, W. J.—This author was only concerned with the effect of fiberglass insulations on the health of the members of the International Association of Heat and Frost Insulators and Asbestos Workers of the St.

Louis area.⁹⁷ Through a complete survey of the medical literature and from clinical examinations, radiological examinations, skin tests, and life insurance rates of the employees of the Owens-Corning Fiberglass plant in Newark, Ohio, the writer concluded that fiberglass, as it was handled by the insulators, offered no occupational hazard.

Fleischer, W.E.—An additional survey conducted by Fleischer et al.,⁹⁸ in 1946, attempted to determine whether the risk of developing asbestosis existed among insulators employed in the construction and repair of naval vessels. Of the 1,074 insulators examined, only three cases of asbestosis were revealed, and the investigators concluded that because of the great variety of insulating materials handled and the inconsistency of exposure to asbestos, the risk of developing asbestosis was negligible.

Marr, W.T.—Marr⁹⁹ reviewed the work described above and compared these results with those of his survey of insulators employed in the construction and repair of naval vessels. Approximately 8% of the insulators in Marr's survey have developed asbestosis, and since a minimum of seven years is required for the development of asbestosis, Marr regarded Fleischer's survey as fallacious, since in Fleischer's survey only 54 of the group of 1,074 insulators had worked at the trade for more than ten years, and only three had sustained 20 or more years of experience. Thus, an adequate opportunity had not existed for the appearance of asbestosis among these men. In Marr's survey, many employees had incurred exposure to asbestos for more than 20 years with the results indicated above. Marr concluded that this was a dangerous trade, and that respirators should be worn by insulators when they were handling dry insulation containing asbestos.

Selikoff, I.J.—A final survey, conducted by Selikoff et al.,¹⁰⁰ in 1964, had as its objective the establishing of the extent, if any, of the relationship between exposure to asbestos and the occurrence of neoplasia among the members of the International Association of Heat and Frost Insulators and Asbestos Workers. This survey of the death records, for the past 20 years, of 255

men in the New York and New Jersey areas, with 20 or more years work of experience in the trade, revealed that the incidence of all types of neoplasia among those men was significantly higher than that in the normal population over the same period of time. Specifically, 17.6% of these men had died of carcinoma of the lung and pleura, and 11.4% had died of cancer of the stomach, colon, or rectum. These men had incurred only intermittent exposure to materials containing limited amounts (often 2% to 20%) of asbestos, under working conditions that had varied from very dusty, as in extracting old insulation in closed quarters, to only slightly dusty, as in building construction in the open air. The results of this survey, in addition to the information on mesothelioma presented in the second major category, definitely justifies concern as to the safety of the men employed in this trade.

In view of Selikoff's results a similar survey was made of the death records of insulators who were members of the International Association of Heat and Frost Insulators and Asbestos Workers in the Cincinnati area. A list was compiled of all the members in the Cincinnati area who had died during the 25 years between Jan 1, 1939 and Jan 1, 1964.¹⁰¹ This particular span was chosen because the insulating trade was severely affected during the depression, but by 1939, the union rolls were made up mainly of men with considerable work experience. Since previous surveys had suggested that neoplasia associated with asbestos exposure seldom occurred until 20 years after the inception of exposure to asbestos, the death records of men with such occupational histories were of special interest.

Of the original 47 men who were listed as union members in the Cincinnati area of Jan 1, 1939; ten men were still actively engaged in the trade on Jan 1, 1964, two men had retired (one in 1960 and one in 1962) and are still living, two men had left the trade in 1956 and are still living, and 33 men had died during the 25 year period under consideration. The death records of these individuals were obtained and classified according to the recorded primary cause of death.¹⁰²⁻¹⁰⁴ Malignant neoplasms had been

the cause of the deaths of eight men, seven of whom had been employed in the insulating trade for more than 20 years.¹⁰²

Malignant neoplasms thus accounted for 24.2% of all deaths during this 25 year period. Specifically, 9.1% had died of carcinoma of the lung and 6.1% had died of cancer of the caecum or sigmoid.

These percentages appear to be extremely high and superficially would suggest a causal relationship, but caution must be observed in interpreting this datum since both the population and sample sizes concerned are very small. This and other statistical considerations prevent these percentages from being statistically significant.

The mortality data so reported are not intended to provide proof of the association between an occupation and the occurrence of neoplasia, but they represent an attempt, with the limited data available, to reveal any existing trend along these lines.

Summary

In this survey the attempt has been made to investigate all of the substances with which insulators come in contact in the normal pursuit of their trade.

In the present state of technology, and in light of existing medical and toxicological data, the majority of the substances examined proved incapable of presenting a serious health hazard to insulators.

The only insulating substance which justifies concern is asbestos. This report and other available scientific data at the present time are insufficient to provide an adequately based conclusion. The most convincing data are those of Selikoff. The further results of his and other investigations will be awaited and should contribute toward sound judgment of this matter.

REFERENCES

1. Kettering Laboratory: Statement and Supporting Documents Concerning the Potential Occupational Hazard of Respiratory Exposure to Air-Borne Fibrous Glass, in Relation to a Threshold Limit Value. Unpublished Report, 1964.
2. Excerpts From the Annual Report of the Saranac Laboratory for the Study of Tuberculosis of the Edward L. Trudeau Foundation for the Year 1940, Saranac Lake, NY.
3. Ciria, P.: Occupational Diseases From Exposure to Glass Fibers, *Med Lavoro* 39:152-157, 1958.
4. Hazard, W.G.: Transcript of Remarks on Health Aspects of Fiberglass Materials. Proceedings of the Rhode Island Industrial Health Institute, Providence, 1943.
5. Owens-Corning Fiberglass Corporation: Health Aspects of Fiberglass Materials, Toledo, Ohio, 1943.
6. Scheper, G.W.H.: The Biological Action of Glass Wool, *Arch Industr Health* 12:280-287, 1955.
7. Scheper, G.W.H.: Pulmonary Histologic Reactions to Inhaled Fiberglass-Plastic Dust, *Amer J Path* 35:1169-1187, 1959.
8. Scheper, G.W.H.: The Pulmonary Reaction to Sheet Fiberglass-Plastic Dust, *Amer Industr Hyg Assoc J* 20:73-81, 1959.
9. Scheper, G.W.H.: Reaction of Monkey Lung to Siliceous Dusts, *Arch Environ Health* 5:278-289, 1961.
10. Gross, P.; Westrick, M.L.; and McNeerney, J.M.: Glass Dust: A Study of Its Biologic Effects, *Arch Industr Health* 21:10-28, 1960.
11. Roche, L.: The Pulmonary Hazards in the Glass Fiber Industry, *Arch Mal Prof* 7:27-28, 1946.
12. Mungo, A.: Affections of Persons Working in the Manufacture of Stratified Glass Wool Products, *Folia Med* 43:962-970, 1960; abstracted *Excerpta Med* (VII), 1961.
13. Pellerat, J., and Coudert, J.: Glass Wool Dermatitis, *Arch Mal Prof* 7:23-27, 1946.
14. Dhers, V.: Fiberglass Industry: Technical Data, *Arch Mal Prof* 7:9-23, 1946.
15. Hein, B.J.: Hazards of Exposure to Glass Wool, Glass Frit or Foam Glass, *JAMA* 124:187-188, 1944.
16. Heisel, E.B., and Mitchell, J.H.: Cutaneous Reaction to Fiberglass, *Industr Med Surg* 26:547-550, 1957.
17. Pellerat, J.: Glass-Wool Dermatitis, *Ann Derm Syph* 1:25-31, 1947; abstracted *Excerpta Med* VIII 3:10, 1949.
18. Suizberger, M.G., et al.: Effects of Fiberglass on Animal and Human Skin: Experimental Investigation, *Industr Med* 11:482-484, 1942.
19. Bohroff, A.: Itching From Ventilator Borne Fiberglass Particles, *JAMA* 186:80-81, 1963.
20. Council on Physical Medicine Fiberglass-Filled Bed Pillows Acceptable, *JAMA* 132:331, 1946.
21. Council on Physical Medicine. Glasdown Pillows Acceptable, *JAMA* 134:145, 1947.
22. Council on Physical Medicine. Fiberglass Filled Sleepytime Pillow Acceptable, *JAMA* 136:398, 1948.
23. Judd, A.R.; Szpalski, J.; and Kopernik, F.: Use of Fiberglass in Pulmonary Tuberculosis, *J Thorac Surg* 27:581-592, 1954.
25. O'Neill, T.J.E.; Redonido Romero, H.P.:

- and Throat, R.G.: Experimental and Clinical Study of Collapse Therapy Using Fiberglass Wool and Fabric, *Thorac Surg* 18:181-187, 1949.
25. Hunter, D.: *The Diseases of Occupation*, Boston: Little, Brown and Co., 1955, pp 874-884.
26. Vorwald, A.J.; Durkam, T.M.; and Pratt, P.C.: Experimental Studies of Asbestosis, *Arch Industr Hyg Occup Med* 3:1-43, 1951.
27. Hygienic Guides Committee: Asbestos, *Amer Industr Hyg Assoc J* 19:161-162, 1958.
28. American College of Chest Physicians: Asbestosis and the Asbestos Dust, 1962.
29. Lanza, A.J.: *Silicosis and Asbestosis*, New York, London, Toronto: Oxford University Press, 1938, pp 23, 59.
30. Lanza, A.J.: *The Pneumoconiosis*, New York: Grune & Stratton, Inc., 1963, pp 13-25.
31. Thomson, J.G.: Exposure to Asbestos Dust and Diffuse Pleural Mesotheliomas, *Brit Med J* 1:123, 1963.
32. Spencer, H.: *Pathology of the Lung*, New York: Macmillan Co., 1960, pp 341-345, 618-619, 729-731.
33. Merewether, E.R.A.: The Occurrence of Pulmonary Fibrosis and Other Pulmonary Affections in Asbestos Workers, *J Industr Hyg* 12:239-257, 1930.
34. Merewether, E.R.A.: A Memorandum on Asbestosis, *Tubercle* 15:69, 109, 152-159, 1933-34.
35. Gloyne, S.R.: The Asbestosis Body, *Lancet* 1:1351-1355, 1932.
36. Isseibacher, K.J.; Klaus, H.; and Hardy, H.L.: Asbestosis and Bronchogenic Carcinoma: Report of One Autopsied Case and Review of Available Literature, *JAMA* 15:721, 732, 1953.
37. Lynch, K.M., and Smith, W.A.: Asbestosis Bodies in Sputum and Lung, *JAMA* 95:659-661, 1930.
38. Gloyne, S.R.: The Formation of the Asbestosis Body in the Lung, *Tubercle* 12:399-401, 1931.
39. Lynch, K.M., and Cannon, W.M.: Asbestosis: VI. Analysis of 40 Necropsied Cases, *Brit J Industr Med* 14:874-890, 1949.
40. Wood, W.B., and Gloyne, S.R.: Pulmonary Asbestosis, *Lancet* 1:445-448, 1930.
41. Cordova, J.F.; Teshuk, H.; and Kandtson, K.: Asbestosis and Carcinoma of the Lung, *Cancer* 15:1181-1187, 1962.
42. Gardner, L.V., and Cummings, D.E.: Inhalation of Asbestos Dust: Its Effects Upon Primary Tuberculous Infection, *J Industr Hyg* 13:65-81, 1931.
43. Anderson, W.A.D.: *Synopsis of Pathology*, ed 5, St. Louis: C.V. Mosby Co., 1960, pp 429-430, 240.
44. Wagner, J.C.: Asbestosis in Experimental Males, *Brit J Industr Med* 20:1-12, 1963.
45. Dreessen, W.C., et al: A Study of Asbestosis in the Asbestos Textile Industry, US Public Health Bulletin 241, 1938.
46. Sayers, R.R., and Dreessen, W.C.: Asbestosis, *Amer J Public Health* 29:205-214, 1939.
47. Menkin, V.: Role of Inflammation in Carcinogenesis, *Brit Med J* 1:1586-1594, 1960.
48. Homburger, F.: The Co-Incidence of Primary Carcinoma of the Lungs in Pulmonary Asbestosis, *Amer J Path* 19:797-805, 1943.
49. Bonser, G.M.; Faulds, J.S.; and Stewart, M.J.: Occupational Cancer of the Urinary Bladder in Drystuffs Operatives and of the Lungs in Asbestos Textile Workers and Iron-Ore Miners, *Amer J Clin Path* 25:126-134, 1955.
50. Doll, R.: Mortality From Lung Cancer in Asbestos Workers, *Brit J Industr Med* 12:81-91, 1955.
51. Lynch, K.M., and Smith, W.A.: Pulmonary Asbestosis: Carcinoma of Lung in Asbestos-Silicosis, *Amer J Cancer* 24:56, 1935.
52. Annual Report of Chief Inspector of Factories for Year 1955, London: Her Majesty's Stationary Office, 1956, p 206.
53. Gloyne, S.R.: Pneumoconiosis: A Histological Survey of Necropsy Material in 1205 Cases, *Lancet* 1:810-814, 1951.
54. Anderson, J., and Campagna, F.A.: Asbestosis and Carcinoma of the Lung, *Arch Environ Health* 1:27-32, 1960.
55. Behrens, W., Jr.: The Clinical Picture and Pathology of Asbestosis, *Z Unfallmed Berufskr* 45:129-140, 1952; abstracted *Bull Hyg* 28:192, 1953.
56. Warren, S.: Survey of Some Current British and European Studies of Occupational Tumor Problems, *Occup Med* 5:249-254, 1948.
57. Braun, D.C., and Traut, T.D.: Epidemiological Study of Lung Cancer in Asbestos Miners, *Arch Industr Health* 17:634-653, 1958.
58. Bohlig, H.; Jacob, G.; and Kallabis, B.: Morbidity and Pathology of Lung Cancer With Asbestosis, *Z Unfallmed Berufskr* 52:64-78, 1959; abstracted *Bull Hyg* 34:1020-1021, 1959.
59. McIntyre Research Foundation, Lanza, A.J.: Asbestosis, read before the 4th Conference of McIntyre Research Foundation on Silicosis held in Noranda, Quebec, Jan 27, 1952.
60. Cartier, P.: Abstract of Discussion, *Arch Industr Hyg* 5:262-263, 1952.
61. Smith, K.W.: Pulmonary Disability in Asbestos Workers, Transactions of the McIntyre-Saranac Conference on Occupational Chest Disease, G.W.H. Schepers (ed.), Chicago: American Medical Association, 1955, pp 196-201.
62. Lanza, A.J.: *Silicosis and Asbestosis*, New York: Oxford University Press, 1938, pp 439.
63. McCaughey, W.T.E.: Primary Tumors of the Pleura, *J Path Bact* 76:517-529, 1958.
64. Klemperer, P., and Robin, C.B.: Primary Neoplasms of the Pleura, *Arch Path* 11:385-412, 1931.
65. Godwin, M.C.: Diffuse Mesotheliomas With

- Comment on Their Relation to Localized Fibrous Mesotheliomas, *Cancer* 10:298-319, 1957.
66. Campbell, W.N.: Pleural Mesotheliomas, *Amer J Path* 26:473-483, 1950.
 67. Wagner, J.C.; Sleggs, C.A.; and Marchand, P.: Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province, *Brit J Industr Med* 17:260-271, 1960.
 68. Keal, E.E.: Asbestosis and Abdominal Neoplasms, *Lancet* 2:1211-1216, 1960.
 69. Smither, W.J.: Mesotheliomas and Asbestos Dust, *Brit Med J* 2:1194-1195, 1962.
 70. McCaughey, W.T.E.: Exposure to Asbestos Dust and Diffuse Pleural Mesotheliomas, *Brit Med J* 2:1397, 1962.
 71. Enticnap, J.B., and Smither, W.J.: Peritoneal Tumors in Asbestosis, *Brit J Industr Med* 21:20-31, 1964.
 72. Haddow, A.C.: Report of Annual Meeting of British Medical Association, Manchester, 1929, *Lancet* 2:230-231, 1929.
 73. Thomson, J.G.; Kaschula, R.O.C.; and MacDonald, R.R.: Asbestos as a Modern Urban Hazard, *S Afr Med J* 37:77-81, 1963.
 74. Bobyleva, A.T., et al.: The Amount of Dust in Residential Districts of the City of Asbest and Its Effect on the Children's Health, *Arch Environ Health* 2:727, 1961.
 75. Wagler, F.; Muller, H.; and Anspach, M.: Is There an Endemic Asbestosis? *Z Ges Hyg* 8: 246-255, 1962; abstracted *Bull Hyg* 37:1025, 1962.
 76. Anonymous: Health Hazards of Some Newer Processes Used in Construction, *Industr Hyg News Rep* 4:1, 1961.
 77. Wilson, R.H., and McCormick, W.E.: Plastics: The Toxicology of Synthetic Resins, *Arch Industr Health* 21:536-548, 1960.
 78. Zapp, J.A., Jr.: Hazards of Isocyanates in Polyurethane Foam Plastic Production, *Arch Industr Health* 15:324-330, 1957.
 79. Shumskaya, N.I.: Hygiene of Work in Manufacturing and Utilization of Polystyrene, *Chem Abstr* 58:10655d, 1963.
 80. "Plastes": *Plastics in Industry*, Brooklyn, NY: Chemical Publishing Co., Inc., 1941, pp 12-13.
 81. Sporn, A.; Schoebesch, O.; and Nicolau, C.: Hygienic Characterization of Plastic Materials Used in the Food Industry: Toxicity of Extracts of High-Pressure Polyethylene, Low-Pressure Polyethylene, and of Polystyrene, *Igiene* 12:21026, 1963; abstracted *Chem Abstr* 59:6887, 1963.
 82. Goggin, W.C.: The Polystyrene Family, American Society for Testing Materials, Symposium on Plastics, 1944, p 186.
 83. Patty, F.A.: *Industrial Hygiene and Toxicology*, ed 2, F. A. Patty, (ed.), New York: Interscience Publishers, 1958, pp 2297-2298.
 84. Struthers, J.A., et al.: *Styrene Resins and Plastics Encyclopedia of Chemical Technology*, R. E. Kirk and D. F. Othmer, (eds.) New York: Interscience Encyclopedia, Inc., 1947, vol 13, p 164.
 85. Schepers, G.W.H.; Durkan, T.M.; and Delehan, A.B.: Effect of Inhaled Commercial Hydrous Calcium Silicate Dust on Animal Tissues, Transactions of the McIntyre-Saranac Conference on Occupational Chest Disease, G.W.H. Schepers (ed.), Chicago: American Medical Association, 1955, pp 343-55.
 86. Schwartz, L.; Tulipan, L.; and Birmingham, D.J.: *Occupational Diseases of the Skin*, ed 3, Philadelphia: Lea and Febiger 253-259, 764-765, 1957.
 87. Fairhall, L.T.: *Industrial Toxicology*, ed 2, Baltimore: Williams & Wilkins Co., 1957, pp 237-238, 268-269, 343-345.
 88. American Industrial Hygiene Association: Hygienic Guide Series, Detroit, Mich, 1955.
 89. National Association of Mutual Casualty Companies: Handbook of Organic Industrial Solvents, ed 2, Technical Guide No. 6, 1961, pp 46, 50, 62.
 90. Jawetz, E.; Melnick, J.L.; Adelbert, E.A.: *Review of Medical Microbiology*, ed 5, Los Altos, Calif: Lange Medical Publications, 1962, pp 149-50.
 91. American Public Health Association: *Control of Communicable Diseases in Man*, ed 9, New York: American Public Health Association, 1960, pp 24-27.
 92. Federal Standard Stock Catalog, Section IV, (Part 5) Federal Specification for Felt: Hair, C-F-201a, June 6, 1933.
 93. Patty, F.A.: *Industrial Hygiene and Toxicology*, ed 2, F. A. Patty (ed.), New York: Interscience Publishers, 1963 vol 2, pp 892-895, 2254.
 94. Cancelli, L. de C.: Suberosis: A Pneumoconiosis Due to Cork Dust, *Industr Med Surg* 32:435-445, 1963.
 95. Cancelli, L. de C.: Suberosis: Pulmonary Changes Associated With Inhalation of Cork Dust, *Bull Hyg* 35:759-760, 1960.
 96. Henderson, Y., and Haggard, H.W.: *Noxious Gases and the Principles of Respiration Influencing Their Action*, ed 2, New York: Reinhold Publishing Corp, 1943, pp 124-131.
 97. Siebert, W.J.: Fiberglass Health Hazard Investigation, *Industr Med* 11:6-9, 1942.
 98. Fleischer, W.E., et al.: A Health Survey of Pipe Covering Operations in Construction Naval Vessels, *Industr Hyg* 28:9-16, 1964.
 99. Marr, W.T.: Asbestos Exposure During Naval Vessel Overhaul *Amer Industr Hyg Assoc J* 25:264-268, 1964.
 100. Selikoff, I.J.; Churg, J.; and Hammond, E.C.: Asbestos Exposure and Neoplasia, *JAMA* 188:22-26, 1964.

101. Hutchinson, A.E.: Personal communication.
 102. Kuntz, D.: Personal communication, 1964.
 103. Taylor, S.: Personal communication, 1964.
 104. Williams, E.G.: Personal communication, 1964.
 105. Hutchinson, A.E.: Personal communication, 1964.
 106. Gruber, C.W.: Personal communication, 1964.
- The following references were not included in the text of this paper, but were of great value in gaining a thorough knowledge of the subjects presented.
107. Watrous, R.M.: Health Hazards in the Pharmaceutical Industry, *Brit J Industr Med* 4: 111-125, 1947.
 108. Schepers, G.W.H., and Delahant, A.B.: An Experimental Study of the Effects of Glass Wool on Animal Lungs, *Arch Industr Health* 12: 276-279, 1955.
 109. Anonymous: New Hazards to Health in Industry: 1. Fiberglass and Resin Plastics: The Toxicity of Their Hardeners (Catalysts), *Industr Med Surg* 31:40-41, 1962.
 110. Grass, P.: Fiber Glass Pneumoconiosis, *Arch Environ Health* 4:645, 1962.
 111. Murphy, G.B., Jr.: Fiber Glass Pneumoconiosis, *Arch Environ Health* 3:704-710, 1961.
 112. Desouille, H., and Diers, V.: Working Conditions in Insulation Industry Using Glass Wool, *Arch Mat Prof* 7:332-344, 1946.
 113. Edwin, J.R.: Fiberglass Plastics Industrial Medical Aspects and Experiences, *Industr Med* 16:439-441.
 114. McKenna, W.B.; Smith, J.F.F.; and Maclean, D.A.: Dermatoses in the Manufacture of Glass Fibre, *Brit J Industr Med* 15:47-51, 1958.
 115. Fiberglass and Skin Irritation, QUESTIONS AND ANSWERS, *JAMA* 177:664, 1961.
 116. Schwartz, L.: Dermatitis From Glass Fabrics, *Arch Derm Syph* 55:258, 1947.
 117. Schwartz, L., and Bolvimick, I.: Skin Hazards in the Manufacture of Glass Wool and Thread, *Industr Med* 12:142-144, 1943.
 118. Kahlau, G.: Fatal Pneumonia After Inhalation of Glass Dust During Preparation of a Synthetic Substance From Glass Wool, *Brit J Industr Med* 6:55, 1949.
 119. Schepers, G.W.H., and Gerrit, W.H.: The Biological Action of Degussa Submicron Amorphous Silica Dust, *Arch Industr Health* 16:125-146, 1957.
 120. Schepers, G.W.H.: The Biological Action of Fiberglass—Plastic Dust, *Arch Industr Health* 18:34-57, 1958.
 121. Gardner, L.V., and Rollin, A.J.: The Significance of Chemical Examination in the Diagnosis of Silicosis, *J Industr Hyg* 24:125-130, 1942.
 122. Gloyne, S.R.: Two Cases of Squamous Carcinoma of the Lung Occurring in Asbestosis, *Tubercle* 17:5-10, 1935.
 123. Wood, W.B., and Gloyne, S.R.: Pulmonary Asbestosis Complicated by Pulmonary Tuberculosis, *Lancet* 2:954-956, 1931.
 124. Kettle, E.H.: The Interstitial Reactions Caused by Various Dusts and Their Influence on Tuberculous Infections, *J Path Bact* 35:395-405, 1932.
 125. Smith, K.W.: Pulmonary Disability in Asbestos Workers, *Arch Industr Health* 12:198-203, 1955.
 126. Morris, G.E.: Toxic Hazards: Asbestos, *New Eng J Med* 260:141, 1959.
 127. Knox, J.R.: Asbestosis and Abdominal Neoplasms, *Lancet* 1:560, 1961.
 128. Desmeules, R., et al: Asbestosis and Cancer of the Lung, *J Industr Hyg* 30:94, 1948.
 129. Stoll, R.; Bass, R.; and Angrist, A.A.: Asbestosis Associated With Bronchogenic Carcinoma, *Arch Intern Med* 88:831-834, 1951.
 130. Peterson, J.E.; Copeland, R.A.; and Hayle, H.R.: Health Hazards of Spraying Polyurethane Foam Out-of-Doors, American Industrial Hygiene Conference Abstracts 18, 1962.
 131. Oppenheimer, B.S., et al: Malignant Tumors Resulting From Embedding Plastics in Rodents, *Science* 118:305-306, 1953.
 132. Lieber, E.E.: Dermatitis: An Industrial Problem, *Brit Plastics* 28:428-429, 1955; abstracted *Chem Abstr* 51:638, 1957.
 133. Parmeggiani, L., and Sassi, C.: Occupational Risk and Pathology in the Production and Manipulation of Some Plastic Material, *Med Lavoro* 46:14-24, 1955; abstracted *Chem Abstr* 49: 9317, 1955.
 134. Vorwald, A.J., and Karr, J.W.: Pneumoconiosis and Pulmonary Carcinoma, *Amer J Path* 14:49-57, 1938.
 135. Selikoff, I.J.: Personal communication, 1964.
 136. Marr, W.T.: Personal communication, 1964.
 137. Rowe, R.J., and Williams, G.H.: Severe Reaction to Cement, *Arch Environ Health* 7: 709-711, 1963.
 138. Morris, G.E.: The Primary Irritant Nature of Cement, *Arch Environ Health* 1:301, 1960.
 139. Moskowitz, S.: How the Adhesive Manufacturer and User Can Eliminate Solvent Hazards, *Industr Hyg Rev* 3:3-14, 1960.
 140. Harrington, J.S., and Smith, M.: Studies of Hydrocarbons on Mineral Dusts, *Arch Environ Health* 8:453-458, 1964.
 141. Harrington, J.S.: Occurrence of Oils Containing 3:4-Benzpyrene and Related Substances in Asbestos, *Nature* 193:43-35, 1962.

142. Dutra, F.R., and Carney, J.D.: Asbestosis and Pulmonary Carcinoma, *Arch Environ Health* 10:416-423, 1965.
143. Gardner, L.U.: Etiology of Pneumoconiosis, *JAMA* 111:1925-1936, 1938.
144. Hannon, J.W.G., et al: Report of Section on Nature and Prevalence Committee on Occupational Diseases of the Chest, American College of Chest Physicians, *Dis Chest* 45:107-111, 1964.
145. Hourihane, D. O'B.: The Pathology of Mesotheliomata and an Analysis of Their Association With Asbestos Exposure, *Thorax* 19:268-278, 1964.
146. Konig, J.: Über die Asbestose, *Arch Gewerbepath Gewerbehyg* 18:159, 1960; abstracted *Occup Med* 3:135-136, 1961.
147. Wright, G.W.: Functional Abnormalities of Industrial Pulmonary Fibrosis, *Arch Industr Health* 11:196-203, 1955.
148. Telischi, M., and Rubenstein, A.I.: Pulmonary Asbestosis, *Arch Path* 72:234-243, 1961.
149. Mancuso, T.F., and Coulter, E.J.: Methodology in Industrial Health Studies, *Arch Environ Health* 6:210-226, 1963.
150. HiAn, S., and Koprowski, I.: Primary Cytologic Diagnosis of Asbestosis Associated With Bronchiogenic Carcinoma, *Acta Cytol* 6:391-398, 1962; abstracted *Arch Environ Health* 6:684, 1963.
151. Elwood, P.C., and Cochrane, A.L.: A Follow-Up Study of Workers From an Asbestos Factory, *Brit J Industr Med* 21:304-307, 1964.
152. Williams, W.J.: Asbestosis and Lung Cancer, *Arch Environ Health* 10:44-45, 1965.
153. O'Donnell, W.M., and Mann, R.H.: Asbestos: Extrinsic Factor in Pathogenesis of Bronchiogenic Carcinoma, *Amer J Path* 33:610, 1957.
154. Annual Report of Chief Inspector of Factories for 1958, London: Her Majesty's Stationary Office, 1959, p 45.
155. Conference on Biological Effects of Asbestos. The New York Academy of Sciences Section of Biological and Medical Sciences, Oct 1964, to be published.

CROWDING ANIMALS

A pair of deer were put on an island in Chesapeake Bay about forty years ago. This is a small island of about 150 acres. The deer were kept well supplied with food. It was found that the colony grew until it reached a density of about one deer per acre. Then the animals began to die off and did so in spite of adequate food and care. Again, when these dead animals were examined there was marked evidence that the adrenal stress syndrome had been in operation. Studies have been made of the crowding of animals in the Philadelphia Zoo. It was found that in some species of animals there was a ten-fold increase in atherosclerosis under conditions of severe crowding, and there were many other symptoms characteristic of stress. John Christian of the Naval Medical Research Institute made population studies in relation to crowding of mice. In his 1950 paper in the *Journal of Mammalogy* entitled "The adreno-pituitary system and population cycles in mammals," he wrote in part: "We now have a working hypothesis for the die-off terminating a cycle. Exhaustion of the adreno-pituitary system resulting from increased stresses inherent in a high population, especially in winter, plus the late winter demands of the reproductive system, due to increased light or other factors, precipitates population-wide death with the symptoms of adrenal insufficiency and hypoglycemia convulsions."—Hoagland, H.: "Cybernetics of Population Control," in *Human Fertility and Population Problems*, Proceedings of the Seminar sponsored by the American Academy of Arts and Sciences, R. O. Green (ed.), Cambridge, Mass: Schenkman Publishing Co., Inc., 1963, p 8.