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November 2, 1970

Msgr. Manning, Executive Director
Girl Scouts of Metropolitan Detroit
151 E. Elizabeth
Detroit, Michigan

Dear Mr. Manning:

This is to call your attention to a potentially hazardous activity suggested in the Girl Scout Manual entitled: "Moreabouts for Brownie Girl Scout Leaders." On page 43 of this manual is a suggestion for making candlestick holders from asbestos dough and on page 64 is a recipe for the asbestos modeling material.

There is extensive literature in medical and environmental health journals which indicate a relationship between exposure to asbestos dust and serious lung diseases. I am attaching copies of two articles that you may find interesting which survey some of these findings: (1) Paul Broder, "A Reporter at Large — The Magic Mineral," The New Yorker, October 12, 1968; and (2) J. Selikoff, "Asbestos," Environment, Vol. 11, March 1969. Incidentally, I have brought these articles in the manual to Prof. Selikoff's attention. Prof. Selikoff, who is Director of the Environmental Sciences Laboratory of the Mount Sinai School of Medicine of the City University of New York, is a recognized worker in the field of asbestos disease epidemiology.

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Mrs. M. Manning

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November 2, 1970

In view of the possible danger of exposure to asbestos dust, you may wish to recommend that reference to asbestos modeling material be deleted from future printings of this (or any other) manual, and that cautionary information be disseminated to appropriate Girl Scout leadership.

Sincerely,

Roy L. Geiler
Roy L. Geiler, Ph.D.
Chemical Engineering Department
Scientific Research Staff

RLG:red

cc: Joseph H. Brinton, Executive Director
Boy Scouts of America Detroit Area Council,
276 W. Warren, Detroit, Mich. 48208

Prof. I. J. Selikoff, M.D.
Mount Sinai School of Medicine of the
City University of New York
5th Avenue and 100 Street
New York, New York 10029

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A REPORTER AT LARGE

THE MAGIC MINERAL

BACK in the nineteen-twenties, when the word "ASBESTOS" frequently appeared in large letters on the fireproof drop curtains that closed off the proscenium hangings of theatre stages, it was customary in certain parts of the country for youngsters attending Saturday-afternoon performances of "Charley's Aunt" or "The Vagabond King" or "The Wiggs of the Cabbage Patch" to greet the raising of the asbestos curtain with the chant "All school boys threw tomatoes on Saturday!" It was not, of course, an exercise in mnemonics but an expression of youthful exuberance, and, to a lesser extent, perhaps, a reflection of the tendency in the country to dismiss strange and exotic substances with jokes. Among older people it was certainly common knowledge that asbestos was noncombustible, or when "Joe and Asbestos"—the syndicated comic strip about horse racing—first appeared in the Baltimore Sun, in 1925, the stableboy named Asbestos explained to the readership that he had acquired his odd moniker because "no matter how hot my tips are, they'll never burn a hole in your pocketbook." Such knowledge was hardly universal, however, for it was also true that a popular put-on of the flapper age consisted of solemnly assuring a susceptible theatre companion that the ununny-looking word on the curtain meant "Welcome" in Latin. The fact of the matter is that although asbestos was linked one way or another in the public mind with fireproof theatre curtains, many people thought its name was a trademark, few people knew that it was being utilized in a wide variety of industrial products, only a handful of medical researchers were even beginning to suspect that it was affecting the health of the workmen who handled it, and usually no one had any idea that it would one day prove to be one of the most important industrial causes of cancer in

or unquenchable, is a broad term embracing a number of fibrous silicate minerals that are found in practically every country in the world. According to geologists, chrysotile asbestos—a variety of the mineral that accounts for ninety-five per cent of the world's production—had its origin millions of years ago, when subterranean upheavals provided fractures in serpentine rock which were penetrated by water heated in the earth's interior. Subsequently, these waters dissolved some of the serpentine, and as the solutions cooled, fibrous silicate crystals began to grow within the fractures. The two other major varieties of asbestos—crocidolite and amosite—originated through somewhat different dynamic and thermal processes in metamorphic rock, and their fibres, though similar to chrysotile, possess different structural and chemical properties. Although known to man as early as the Stone Age, when it was used in pottery, asbestos was considered a novelty until the late eighteen-seventies, when it began to be mined on a commercial basis. Since then, it has achieved enormous industrial importance because of its unique and astonishing physical properties. It appears to be highly combustible, yet it can withstand the fiercest heat. It seems as perishable as grass, but by virtue of being almost immune to

the forces of corrosion and decay under almost every condition of temperature and moisture (and of being resistant as well to the action of most acids, alkalis, and other chemicals) it is just about indestructible. It looks extremely fragile, yet its fibres have a tensile strength equal to that of piano wire. Apparently as light and feathery as thistle-down, it is actually as heavy and dense as the rock from which it is extracted. In one sense, then, it is a fibre of stone. In another sense, however, it is a mineralogical vegetable whose fibres are so soft and flexible that they can be carded, spun, and woven as easily as fibres of cotton or flax.

Asbestos is the only mineral that can be woven into cloth, and its fibrous structure is, if anything, even more amazing than its remarkable ability to withstand heat. In fact, if it were not for the electron microscope, the extent to which asbestos is fibrous would be difficult to believe, for there are approximately a million individual fibrils lying side by side in a linear inch of chrysotile asbestos, whereas only thirty-eight hundred glass fibrils, such as those found in various insulation materials, or six hundred and thirty human hairs can be aligned along the same distance. Moreover, in addition to their extreme fineness, high tensile strength, unusual flexibility, spinnability, and resistance to heat and the elements, asbestos fibres possess great powers to adsorb and to



ASBESTOS, which is derived from the Greek word *asbestos*, meaning unquenchable



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filter. Small wonder, then, that asbestos, which had been known to the ancients as "the magic mineral," in effect, rediscovered less than a hundred years ago in the age of industrial expansion, it was put to work.

Since that time, the demand for asbestos has risen almost as fast as the stuff could be mined from the earth and milled from its host rock. In 1868 when the world's first commercial asbestos mine was opened at Thetford in the Province of Quebec, only a few hundred tons of the mineral was produced. By 1910, annual world production had jumped to thirty thousand tons; by the middle thirties, it had increased to five hundred thousand tons and today the total output is more than four million tons a year. Until a few years ago, asbestos was often called "the mineral of a thousand uses" by those who sold it, but this label has become obsolete, especially in the United States, where it is estimated that asbestos may have many as three thousand different industrial applications. The United States is the largest consumer of asbestos in the world, importing nearly a million tons a year, mostly chrysotile from Canada, and since fully ninety per cent of this amount is used in combination with other raw materials—such as Portland cement, asphalt and vinyl and other plastics—asbestos is present as an invisible and anonymous ingredient in a myriad of industrial products, many of which are not identified as containing asbestos even though it may constitute up to fifty per cent of their content.

The asbestos-cement industry, which is the principal consumer of the mineral, makes shingles for roofing, siding, sheets for exterior and interior walls, insulation board, clapboard, casings for electric motors, pipes for carrying water, sewage, and gas, and numerous other products. Asbestos textiles are used not only in fireproof theatre curtains but also in lagging and other insulating wrapping, in conveyor belts and safety clothing, in pot holders, ironing-board covers, draperies, rug and motion-picture screens, as filters for gas masks and in the processing of fruit juices, acids, beer, and medicines, as in mailbags, prison-cell padding, airplane fittings, stove and lamp wick, sparkplugs, and fire hose. The electrical-equipment industry has depended for years on asbestos yarn and tape for insulating conductors ranging from fine-gauge wire to heavy cable, a woven asbestos friction material, such as brake linings and clutch facings.

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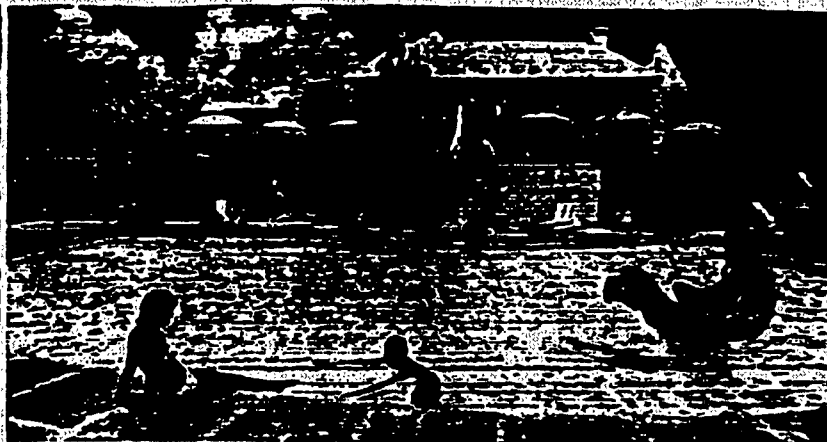
ings—are found in cars, trucks, tors, power shovels, hoists, and industrial machines, as are asbestos cloth packings, seals, and gaskets. Bestos papers and felts are used for roofing, for piano padding, for stoves, heaters, filing cabinets, helmets, and the mufflers and of automobiles, and they also are in armored cars, carpets, and bridges, and as boiler jackets and motor covers. Asbestos millboard, is a heavier version of asbestos, is used for acoustical ceilings, as terboard and fireproof wallboard, in electric switch boxes, safes, pads, stove mats, ovens, and dry. Vinyl and asphalt floor tiles contain anywhere from thirty to fifty per cent by weight, of short asbestos fibres. The floor-tile industry has become the second-largest user of the mineral. Since the development of phenol-formaldehyde resins in 1909, asbestos has been used in large quantities as a reinforcement and filler by the plastics industry, and today there are countless plastic products that contain asbestos, ranging from frying-pan handles to rocket nose cones. Asbestos is also an ingredient of many paints and sealants, of compounds used for road coating and road building, of putty, caulking, and other crack fillers, of clays used for pottery and sculpture, of artificial snow, of plaster and stucco, of fireproof insulation sprayed on structural steel, and of undercoating spray on automobile bodies.

As a result of these multiple and miscellaneous applications, asbestos has become practically ubiquitous in modern society. There is not an automobile, airplane, train, ship, missile, or engine of any sort that does not contain asbestos in some form or other, and it has found its way into literally every building, factory, home, and far across the land. And, because its minuscule fibres are eminently respirable, asbestos has also found its way into the lungs of man, where, by remaining indestructible as it does in nature, it wreaks terrible havoc.

THE adverse biological effects of asbestos were observed as early as the first century by the Greek geographer Strabo and by the Roman naturalist Pliny the Elder, both of whom mentioned in passing a sickness of the lungs in slaves whose occupation it was to weave asbestos into cloth. Strabo and Pliny were calling attention for the first time in history to a disease that would one day be known as asbestosis—a form of pneumoconiosis (

eral term for all dust diseases of the lungs caused by the inhalation of fine particles of asbestos—but the ancients were much too awed by the magic of the mineral to be concerned with the possibility that it might constitute a health hazard. Indeed, their attitude toward asbestos sometimes bordered on veneration. Strabo and Pliny noted that the "perpetual" wicks in the sacred and inextinguishable lamps of the vestal virgins were made of asbestos, and Pliny described asbestos cloth, which had been used for centuries in cremations, as the rare and costly "funeral dress of kings." It was generally assumed in ancient times that asbestos was of vegetable origin—they called it "linum usum" and a second-century Greek topographer Ptolemy referred to it as "the only cloth that is not consumed by fire"—and this misconception endured for centuries. (Only a few years ago, a major shareholder of an English asbestos company who was visiting the source of its supplies in South Africa asked the resident manager where the asbestos came from. The manager's explanation was: "The Romans were specially impressed by the fact that when cloth made of asbestos was exposed to flame it always came out whiter than before—hence the Latin word for asbestos, *amantus*, meaning unpolluted or undefiled—and they are said to have cleaned asbestos dinner napkins by tossing them into the fire.")

The use of asbestos in Europe appears to have diminished greatly during the Dark Ages, although it is said that the Emperor Charlemagne convinced some warrior guests from a rival kingdom that he possessed magic powers by throwing an asbestos tablecloth into the fire and then withdrawing it, unaltered, from the flames. In the latter part of the thirteenth century, when the defatigable Venetian traveller Marco Polo was traversing a part of Siberia then known as the Great Empire of Tartary, he was shown some cloth that would not burn, and was told by his Tartar hosts that it was made from salamander's wool. Too incredulous to be taken in by a story like that, Polo examined the material carefully and after making some inquiries, concluded that there was a mineral in the mountains of the district that contained threads like those of wool. In 1868 asbestos deposits were discovered in the Great Smoky Mountains, and forty years later a factory was built to manufacture asbestos products. Because the demand for the mineral were few, the demand was limited, this first commercial attempt to revive the long-



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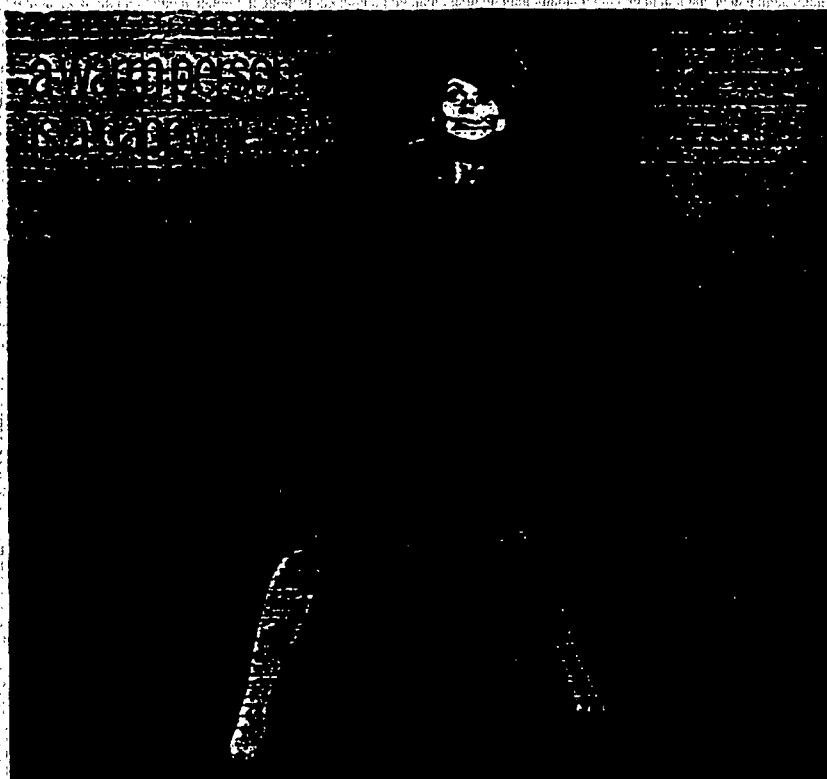


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dormant asbestos industry was but the second attempt, which placed slightly more than a cent in place beyond the wildest dreams of its backers. As a result, the asbestos could produce lung disease which had been forgotten since and Pliny first recorded it around the time of Christ, soon manifested again.

Modern knowledge of asbestos dates from the year 1900, when a mortem examination was performed by Dr. H. Montague Murray, a physician in London's Charing Cross Hospital, on the body of a thirty-three-year-old man who had worked for four years in an asbestos-textile factory. The patient, found to have been suffering from severe pulmonary fibrosis, which is scarring of the lungs, had been the last survivor of a group of ten men who were working in the carding room of the factory in 1886, and since Dr. Murray found spicules of asbestos in the lung tissues at autopsy, he was able to establish a presumptive connection between the man's occupation and the disease that killed him. A few years later, an inspector in the Department of Labor at Caen, whose name was Auribault, reported the results of what was undoubtedly the first study of mortality among asbestos workers. According to Auribault, whose account appeared in the *Bulletin de l'Inspection du Travail et de l'Hygiène Industrielle*, an asbestos-weaving mill had been established in 1890 at Condé-sur-Noireau, in Calvados, and during its first five years of operation, when there was no artificial ventilation and the employees were heavily exposed to dust from the looms, fifty of them died. Moreover, the director of the factory, who had previously owned a cotton mill at Gonneville, in Manche, had recruited seventeen workers from his former staff, of whom sixteen were wiped out by what Auribault, who was not a physician, mistakenly described as chalicosis—a pneumoconiosis common among stonecutters.

Unfortunately, neither Murray nor Auribault realized the importance of their findings. Testifying before a Departmental Committee on Compensation for Industrial Diseases, in 1906 Murray said, "One hears, generally speaking, that considerable trouble is now taken to prevent the inhalation of dust, and so the disease is not so likely to occur as heretofore," and the concluding recommendation of Auribault's report was simply that people under eighteen years of age should not be allowed to work in dusty conditions. Be-

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cause of ignorance about the cause of the disease, and because it manifested itself slowly and insidiously over a period of from ten to twenty years or more, asbestosis was probably more often than not misdiagnosed in the early part of the century as pulmonary tuberculosis, silicosis, or fibrosing pneumonia. In addition, cynicism on the part of industry, timidity on the part of labor, and obtuseness on the part of the medical profession often combined to make early investigations into the health of asbestos workers meaningless. As a result, even though the asbestos industry was growing very rapidly in all the industrialized countries of the world, almost nothing was done to control dust levels in asbestos factories, and almost no medical information on asbestosis was gathered during the quarter of a century that followed the disclosures of Murray and Aurihault—although asbestos workers were probably dying like flies of it.

It was 1924 before the first clear case of death due to asbestosis appeared in medical literature. In that year, Dr. W. E. Cooke, an English physician, who gave the disease its name, performed a post-mortem examination on a thirty-three-year-old woman patient who had started working at the age of thirteen in an asbestos-textile factory in which there was no provision for the removal of dust. By 1917, after thirteen years of exposure, she had been coughing and in bad health, and from then on her attendance at work had been intermittent because of shortness of breath and lassitude. The autopsy showed extensive pulmonary fibrosis and dense strands of abnormal fibrous tissue connecting the lungs and the pleural membranes surrounding them. In addition, Cooke noted the presence of solid yellowish-brown particles, which he called "curious bodies" in the areas of fibrosis. "We have never seen anything parallel to this in pneumoconiosis due to other dusts, nor have we been able to find such occurrence in literature," he declared, and he added, "We cannot think there is any reasonable doubt that the particles in the lung are the heavy, brittle, iron-containing fragments of asbestos fibre."

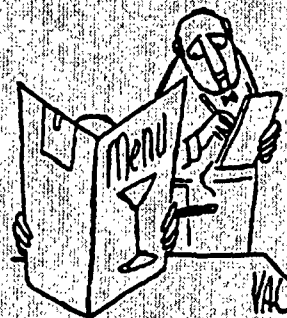
Cooke's discovery, which was published in the *British Medical Journal* of July 26, 1924, and republished in 1927, provided the point of departure

for a scientific journey that started on an apparently straightforward path of occupational hygiene and that had branched out into epidemiology, biology, physical chemistry, physiology, and experimental pathology. As a result of studies made between 1927 and 1931, a whole literature relating to asbestosis appeared in Great Britain which included clinical and X-ray findings, descriptions of the histology of the disease, and the hypothesis (soon accepted) that the strange-looking, yellowish-brown bodies noticed by Cooke were asbestos fibres that had been altered by the reaction of lung tissue, coated with a colloidal substance, and in fact, Cooke's "curious bodies" proved to be so abundant in the lungs of autopsied asbestos workers that many of which contained literally millions of them, that they quickly came to be called "asbestos bodies."

The most important investigation of this period was conducted by Dr. E. R. A. Merewether, a medical inspector for the Factory Department of the British Home Office. Between 1927 and 1929, he examined three hundred and sixty-three asbestos-textile workers out of an estimated total of twenty-two hundred asbestos workers employed in Great Britain at the time, and found that ninety-five, or slightly more than twenty-five per cent, showed evidence of suffering from pulmonary fibrosis. He also demonstrated that the incidence of fibrosis increased in direct proportion to the number of years of exposure, reaching eighty-one per cent in the group of workers who had been employed in the industry for twenty years or more. As a result of Merewether's report, Parliament passed legislation in 1931 that made asbestosis a compensable disease.

required improved methods of exhaust ventilation and dust suppression in asbestos-textile factories, and instituted periodical medical examinations for workers engaged in particularly dusty processes in the asbestos-textile industry.

After this promising burst of activity, however, progress in the field of asbestosis was depressingly slow, for, as things turned out, the doctors had only begun to scratch the surface of a problem of bewildering complexity. Many investigators assumed that the new regulations would prove effective in controlling the disease, thus failing to appreciate the fact that, once inhaled



Asbestos, which is virtually indestructible, continues to react within the lungs for a lifetime, and to heed the warning of Dr. Merewether, who, when asked if two years of exposure was sufficient to cause asbestosis in a young girl, replied, "Yes, if she lives long enough." Moreover, techniques of dust sampling were primitive and inaccurate, and dust levels varied so greatly—not only from factory to factory but from area to area within a given factory—that it would have been almost impossible to establish criteria upon which to recommend a level of exposure. But as time went by, the generally lowered dust levels that did result from the regulations of 1931 appeared to be slowing down the attack rate of the disease, at least in Great Britain, and the life expectancy of the average asbestos-textile worker was further increased by the advent of antibiotics, which drastically reduced the death rate from infectious pulmonary diseases that often had to be feared as a complication of asbestosis. The totally unforeseeable consequence of all this was that asbestos workers, instead of dying of asbestosis, began living long enough to develop asbestos-induced cancer.

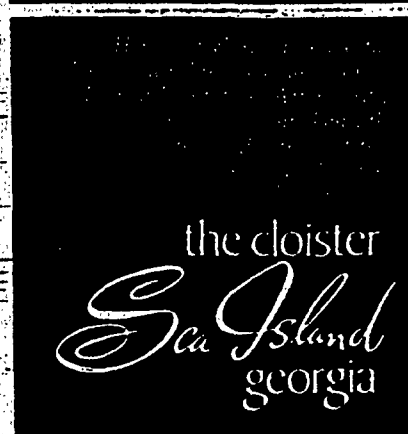
The first published suggestion of an association between asbestosis and malignant disease was made in 1935 by Dr. Kenneth M. Lynch, professor of pathology at the Medical College of South Carolina, who described the case of a fifty-seven-year-old asbestos weaver who died of asbestosis and in whose lungs cancer was also found. Because the regulations of 1931 required British coroners to conduct autopsies on men who had been suspended from work with asbestos on account of pulmonary disability or when their length of service rendered it likely that their occupation was a contributory cause of death, pathologists in Britain were able to investigate the suggested link between asbestosis and lung cancer much more easily than their counterparts in the United States, where few states had workmen's compensation covering dust diseases, and where there were no specific regulations requiring post-mortem examinations of asbestos workers. The British investigations were not made the subject of public comment until 1949, however, when Dr. Merewether, who was then Inspector of Factories, published a review of data that had been accumulated from 1924 to 1946, which revealed that lung cancer was present in thirty-one out of two hundred and thirty-five deaths that were caused by asbestosis or in which asbestosis was

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found at autopsy. This was an incidence of slightly more than thirteen per cent, and Merewether found it highly significant, for lung cancer, according to its incidence in the general population, should only have been present in approximately one per cent of the cases. At the time, no formal claim of an association between the two diseases was made, but during the next few years an increasing amount of evidence began to establish one. Then, in 1955, Dr. Richard S. Doll, director of the Statistical Research Unit of the British Medical Research Council, completed a study of a hundred and thirteen consecutive autopsies of men with more than twenty years of exposure in a large asbestos-textile factory, which showed that the extent to which they had risked lung cancer over the whole period was eleven times as great as that of men in the general population of England and Wales. From then on, many doctors were convinced that a causal relationship between asbestos exposure and lung cancer had been demonstrated, although almost no part of the vast asbestos industry either acknowledged the relationship or found it advisable to aid the independent researchers who were investigating it.

AT this point, many epidemiologists had concluded that the hazards to human health presented by asbestos were limited to the relatively small group of heavily exposed workers engaged in making asbestos textiles. The trouble was that very few studies had been made of the tens of thousands of workers engaged in other branches of the industry, which was then expanding at an enormous rate all over the world, so that no one really had any way of knowing whether there was any danger from moderate or light occupational exposure—or, for that matter, from indirect, non-occupational exposure. Such studies were under way in a number of countries, however, and when the results began to appear, in the late fifties, it became evident that the hazard posed to man by asbestos had once again been underestimated.

The most dramatic development was the discovery of an association between exposure to asbestos and mesothelioma, an almost always malignant tumor of the pleura, which is the delicate membrane that encases the lungs, or of the peritoneum, a similar membrane that lines the abdominal cavity. Previously, mesothelioma had been so rare that it was considered a pathological curiosity. It was found in only one out of every ten thousand autopsies. It was

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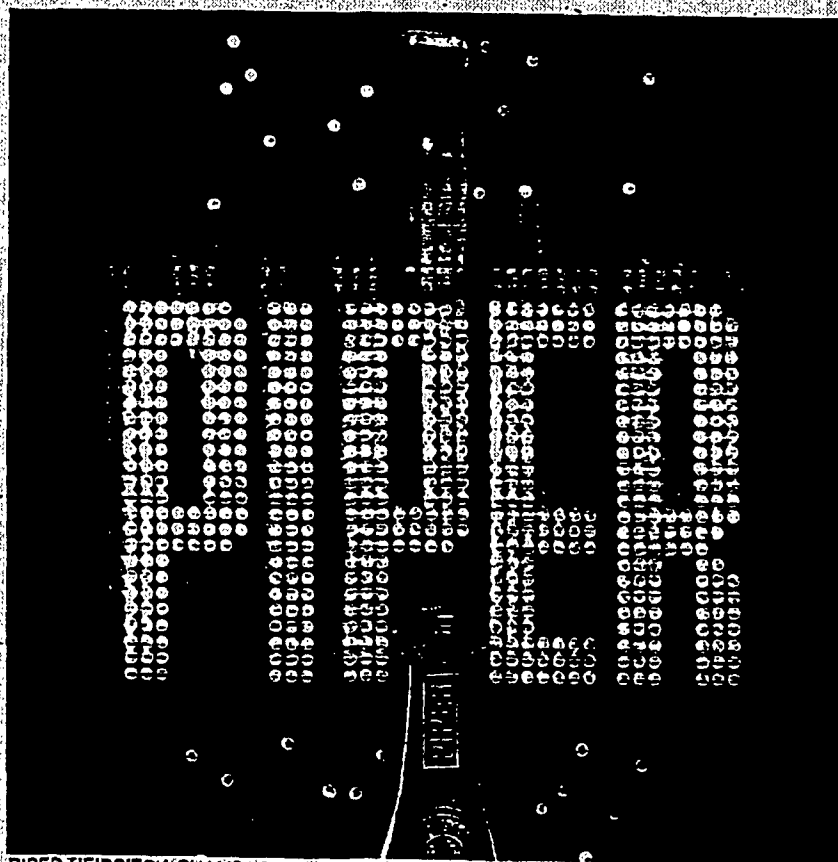
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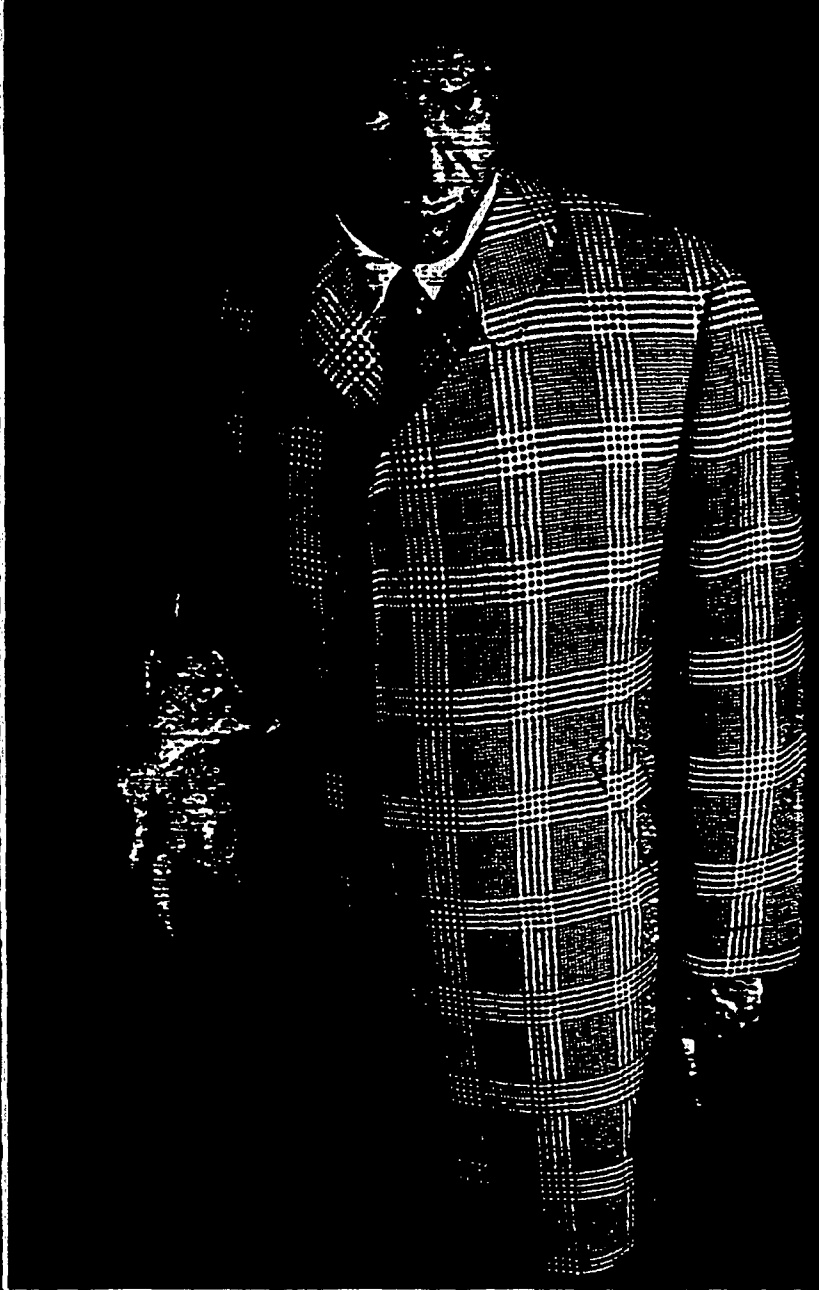
not listed in most medical books; it was not even cited separately. World Health Organization's International Classification of Diseases, 1956 and 1957, however, sixteen of this rare tumor—tantamount to an epidemic—were diagnosed at the Mesothelioma Research Unit in Johannesburg, South Africa, by a pathologist named J. Christopher Wagner. The circumstances led Dr. Wagner and his associates to suggest that asbestos may play some part in the mysterious break: asbestos bodies had been found at autopsy in the lungs of the very first case, and ten of the cases had been referred from a hospital to which patients suspected of suffering from tuberculosis were referred from a large crocidolite asbestos mining area.

At the beginning, this hypothesis seemed rather tenuous, for none of the sixteen patients who suffered from mesothelioma had ever worked in asbestos mines or mills. Within a short period, however, cases of mesothelioma began to crop up elsewhere in South Africa, and when Wagner and his associates discovered that the father of one of their new mesothelioma patients had been the manager of an asbestos mine and that the patient had played around the mine and on the adjacent dumps as a child, they undertook to reinvestigate the sixteen original cases and to obtain detailed life histories in all future cases. By the end of 1961, they had diagnosed a total of eighty-seven mesotheliomas of the pleura and two mesotheliomas of the peritoneum. Only twelve of the cases proved to have had industrial exposure to asbestos, but all the rest came from the windswept region of the Cape asbestos fields, where they could easily have obtained exposure simply by living in the vicinity of asbestos mills and dumps. A particularly striking case was that of a woman who was born in a town on the asbestos fields, left the region at the age of five, and died of a pleural mesothelioma at the age of fifty-seven. Detailed questioning about her childhood revealed that she had attended a kindergarten near an asbestos dump and that she and her playmates (two of who also died of mesothelioma) used to enjoy sliding down the dump on their way home from school. Another example was provided by the youngest patient of the series—a man who died at the age of twenty-one. He had been exposed to asbestos from the age of three weeks until he was weaned—a period during which his mother took him along with her when she went to work at a cobbing site, which is a place where

Asbestos is separated from its host rock. Among those patients who had worked in asbestos mines, the actual exposure appeared to have been slight. In most cases the lungs showed no evidence of asbestosis but only a few asbestos bodies and fibres in the air spaces. From this, Wagner drew the chilling conclusion that mesothelioma might result from an exposure to asbestos dust that was considerably less than that needed to produce fibrosis—the condition that had always been considered essential for the development of asbestos lung cancer. (In some areas of South Africa, occupational exposure to asbestos was so heavy, however, that fibrosis of the lungs was almost certain—killing people before cancer could develop. According to Dr. G. W. H. Schepers, a native of South Africa who is now director of the Bureau of Laboratories in the Department of Health in Washington, D.C., conditions of industrial hygiene in the asbestos mines and factories were often “deplorable.” In 1949, when Schepers was sent into the northeastern Transvaal to make the first official radiological and clinical survey of the asbestos industry for the South African government, he found barefoot Bantu children spending their days inside large jute shipping bags, where they were forced by overseers armed with whips to trample down fluffy amosite asbestos as it came cascading over their heads into the bags. Schepers found that a number of these children had developed asbestosis with cor pulmonale—right-sided heart failure, which often accompanies the disease—before the age of twelve, and he has said that he would be very surprised if any of them had lived long enough to develop either lung cancer or mesothelioma.)

In 1960, the year that Wagner published the first of his disturbing reports about mesothelioma, there were two additional developments that caused grave concern among scientists investigating the asbestos problem. One of them occurred in Finland, where Dr. Raimo Kiviluoto, a radiologist, had been surveying the general population for tuberculosis; he detected deposits of calcium on the pleura, which had previously been seen almost exclusively in asbestos workers, in four hundred and ninety-nine out of sixty-three hundred and twelve people (mostly farmers and their wives) who lived in a county where asbestos had been mined and milled for many years, and no pleural calcifications in seventy-one hundred and one people who lived in a neighboring county where there was no as-

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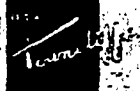
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bestos industry. The other development took place back in South Africa, where Dr. J. G. Thomson, professor of pathology at the Medical School of the University of Cape Town, was examining lung-fluid smears from five hundred consecutive autopsies to determine the extent to which the ordinary urban dweller was exposed to the inhalation of asbestos; he found asbestos bodies in twenty-five per cent of the total. Thomson's discovery astonished the medical world, for up until that time no one suspected that asbestos bodies might be present in the lungs of people who did not work in asbestos mines or factories. He pointed out, however, that there had been a tremendous increase in the use of asbestos throughout the world over the previous twenty years—three million tons were marketed in 1960—and that asbestos was being incorporated into an increasing number and variety of industrial products with which the ordinary urban dweller came into intimate daily contact. He also suggested that the air of city streets was a likely source of asbestos exposure, and that the average motor vehicle, which wears out three or four sets of asbestos brake linings and one or two asbestos clutch facings in its lifetime, might well be a major cause of the liberation of asbestos dust. Thomson reminded his colleagues that, compared to a half-life of twenty-eight years for strontium 90, the half-life of asbestos fibre was an infinity of years, and he concluded that, because of the continuous disintegration of asbestos products such as brake linings, floor tiles, and roofing materials, as well as the demolition of buildings containing numerous other asbestos products, asbestos was constantly finding its way into the atmosphere as an everlasting contaminant. He even went so far as to predict that in future decades, as the accumulation of asbestos in urban areas led inexorably to higher concentrations of asbestos fibres in the lungs of urban dwellers, asbestos-induced neoplasms would rival cigarette-induced lung cancer as a cause of death.

As might be expected, the disclosures made by Wagner, Kiviluoto, and Thomson provided a powerful stimulus for further investigation of the problem of asbestos exposure. During the next few years, a hunt for mesotheliomas and asbestos bodies was undertaken by pathologists all over the world.

Thomson's observations were confirmed by studies made in Miami, London, and in Belfast, while in Edinburgh asbestos bodies were found in lungs of forty-six of a hundred consecutive autopsies at Presbyterian University Hospital. And Wagner's findings were corroborated by investigations that associated mesothelioma exposure to asbestos in half a dozen cities in Europe and the United States. Perhaps the most striking confirmation of Wagner's data came from London, where Dr. Muriel L. Newhouse, Dr. Hilda Thompson, of the Department of Occupational Health at the London School of Hygiene and Tropical Medicine, investigated the life histories of seventy-six cases of mesothelioma that had been confirmed by biopsy or biopsy in London Hospital. To no one's surprise, thirty-one of the seventy-six patients had worked with asbestos, but eleven of those who had died had simply lived within half a mile of an asbestos factory, and nine others—seven women and two men—were relatives of asbestos workers. Most of these women had washed their husbands' work clothes regularly. The investigators learned that one of the husbands, a docker, had come home "white with asbestos" every evening for three or four years, and that his wife had brushed him down. Both of the men in this group, when born of eight or nine years, had had sisters who worked in asbestos-textile factories. One of the sisters had been employed as a spinner from 1925 to 1936 and had died of asbestosis in 1947, at which time it was determined at an inquest that "she used to return home from work with dust on her clothes." Her brother, who had apparently sustained no other exposure to asbestos in his lifetime, died in 1956 of a pleural mesothelioma.

Further corroboration of Wagner's findings came from southeastern Pennsylvania, where Dr. Jan Lieben, then head of the Division of Occupational Health at the State Department of Health, in Harrisburg, investigated the backgrounds of forty-two people who had died of mesothelioma between 1958 and 1963. Of the forty-two patients, ten proved to have had definite occupational exposure to asbestos, three were relatives of asbestos workers, eight either lived or were employed in the vicinity of an asbestos factory, and ten



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others were discovered to have had
less some minor exposure to the
eral. The ten people with
exposure included a self-employed
maker of Swiss cheese who used a
hundred-gallon boiler covered with
asbestos insulation that flaked off
contact, a salesman who had mixed and
applied asbestos-cement insulation
boilers in his home, and a fourteen-
year-old boy who at one time
helped his father saw up and install
plasterboard during an extensive
modelling of their house.

From an epidemiological point
view, the investigations that tended
support the findings of Wagner and
Thomson were weak, because they
lacked a defined population as a base
against which the data could be mea-
sured. On the other hand, the presence
of asbestos bodies in the lungs of such a
large percentage of consecutive auto-
psies in so many widely distributed cor-
ners of the world was sufficient to indicate
that asbestos fibres were probably being
inhaled by a majority of urban dwell-
ers everywhere, and the association
between asbestos and mesothelioma
was striking enough to suggest that a
history of exposure to the mineral
was likely to be found whenever a
positive diagnosis of the tumor was
made. As a result, epidemiologists were
forced to revise their idea that asbestos
was only an industrial hazard, and to
give serious consideration to Thom-
son's prediction that it might prove
to be dangerous for untold numbers
of unsuspecting people in the general
community.

IN October of 1964, in order to re-
view the data collected in the past
and to discuss the problems awaiting
solution in the future, the New York
Academy of Sciences sponsored an in-
ternational Conference on the Biologi-
cal Effects of Asbestos, at the Waldorf-
Astoria. The conference enabled more
than four hundred scientists from all
over the world to discuss the immen-
sely complicated epidemiological and
clinical problems that had appeared in
the forty years since Dr. Cooke de-
scribed the first clear case of asbestosis
and its planning was largely the work
of one of its co-chairmen, Dr. Irving
J. Selikoff, who is head of the Divi-
sion of Environmental Medicine at
the City University's Mount Sinai
School of Medicine, director of its
Environmental Sciences Laboratory
and a pioneer in the field of moder-
n asbestos epidemiology.

Like many of his colleagues at the
conference, Dr. Selikoff became inter-

ated in asbestos in a roundabout way. A native of New York City, he interned at Beth Israel Hospital in New York, did his pathology at Mount Sinai, where he has been a member of the staff since 1947, and became a chest physician at the Sea View Hospital on Staten Island, specializing in tuberculosis. In 1951, he participated in the research on isoniazid—the anti-tubercle drug that, by effectively killing tubercle bacilli, has provided a cure for tuberculosis—for which he received the American Public Health Association's Albert Lasker Award in Medicine. In 1953, Dr. Selikoff founded a medical clinic in Paterson, New Jersey, where, by chance, seventeen of his early patients were men referred for chest-disease consultations who worked in a local factory that had been making asbestos-insulation materials since 1940. When the factory closed down in 1954, the seventeen men went into other work, and at that point Dr. Selikoff decided to continue his observation of these patients with X-ray examinations and pulmonary-function tests in order to determine the history and the natural course of asbestosis in men who would not be subjected to further exposure.

At the time, Dr. Selikoff was chiefly interested in asbestosis because, as he has explained, "I was still unconvinced of the relationship between asbestos and lung cancer. There wasn't much data, and what there was scarcely seemed to indicate that it would prove to be much of a problem." As things turned out, he changed his mind. In 1954, all seventeen men from the Paterson factory were able-bodied and working. By 1961, however, six of them were dead, and today only six of them are alive. Of the eleven who died, four were victims of lung cancer, three of other cancers, one of mesothelioma, and two of asbestosis. Of the six who are still alive, two are disabled because of respiratory insufficiency caused by fibrosis of the lungs, and one has recovered from surgery for cancers of the lung and larynx. Dr. Selikoff shook his head in describing the fate of these patients, but as early as 1961 their ranks had been thinned out so drastically that, as he said, "I was forced to the awful realization of what could happen to men who were occupationally exposed to asbestos." After the company that owned the factory was unable to make its employment records available, he wrote to other large asbestos manufacturers in the United States, inquiring about the health experiences in their

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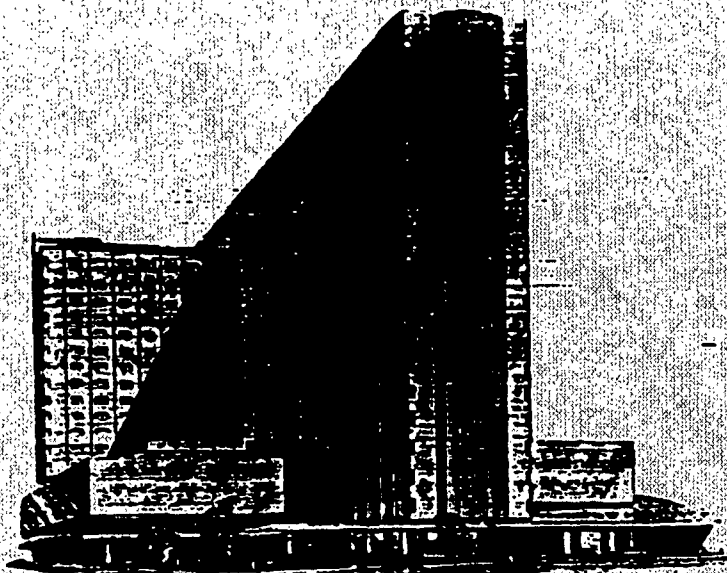
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plants, and was unable to obtain information from any of them. Meanwhile, he and Dr. Jacob Churg, a pathologist at Barnert Memorial Hospital, in Paterson, who had been finding asbestosis and lung cancer in a large number of workers from the Paterson factory, took their data to Roscoe P. Kandle, Commissioner of the New Jersey State Department of Health, and Dr. Kandle applied to United States Public Health Service funds to undertake a study of the Paterson factory and to make a statewide survey to determine how many people were being occupationally exposed to asbestos. However, the request was denied for lack of resources.

Since Dr. Selikoff already knew the men who had worked in the factory that made insulation materials were dying of asbestosis and lung cancer at an alarming rate, he considered the men who were installing such materials might also risk disease, and early in 1962 he made contact with officials of New York Local 12 and Newark Local 32 of the International Association of Heat and Frost Insulators and Asbestos Workers. The asbestos insulators had been trying for years to interest doctors and various government agencies in their medical problems, so they were only too glad to cooperate, and urged Dr. Selikoff to make a study of the depth of the effects of asbestos exposure among their members.

During the next six months, Dr. Selikoff and his administrative assistant Mrs. Janet S. Kaffenburgh, poring through the unions' records and compiled a list of the names and addresses of the six hundred and thirty-two men who had been members of either of the metropolitan locals on December 31, 1942, and of the eight hundred and ninety men who had joined between then and December 31, 1962. From the unions' employment records, they obtained detailed work histories of the total membership of fifteen hundred and twenty-two men, including their withdrawal from work for other employment, war service, retirement, or illness. This enabled them to calculate the onset and duration of exposure to asbestos for each worker. Records of the unions' health and welfare funds provided them with the dates and places of death of the two hundred and sixty-two workers who had died between 1942 and 1962, and copies of the death certificates of all but one of them were obtained. In addition, autopsy protocols, histological specimens, and hospital records were reviewed by Dr. Selikoff and Dr. Churg in the

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deaths (approximately one-half of the total) which occurred in hospitals.

In the next phase of the study, Dr. Selikoff and Dr. Churg were joined by Dr. E. Cuyler Hammond, Vice-President for Epidemiology and Statistics of the American Cancer Society, who had participated in an analysis of the medical effects of the atomic explosions that devastated Hiroshima and Nagasaki, in 1945, and whose large-scale epidemiological studies of more than a million men and women provided a major basis for the conclusions drawn in the 1964 Surgeon General's report on the effects of cigarette smoking. Since previous studies had suggested that lung cancer associated with asbestosis seldom develops until twenty years after initial exposure to asbestos dust, Dr. Selikoff and Dr. Hammond decided to limit their first report to the six hundred and thirty-two men who were on the union rolls as of December 31, 1942. Taking the men's ages into consideration, Dr. Selikoff and Dr. Hammond then set about comparing the number and causes of death among them with those of the general male population in the United States. The results were depressing. According to the standard mortality tables, two hundred and three deaths could have been expected among the six hundred and thirty-two workers. Instead, there were two hundred and fifty-five, not counting seven men who had died before incurring twenty years of exposure—an excess of twenty-five per cent. The reason for the excess was not hard to find. The fact that twelve of the deaths were attributed to asbestosis was not particularly surprising, but only six or seven deaths from cancer of the lung, pleura, or trachea were to be expected, whereas there were forty-five. And where nine or ten gastro-intestinal cancers were to be expected there were actually twenty-nine. Since the lung-cancer death rate was known to be more than ten times as high among cigarette smokers as among non-smokers, Dr. Selikoff and Dr. Hammond realized that they would have to take the smoking habits of the asbestos-insulation workers into account if their findings were to have any validity. It was, of course, impossible for them to ascertain this with accuracy in the cases of the two hundred and fifty-five men who had died, so, for purposes of calculation, they assumed that all six hundred and thirty-two men had smoked a pack or more of cigarettes each day, and they demonstrated that even if this had been the case it would have produced a lung-cancer death rate

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only three and a half times that of the general male population. Cigarette smoking, therefore, could not explain the fact that in this group of asbestos-insulation workers the rate of death from lung cancer was seven times the expected rate.

Because of its objectivity, its scope, and its thoroughness, this study conducted by Drs. Selikoff, Churg, and Hammond had a great impact on the medical community when it was published, in the spring of 1964, in the *Journal of the American Medical Association*, and when it was delivered before the Conference on the Biological Effects of Asbestos that October. It was the first study ever made that had taken a large enough group of asbestos workers from a point far enough back in time and followed them long enough to determine unequivocally what their health experience had been. Unlike almost all the previous investigations, which simply indicated that there was an association between asbestos and various kinds of cancer, it was based upon the incidence of disease within a defined population, and thus answered a fundamental epidemiological question of how many cancers had developed out of how many persons exposed. In doing so, it furnished the first incontrovertible evidence that industrial exposure to asbestos was hazardous; it established sound methodology for future studies; and it marked a turning point in the views held by doctors and health officials around the world.

In the autumn of 1962, Dr. Selikoff—using donations from private sources and a grant awarded by the Health Research Council of New York City, headed by Dr. George S. Mirick—had set up informal clinics in union halls in New York and Newark and started giving X-ray examinations, pulmonary-function tests, and blood tests to the members of the two asbestos workers' locals. During the next year, he examined seven hundred and seventeen workers, and he also questioned them closely about their smoking habits, the kinds of materials they used, and the conditions under which they worked. "The insulation workers, an intelligent and highly skilled group of craftsmen, particularly educated me concerning their work," he once said. "I learned that they had comparatively light and intermittent exposure as asbestos workers because they often worked out on construction projects, and most of the materials they used had an asbestos content of less than five

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teen per cent. I also found out how their problems had been neglected. Most of them had never seen a labor inspector in their lives, and only a handful could remember seeing anyone make a dust-level count in the areas where they were at work." Dr. Selikoff also learned that although some of the workers knew they ran a risk of developing asbestosis (which they usually referred to as silicosis) almost none of them had any idea that they also risked cancer because of their occupation. As things turned out, he found radiological evidence of asbestosis in fully half of the eleven hundred and seventeen men. The extent of the fibrosis varied directly with the duration of exposure. Of three hundred and forty-six men whose exposure had begun less than ten years before, only ten per cent showed any abnormality, and of three hundred and seventy-nine men who had been exposed for between ten and nineteen years more than half still had normal X-rays; in neither group was there any discernible evidence of cancer. On the other hand, Dr. Selikoff found that among three hundred and ninety-two men with more than twenty years of exposure three hundred and thirty-nine had developed asbestosis, which had by that time become moderate or extensive in more than fifty per cent of the cases; he also found that eleven of the men had developed cancers of the lung or pleura.

Aided by yearly grants from the Health Research Council, Dr. Selikoff has maintained a close watch on the asbestos workers' health, paying particular attention to those men with more than twenty years of exposure by examining them once or twice a year. As a result, he has been able to detect symptoms of illness in many of the men much earlier than they might have been detected and to observe with great accuracy the mortality experience of the three hundred and seventy whom he found in 1963 to be survivors of the original six hundred and thirty-two members of the union in 1942. It continues to be disastrous. Between January of 1963 and March of 1968, fifty deaths would normally have been expected to occur among these men. Instead, there were a hundred and thirteen. Fifteen of them were caused by asbestosis, and, as with the earlier study, the rest of the excess turned out to be caused by cancer of various kinds. Two or three bronchogenic cancers would have been normal, but twenty-eight occurred, and where the actuarial tables predicted only two gastro-intestinal cancers there were eight. An un-

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expectedly sharp rise in the number of mesotheliomas was observed. In the earlier study, there had been only four in two hundred and fifty-five deaths; between 1963 and 1968, however, thirteen of these rare tumors were found in a hundred and thirteen deaths. Dr. Selikoff suspects that the latent period for mesothelioma may be longer than that for either asbestosis or lung cancer, but so far he regards the heavy increase of mesothelioma among the insulation workers as something of a mystery.

Dr. Selikoff and Dr. Hammond found special significance in the fact that all twenty-eight bronchogenic carcinomas occurred among asbestos workers who smoked cigarettes regularly. None were found among eighty-seven men who never smoked cigarettes. Dr. Selikoff and Dr. Hammond have reservations about these data, which suggest that asbestos workers who smoke cigarettes are ninety times more likely to die of lung cancer than men who neither work with asbestos nor smoke, because they are based upon the experience of a relatively small number of people. They do conclude from it, however, that an important co-carcinogenic effect exists between asbestos exposure and cigarette smoking, and in a recent article published in the *Journal of the American Medical Association* they recommend "that asbestos exposure be minimized, that asbestos workers who do not smoke should never start, and that those now smoking should stop immediately." It has been suggested in some quarters that asbestos fibres, which are known to be highly adsorbent as well as virtually indestructible, may retain the substance known as benzo[a]pyrene from cigarette smoke for long periods of time in lung tissue, thus tending to produce lung cancer by a synergistic effect. No one knows for sure, however, and Dr. Selikoff will not hazard a guess about the matter until more information is gathered. He expresses certainty about the effects of asbestos exposure only when he has epidemiological proof. He knows, for example, that one out of every five asbestos-insulation workers who die in the New York-New Jersey metropolitan area dies of lung cancer, and that almost half of the three hundred and sixty-eight deaths he has recorded among these men in the past twenty-five years have been caused by cancer of one type or another. Last March, citing these figures before a House subcommittee on labor, he warned that "if they are projected to the country as a whole, and if no im-

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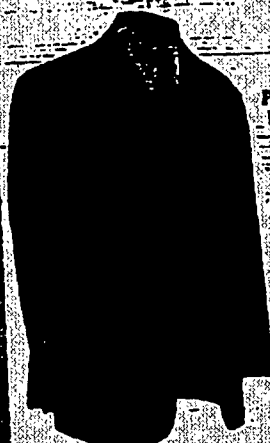


Improvement in the health hazards these men face is achieved, we may look forward to perhaps six thousand deaths from lung cancer and to perhaps fifteen thousand deaths from cancer in general among men in this single asbestos trade alone." Considering that the total membership of the International Association of Heat and Frost Insulators and Asbestos Workers is only eighteen thousand men, and that there are about the same number of non-union asbestos workers, his prediction has staggering implications.

I RECENTLY spent several days with Dr. Selikoff in the Environmental Sciences Laboratory at Mount Sinai, where he has assembled a team of thirty men and women to work on various aspects of the asbestos problem. The rooms include laboratories for histology, pathology, electron microscopy, chemistry, and mineralogy, and among the imposing array of equipment they contain is an electron microprobe that is capable of identifying particles of infinitesimal size. Dr. Selikoff's office is large and modern. Its walls are lined with shelves and cabinets containing hundreds of bound volumes of research papers and reports on asbestos, and at one end are a large conference table and a blackboard. The place gives an immediate impression of rather cold efficiency, but this is tempered to a great degree by the affability of the director. As we talked, Dr. Selikoff, a large, soft-spoken man in his early fifties, was frequently interrupted by telephone calls and by questions from staff members, but he generally resumed—often with an enthusiastic "Where were we?"—and rattled off statistics about asbestos disease the way some baseball fanatics recite the batting averages of big-league ballplayers. On the day of my first visit, however, he received a call from a doctor in New Jersey who informed him that a lung cancer had just been diagnosed in another of the insulation workers. Dr. Selikoff told the physician that the patient's last X-rays showed moderate asbestosis, that he had had more than twenty years of exposure, and that he smoked a pack of cigarettes a day. When he hung up the phone, he was visibly upset, and fell silent. A few moments later, he told me that he had taken more calls like that than he cared to remember. "I don't relish the role of Cassandra, but I feel we've got to forge ahead on this problem as quickly as possible," he said. "There are about thirty-six thousand insulation workers—union and non-union—in the



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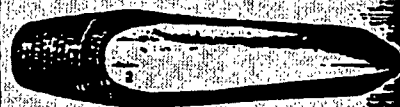


United States. All in all, including asbestos-textile workers and people engaged in manufacturing other asbestos products, there are probably more than a hundred thousand workers who are regularly and directly exposed. The hazard would be serious enough if this were the only group at risk, but it's obvious that the problem extends further, simply because floating asbestos fibres do not respect job classifications. Dr. William H. Stewart, who is the Surgeon General of the United States, told the House subcommittee that the asbestos-insulation workers are probably sharing their exposure to some extent with more than three and a half million other construction workers—steam fitters, electricians, welders, carpenters, ironworkers, plumbers, masons, tile setters, and the like—who are employed on the same projects, often in the same room or general area, and who therefore must be inhaling the same dusts."

Because of their concern with the possible effects of such indirect occupational exposure, Dr. Selikoff and his assistants have spent months compiling the names, addresses, and work histories of twelve hundred tile setters, three thousand roofing workers, and three thousand tunnel, foundation, and caisson workers in the New York metropolitan area. They plan to examine these men for possible health problems as they did the insulation workers, and to determine whether they have significant indirect occupational exposure, and, if they do, whether it is associated with risk of asbestos disease. "We will study defined populations that have been selected in terms of what appear to be diminishing levels of exposure to a known risk," Dr. Selikoff told me. "It's as if a rock had been thrown into a pond and we're watching how far the ripples extend. Since the pond in our case is the general community, you can see how difficult the epidemiological detective work will be. If we discover, for example, that the tile setters run a significant risk of developing asbestos disease—and there are some preliminary indications that they might—then we must look into the question of fibre fallout from the construction sites where they're employed. We're not going to wait for the results of the tile-setter study, however, because the problem is too urgent. We're initiating a whole cluster of studies designed to provide information on whether environmental asbestos pollution carries a risk for the general population. At the same time, by analyzing the ripples, we can determine which rocks thrown into the pond

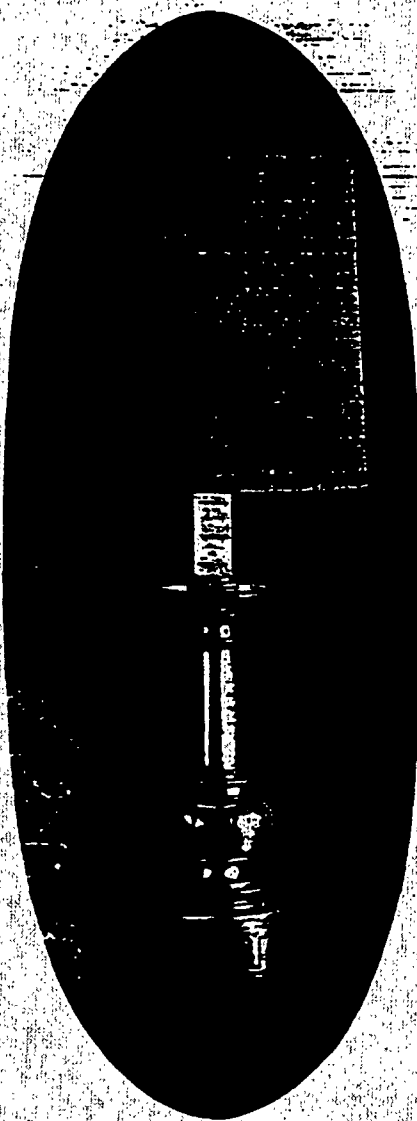


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make the biggest splash. By thus identifying the sources of contamination, appropriate control measures will become feasible. Our plan of attack is to discover where asbestos-associated diseases may occur, and to control the sources in such a way as to eliminate them as a hazard. It must be a team effort, of course, including epidemiologists, basic scientists, engineers, public health officials, and industry and labor."

One of Dr. Selikoff's studies will examine the possible health problems arising from interrupted exposure by tracing thirteen hundred and sixty-seven men who worked at the asbestos-insulation plant in Paterson—closed down in 1954—where Dr. Selikoff's first seventeen patients were employed. Another will look into the matter of neighborhood exposure by tracing and investigating the health experience of seventy-five hundred people who lived within a half-mile radius of the plant in 1942. "The question of neighborhood exposure was emphasized as a result of the mesotheliomas discovered by Christopher Wagner in South Africa, and later by Molly Newhouse and Hilda Thompson in London and by Jan Lieben in Pennsylvania," Dr. Selikoff told me. "Since current evidence suggests that the tumor rarely occurs without a history of exposure to asbestos, epidemiologists consider it a 'marker' disease, and we'll be looking for it when we trace the medical history of the people in Paterson. Incidentally, there's one preliminary indication that leads us to suspect we may find it. In 1955, the proprietor of a junk yard next to the Paterson insulation plant died of a pleural tumor." A third study in Dr. Selikoff's cluster will aim at the protection of relatives of asbestos workers. As in the case of neighborhood exposure, previous investigators of this group had no population base against which to measure the occurrence of the disease they discovered. Dr. Selikoff, however, has a definitely established group to work with. It is composed of the wives and children of all the insulation workers, and their health problems will be carefully scrutinized, especially for evidence of mesothelioma. In Dr. Selikoff's opinion, this study will be of tremendous importance, because its subjects have been far removed from direct exposure. "If the families of insulation workers turn out to be in trouble, the implications will be very serious indeed," he said. "On the other hand, if we find no trouble among them—and this is my hope—we'll be on safer ground. Our observations will also enable us



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to recommend whether special facilities should be provided so that insulation workers can change their work clothes before going home to their families."

With the aid of a grant from the United States Public Health Service, Dr. Selikoff and his associates are currently conducting an investigation that covers an even wider range, by studying samples of lung tissue from three thousand consecutive autopsies that have been performed at three different hospitals in New York City and tracing the work histories of each of the dead people. So far, they have examined two thousand lung samples, and have found asbestos bodies in nearly half of them. According to Dr. Victor Baden, a histologist at the Environmental Sciences Laboratory, from one to five or more asbestos bodies are found in fifty per cent of the tissue slides he examines. Since each slide is composed of seven sections that together represent less than one half-millionth of an entire lung, and since it is widely assumed that asbestos bodies are evenly distributed throughout a lung, the finding of just one of them can be projected to mean that half a million may be present.

Dr. Selikoff and his fellow-epidemiologists do not know whether the relatively small amount of asbestos now being inhaled by the general public carries a risk of inducing mesothelioma, or whether the presence of what appear to be asbestos bodies in the lungs of many people in the general population represents a serious public-health problem. Nor do they know whether relatively light inhalation of asbestos is associated with a special lung-cancer risk in cigarette smokers. "At present, we simply have no way of telling what the dose-disease relationships are," Dr. Selikoff said. "Is it better to have breathed in only fifty asbestos fibres than fifty thousand or fifty million? Does each fibre have the potential of causing a cell with which it comes in contact to turn cancerous? The answers to these immensely complicated questions will have to come from epidemiology over a long period of time. An eightfold increase in asbestos production over the last thirty years would appear to make it likely that asbestos cancers will increase, but only the future will tell. Remember that current cases of mesothelioma must be associated with inhalation that took place twenty, thirty, or more years ago, when world consumption of asbestos was only about five hundred thousand tons a year, and that the neoplasms associated with today's production of over four million tons a

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will not be evident until the nine-
ties. Moreover, we have
found that all varieties of asbestos
may appear to be implicated in meso-
thelioma, and not just crocidolite, as
some of us had hoped following Wag-
ner's observations in South Africa. The
problem would have been simple if
only one variety of asbestos had been
linked to this tumor. Control proce-
dures could have been narrowly fo-
cused. As it is, however, a control pro-
gram will have to be concerned with
asbestos in general."

I asked Dr. Selikoff if he thought
that asbestos air pollution was respon-
sible for the occurrence of asbestos
tumors in the lungs of people in the
general population, and he replied that
although there was relatively little
direct confirmation, he felt it was at
least partly responsible. Pointing out
again that direct and indirect occupa-
tional exposure were much more com-
mon than had been suspected, especially
in the construction and shipbuilding
industries, he went on to say, "The
possible sources of environmental con-
tamination are numerous. Mining
and milling operations, manufacturing
processes, and the delivery and storage
of finished products constitute just one
part of it. The constant use of asbestos
materials in the construction industry is
another, and the destruction and de-
terioration of asbestos products in the
community at large constitutes a third.
There's no question in my mind that
unsuspecting people are being exposed
to small quantities of fibres from such
sources as torn ironing-board covers
and pot holders, and who knows how
much asbestos is inhaled by hobbyists
engaged in do-it-yourself projects
around the house—like sawing up wall-
board, mixing crack-filler compounds,
insulating pipes with asbestos materials,
and repairing furnaces and boilers with
asbestos cements. I agree with those
who suggest that small numbers of fi-
bres may be liberated from many prod-
ucts in widespread use, such as asbestos-
cement pipe and asbestos filters, which
are employed in the food and pharma-
ceutical industries, but we have no in-
formation that this carries a disease
potential. What troubles me is that
relatively few people are aware of the
fact that it can be dangerous to inhale
asbestos. Not long ago, we discovered
that children in a number of kinder-
gartens in New York, California, and
other states were being allowed to mix
asbestos with water to make a
certain form of play dough. We have
also learned that the powders in some
children's cosmetics kits and in certain



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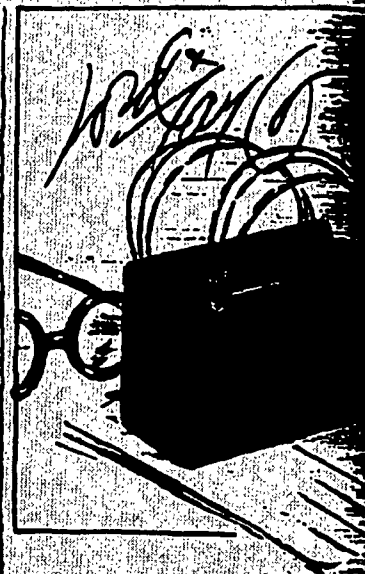
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powder-water-mix jewelry kits contain asbestos. In addition, within the past couple of years two different thoracic surgeons have published articles in leading medical journals which advocate the sprinkling of asbestos on surgical incisions in order to promote the adhesion and healing of tissue. This is very bad advice. Why, Dr. William E. Smith, who conducts animal studies at Fairleigh Dickinson University, has readily induced mesotheliomas in hamsters by injections of asbestos fibres into their pleural cavities!"

Dr. Selikoff went on to say that he understands full well that asbestos is an extremely valuable material, and that it has become essential in modern industrial society. "We epidemiologists recognize and study its dangers so that we may devise ways of minimizing or avoiding them," he said. "Improvements in the practice of industrial hygiene are needed to eliminate unnecessary exposure among asbestos workers and men in other building trades, and awareness of the potential risks of environmental contamination is needed until our studies indicate just how far out into the general population the problem extends. All I can say is that the public should be made aware of the problem, and that no one, particularly children and young people, should be unduly exposed. After all, today's children are almost certainly inhaling more asbestos than their parents did, and we now know that if a person inhales significant amounts of asbestos dust, he carries a burden that will provide a latent potential for the development of cancer for the rest of his life. Here's an example I'll never forget. A couple of years ago, just after Guyler Hammond, Jack Churg, and I published an article about asbestos and mesothelioma in the *New England Journal of Medicine*, I received a phone call from a doctor in Massachusetts who said that when he read the piece he immediately told his forty-three-year-old wife about it, because her sister had died of mesothelioma the year before. 'Imagine my horror,' this physician went on, 'when my wife proceeded to tell me that in 1943, while she and her sister were students in college, they had responded to a patriotic appeal and spent six weeks in a shipyard insulating pipes in destroyer escorts.'" Dr. Selikoff paused. "Two sisters with six weeks of exposure in 1943," he murmured. "Twenty-three years later, one of them dies of mesothelioma. And today, the other one—the doctor's wife—is being examined every few months to keep track of



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some suspicious-looking shadows on her X-rays."

AS I was arriving at the Environmental Sciences Laboratory one day, I happened to notice a newspaper clipping tacked up on a bulletin board near the entrance. The story, which was from the *Times* of September 2, 1967, started out as follows: "The scene looked wintry on Madison Avenue, between 57th and 58th Streets, yesterday. Flurries of what seemed to be snowflakes fell on the area from between the 25th and 30th floors of the General Motors Building, which is nearing completion on the site of the former Savoy Plaza Hotel." The article went on to say that residents of the neighborhood had complained to the contractor, and that one of the project engineers had explained that the white flakes were fallout from breproofing material that was being sprayed on the structural steel girders, beams, and decks of the fifty-story skyscraper. According to the *Times*, the engineer would not identify the insulating material, which was being applied with spray guns, "because I don't want to read newspaper stories about how we are messing up city streets." He was also quoted as saying, "This is a job condition all over the city." The last paragraph of the article read, "The city's Buildings Department said that it had not received any complaints about the Madison Avenue 'snowfall.' The department said the use of tarpaulins was required for spraying operations in construction work to prevent inconvenience to neighborhoods." Beneath the article was this typed notation: "Examined in our laboratory, the material was found to contain amorphous glass fibres (presumably rock wool), and chrysotile asbestos. It is known that other sprayed insulating materials contain amosite asbestos, rock wool, while still others contain asbestos fibres alone."

When I asked Dr. Selikoff about the bulletin-board items, he said that the biologist in the laboratory, Dr. Arthur M. Langer, had collected a sample of the material and identified its composition by means of electron diffraction and electron microscopy, and with the aid of the electron microprobe. He went to a shelf and took a small glass jar that contained a wad of fibrous-looking stuff, the size of a half dollar. "This is sample Dr. Langer brought back analyzed," he told me. "It comes in a type of insulation material that is twenty-five per cent asbestos."



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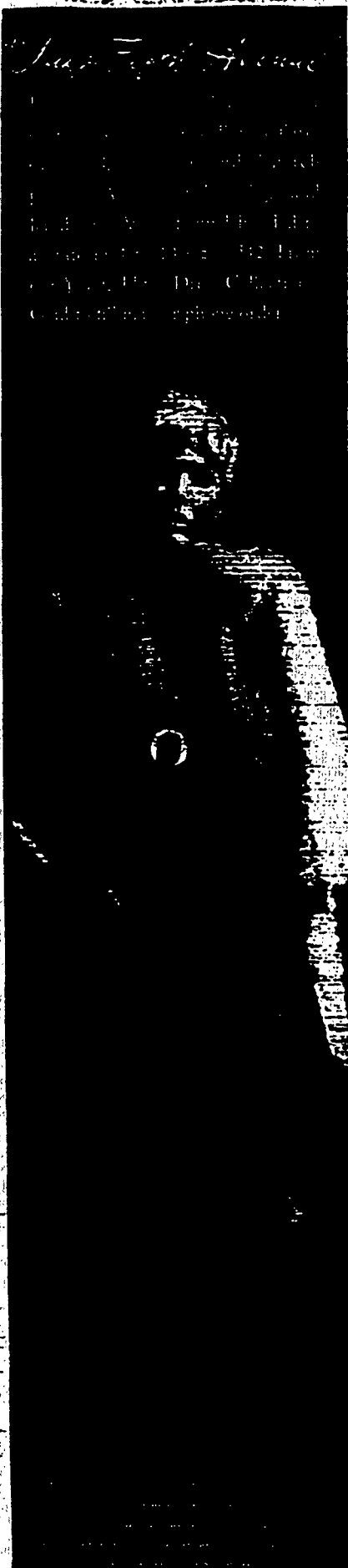
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and in this tiny wad alone there are literally billions of fibres. Of course, the 'snowfall' those people saw and complained about was not what may have been hazardous. What may have been hazardous was what they couldn't see—an untold number of sub-microscopic and highly respirable particles. The 'snowfall' was a perfect example of how asbestos gets into the ambient air, and that engineer was right when he said it's a job condition all over the city. You'll see the same material floating down from many other building projects in town. It's sprayed on by men in the Plasterers and Cement Masons Union. Incidentally, as far as I know, no one has ever made a study of their health."

It was a blustery day, filled with intimations of the season's changing, and when I left the hospital I took a Fifth Avenue bus downtown. I sat by a window, and was looking idly out at people strolling along the sidewalks when the bus came to a stop in the upper Forties beside a dump truck parked in front of a small office building that was being gutted. Several workmen were carrying canvas sacks of refuse across the sidewalk and heaving them up to a tall, well-built Negro who stood in the rear of the truck, emptying the sacks at his feet. As he did, clouds of dust rose into the air around him. Some of it showered the window beside me. It appeared to be ordinary cement dust, but then I saw that the pile of debris on which the man was standing contained broken pieces of acoustical panelling and wallboard, both of which often contain asbestos, and lengths of pipe coated with crumbling white insulation material that looked very much like asbestos.

When I got off the bus, I walked west along Forty-fourth Street. Halfway down the block, I saw two workmen slide a large rectangular container from a pickup truck and carry it into a building. The container was full of white powder, some of which was being blown away by the wind, and the men's clothes were covered with white dust. When I looked into the truck, I saw half a dozen large paper sacks labelled "Fireproof Plaster." One of the workmen returned for his tools, and I asked him if the plaster contained asbestos. "Sure," he replied. "That's what makes it fireproof."

I walked on to the corner of Sixth Avenue, where a forty-four-story office building had been under construction for several months. During that period, I had passed the building nearly every day, but now, for the first time, I



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ed up and scrutinized it carefully. Out halfway up its side, I saw some tarpaulins billowing in the wind. Above the tarpaulins, the steel girders and beams were reddish brown; between the tarpaulins and the ground, however, I had been sprayed with asbestos insulation, which clung to them like gray stuff. Then, all at once, I saw wads of the same gray stuff falling out of the tarpaulins. Some of the pieces were carried away on the wind; others that were heavier came tumbling down around me. One fell on the sidewalk at my feet. I picked it up, crumbled it between my fingers, and saw dozens of glass fibres float away. It was the same color and texture as the material that Dr. Selikoff had shown me half an hour before. The sidewalk was covered with it, and even as I stood there more of it came showering down. A woman hurrying to make the light with a friend brushed some off the sleeve of her coat, and said, "What the devil is that stuff?" I watched her cross the avenue, and saw asbestos insulation blowing over the pavement like thistle-down across a field. The wind was southerly, and most of the asbestos was travelling north, in the direction of Mount Sinai Hospital, where Dr. Selikoff and his associates are still following the trail Dr. Cooke set out on in 1924. But I was not thinking about Dr. Selikoff or the difficulties of epidemiology just then. I was thinking about the children who play in Central Park every day, and about all the other children who live in this city and breathe its air. —PAUL BRODEUR

THE CRIES WE DOUBT EVER GOT WE CRIED OUT: RAPTURE OR NO RAPTURE

[From the *St. Moritz Courier*]

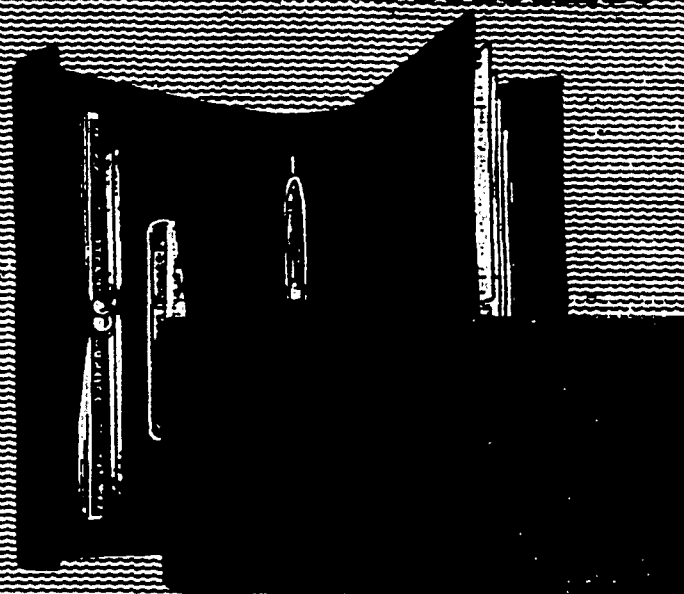
When GIOVANNI SEGANTINI first came to the Engadine from Lake Como, he was full of rapture:

"How before this earth which is hallowed by beauty; I kiss the grasses and flowers under this blue vault of heaven while the birds sing and meet to flight and the bees suck honey from the open blooms, I drink at these fountains where beauty is ever-renewed where love is renewed that gives life to all things."

Observers believed Brezhnev's visit would aid anti-American propaganda while leaving the Kremlin to deal with almost any future crisis, except possibly Richard Nixon. —*Washington Post*.

But till he sees the New Nivox.

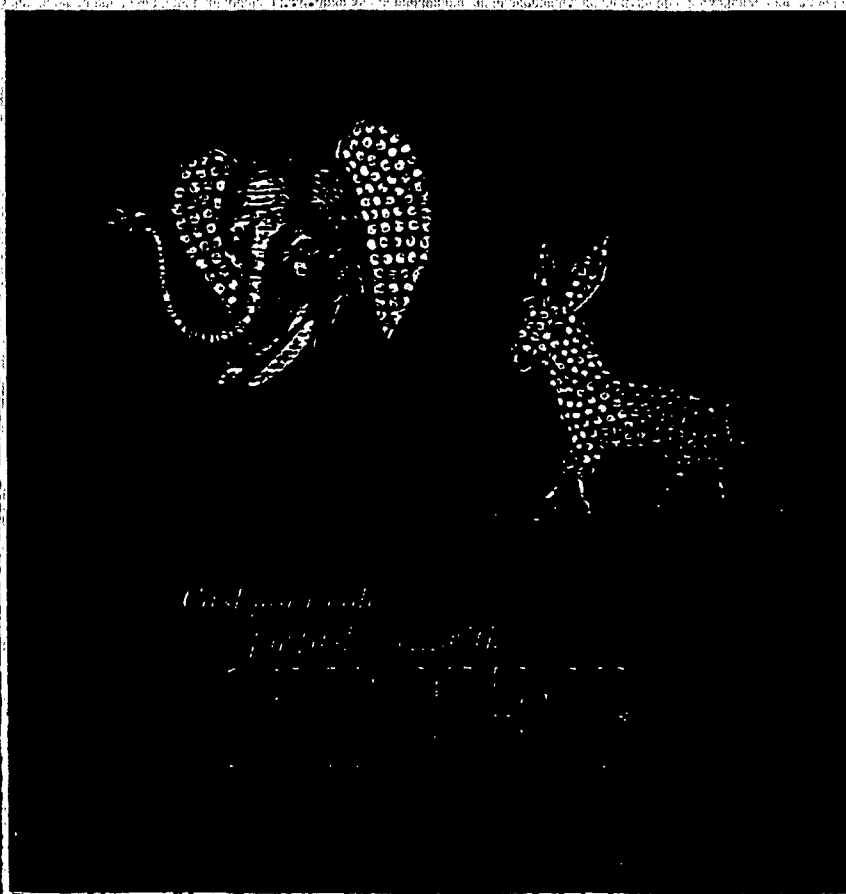
THE CARETAKER



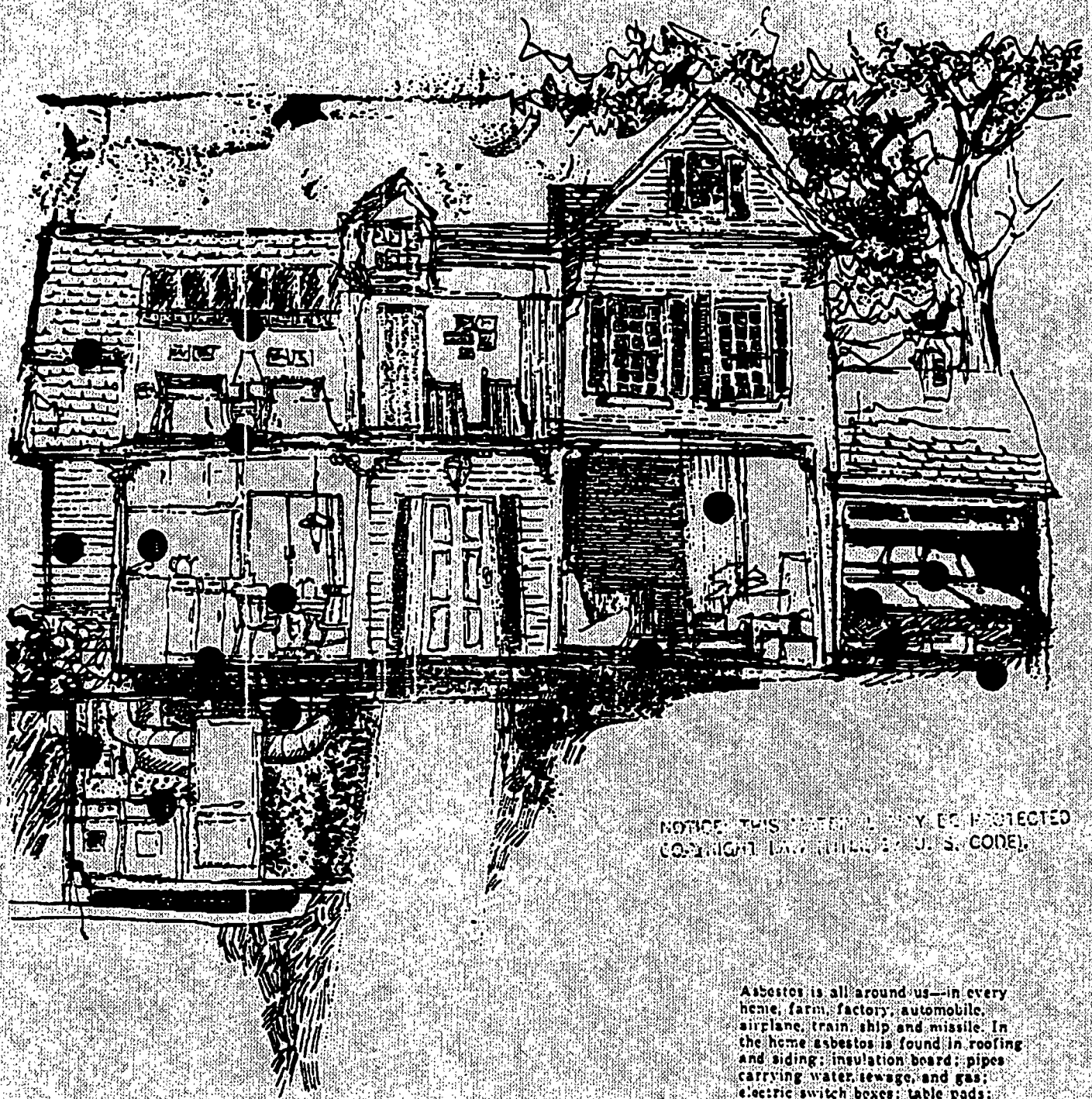
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Asbestos is all around us—in every home, farm, factory, automobile, airplane, train, ship and missile. In the home asbestos is found in roofing and siding; insulation board; pipes carrying water, sewage, and gas; electric switch boxes; table pads; ovens; vinyl and asphalt floor tiles; piano padding; frying pan handles; draperies; and rugs. Loose asbestos fibers from a torn ironing board cover, for instance, like the asbestos dust in a factory, may enter the human lung. Automobile sparkplugs, hoods, brake linings, clutch facings, and mufflers may be a source of the fine particles which could pose a hazard.

Illustration by Walter H. Miller

ASBESTOS

By I. J. Selikoff

I DO NOT KNOW if it is entirely appropriate to consider our current problems with asbestos an "unanticipated environmental hazard." As I look back, it would seem that we should have had some premonition.

For example, 70 years ago H. Montague Murray, then Senior Physician at the Charing Cross Hospital in London, examined a man of 33 suffering with pulmonary fibrosis. He had been working in the carding room of an asbestos textile factory. Of the ten men working in that room thirteen years before, he was the sole survivor. He, too, died a year later; at post mortem it was found that he had severely scarred lung, containing numerous asbestos fibers. Dr. Murray described this case to the Departmental Committee on Compensation for Industrial Disease which, after thanking Dr. Murray, noted that "... considerable trouble is now [in 1907] taken to prevent the inhalation of the dust, so that the disease is not so likely to occur as heretofore."

The disease which was referred to then had no name, nor did it when Auribault described numerous deaths among French workers in an asbestos textile factory in 1906, nor did it when Pancoast found abnormal X-rays of the chest in 1919 in a group of American asbestos workers. Surely, taxonomy has a particular virtue in science; individuals dying of this non-disease became much more evident when Cooke described another case in 1924 and 1927 and gave the illness its current name—*asbestosis*. We can now accurately record, for example, that ten percent of the deaths among asbestos insulation workers in New York in the past 25 years were due to asbestosis.

The new category was soon replete with cases.

However, comfort was provided, this time in the Home Office Report put before the English Parliament in 1930. While pulmonary disease among asbestos workers in the past was acknowledged, it was simultaneously concluded that the outlook was good. "In the space of a decade, or thereabouts, the effect of energetic application of preventive measures should be apparent in a great reduction in the incidence of fibrosis."

Such optimism persisted for the next 30 years; asbestosis was acknowledged as a rather rare disease, occasionally occurring among a small group of workmen employed in an isolated, esoteric industry.

There were a few rumblings during this time whose echoes should perhaps embarrass us now. Thus, Kenneth Lynch reported a death from lung cancer in association with asbestosis in 1935, and several similar reports of this association between two then uncommon diseases followed. For the most part, however, these reports were categorized as bibliographical curiosities. Even the careful studies of Richard Doll reported in 1955 failed to disturb our calm, perhaps because of the reticence with which the statistician summarized his findings. Listing eighteen deaths due to lung cancer in 106 consecutive post mortem examinations at one asbestos works in England, he concluded that "... lung cancer was a specific industrial hazard of certain asbestos workers ...". Even those who accepted Doll's conclusions—and there were some who did not—could point to a problem with a rather narrow spectrum. A single neoplasm (lung cancer) was (past tense) a specific industrial hazard (no hint that it could be more than this) of certain asbestos workers (the heavily exposed individuals in this factory).

In the past several years, the spectrum has widened considerably. First, it has been recognized that the hazard associated with direct occupational exposure to asbestos is by no means a narrow or infrequent one. The experiences of insulation workers in this country are instructive. These men, employed in the construction industry and often working out-of-doors and with materials containing only limited amounts of asbestos, are much less exposed than many factory workers had been. Yet their health experiences have been disastrous.

We have studied all members of the Insulation Workers Union in the New York metropolitan area. On December 31, 1942 there were 632 members of this Union. Calculations in studies by E. Cuyler Hammond, J. Churg and myself indicated that by December 31, 1962, according to life expectancy in the general population of U.S. white males, there should have been 203 deaths among these men. Instead, there were 255—an excess of twenty percent. Using age, year and sex-specific rates, there should have been six or seven deaths due to cancer of the lung or pleura. Instead, there were 45. There should have been nine or ten deaths due to cancer of the stomach or colon; there were 29. There should, of course, have been no deaths due to asbestosis; there were twelve.

Similar calculations to mid-1967 gave similar results. Whereas 47 deaths were expected, there were 94. Where eight or nine deaths due to cancer were predicted, there were 49. Again, there was a significant excess of specific types of cancer. While there should have been two or three deaths due to cancer of the lung or pleura, there were 27, and there were eight deaths due to gastrointestinal cancer against two expected. Once more, asbestosis claimed fifteen lives against none expected.

Studies are now under way in our laboratory of insulation workers in other parts of the country. Mortality data indicate that the New York findings are unfortunately true elsewhere, as well. It is not known exactly how many asbestos insulation workers are employed in the United States; estimates range from 50,000 to 100,000. In the face of the mortality information, one hopes the lower figure is more accurate.

Information is incomplete concerning the number of workmen in other trades in the United States

with direct occupational asbestos exposure. Totals of 100,000 to 125,000 are often proposed. Again, the lower figure is hoped for, especially in view of the absence of satisfactory mortality information concerning such asbestos factory workers, and since Mancuso's findings are disquieting.

Analysis of these mortality experiences is disturbing from another point of view, in addition to its magnitude. I refer to the occurrence of pleural

TABLE I
Observed and Expected Number of Deaths
among 632 Asbestos Workers Exposed to
Asbestos Dust Twenty Years or Longer, 1943-1962

Cause of Death	1943- 1947	1948- 1952	1953- 1957	1958- 1962	Total 1943- 1962
Total cancer, all sites					
Observed	13	17	26	39	95
Expected	5.7	8.1	13.0	9.7	36.5
Cancer of lung and pleura					
Observed	6	8	13	18	45
Expected	0.8	1.4	2.0	2.4	6.6
Cancer of stomach, colon and rectum					
Observed	4	4	7	14	29
Expected	2.0	2.5	2.6	2.3	9.4
Cancer of all other sites combined					
Observed	3	5	6	7	21
Expected	2.5	4.2	8.4	5.0	20.5
Asbestosis					
Observed	0	1	4	7	12
Expected					less than 1
Total, all causes					
Observed	28	54	85	88	255
Expected	39.7	50.8	58.6	54.4	203.5

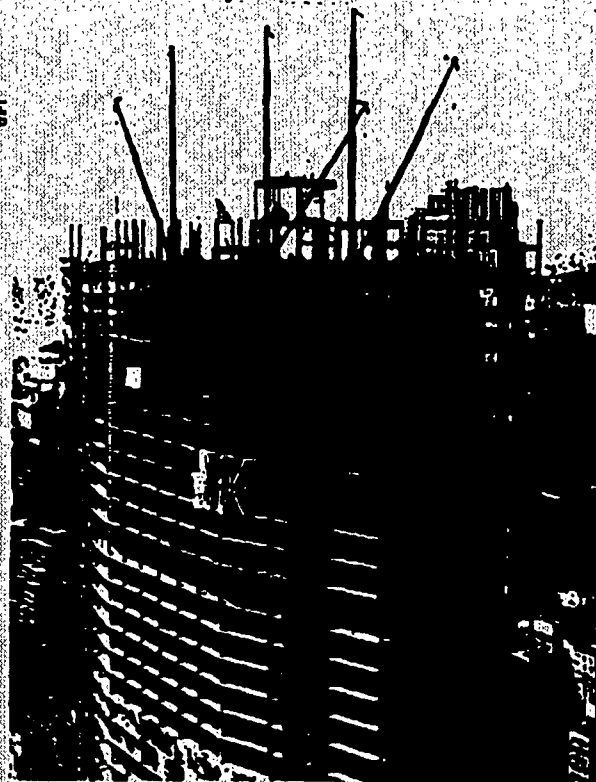
and peritoneal mesothelioma (tumors of the lining of the lung and abdominal cavities) among these workmen with direct occupational exposure. During the 1950's, there were several reports of deaths in asbestos workers caused by these diffuse tumors of the mesothelial surfaces. Such isolated cases would have received little notice had it not been for the fact that the tumor has always been considered extraordinarily rare. It is no longer rare among asbestos workers. Indeed, it is so common a cause of deaths among them now—while still rare among individuals not known to be exposed to asbestos—as to almost constitute a tumor specific to asbestos exposure. Several reviews of autopsy files suggest that this rapid increase, with hundreds of cases now known in Great Britain, South Africa, the United States and Canada, is not due to refinement in diagnosis. Thus, review of all possible cases in several major pathology departments in Scotland has demonstrated an increase from one or two cases a year in the 1950's to some twenty cases a year or more at the present time. Fourteen of the last 120 deaths in New York among asbestos insulation

A pioneer in modern asbestos epidemiology, Dr. Irving J. Selikoff is Professor of Environmental Medicine at New York City University's Mount Sinai School of Medicine, Director of its Environmental Sciences Laboratory, and is also President of the New York Academy of Sciences. He was awarded the American Public Health Association's Albert Lasker Award in Medicine for his research on isoniazid, an antitubercular drug widely used in the treatment of tuberculosis.

workers have been due to mesothelioma. The problem has assumed sufficient importance to have warranted the establishment of Mesothelioma Reference Panels in a number of countries, by the International Union Against Cancer, and the recent establishment of a Mesothelioma Registry in this country by the National Institute of Environmental Health Sciences of the U.S. Public Health Service.

These experiences highlight one portion of the current spectrum of asbestos disease. It is now recognized that *direct occupational exposure* to asbestos results in a hazard much more significant than formerly appreciated. In the last several years, however, additional problems have been recognized which add another dimension: the possibility that the utilization of asbestos is associated with a much wider risk, perhaps one of the community at large. This is less well defined, much less well documented, but because of its potential importance, of considerable concern.

The recognition of this potentially wider spectrum of asbestos disease has been based upon three germinal observations. The first was the publication in 1960 by Wagner and his colleagues of 47 cases of mesothelioma in South Africa, all from the northwest Cape Province, an asbestos-rich area of the country. Analysis of the cases showed that few had worked directly with the mineral but that 45 of the 47 may have had contact with it, often indirect. They had lived in an area in which asbestos was mined, or had worked in an environment in which indirect, haphazard exposure was conceivable. In striking contrast, there were no cases of mesothelioma in the rest of South Africa reported in the



Insulation workers employed in the construction industry have had disastrous health experiences even though their work is often outdoors and involves only limited contact with asbestos. Up until the past several years, it was thought that lung cancer from asbestos was an industrial hazard limited to the heavily exposed individuals in asbestos factories.

TABLE 2

Observed and Expected Number of Deaths among
370 Asbestos Workers from January 1, 1963
to April 30, 1967

Cause of Death	Observed Deaths	Expected Deaths*
Total cancer (all sites)	49	8.6
Cancer of lung, pleura, bronchus, and trachea	27	2.3
Bronchogenic carcinoma	24	..
Pleural mesothelioma	3	...
Peritoneal mesothelioma	7	...
Cancer of stomach	3	0.6
Cancer of colon and rectum	5	1.2
Cancer of all other sites combined	7	4.5
Asbestosis	15	...
Heart and circulatory disease	...	...
Including stroke	22	28.5
All other causes of death	8	10.4
Total, all causes	84	47.5

*Based upon U.S. mortality data disregarding smoking habits.

**United States data not available, but figure should be only slightly less than 2.3.

***United States data not available, but these are rare causes of death in general population.

same period, and none in 10,000 consecutive autopsies of individuals with silicosis. This report unequivocally raised the question of whether asbestos-associated mesothelioma was limited to direct occupational exposure, or whether much less intimate contact could produce the same result.

In the same year, 1960, Kiviluoto raised an analogous question. Pleural calcification had been known to occur frequently in characteristic form among asbestos workers. In our experience, indeed, the majority of older asbestos workers may be expected to show these changes when X-rayed. It was, therefore, striking to find typical "asbestotic" pleural calcification in 499 of 6,812 individuals X-rayed in one county in Finland while the neighboring county had none in 7,101. Kiviluoto astutely noted that the first county contained an asbestos mine and while none of the individuals with pleural calcification had worked in this mine, they had lived nearby. His countrymen, Laamanen and his colleagues, later demonstrated the presence of asbestos fibers in the air in the vicinity of the mine and while this may not reflect the true explanation for Kiviluoto's findings, the question of environmental contamination was strongly suggested.

The third observation was equally sobering. It had long been known that asbestos fibers, when

Inhaled into the human lung, may become coated with an iron-rich characteristic golden brown material, forming an "asbestos body." These are found, for example, in very large numbers in the lungs of asbestos workers. In 1963, Thomson reported that he found bodies with the same appearance in the lungs of people who were not asbestos workers. Smears of the lung bases allowed him to demonstrate these bodies in 26 percent of 600 consecutive autopsies in Capetown. His conclusion was that asbestos had indeed become a modern urban hazard, and he predicted that asbestos-induced mesothelioma might become as frequent as cigarette-associated lung cancer is at the present time.

Thomson's observations have been widely confirmed, and particles having the appearance of asbestos bodies have been reported in similar percentages in Montreal, Milan, London, Newcastle, Glasgow, Belfast, Dresden, Pittsburgh and Miami. In New York City, using a somewhat more sensitive technique, we find that approximately half of the lungs in 1,975 consecutive autopsies examined by us show these particles. When one considers that we examine only one square centimeter of lung tissue 0.0175 centimeter in thickness, it is obvious that these particles are now very common among city dwellers at this time.

Opportunity for non-occupational contact with asbestos at the present time can easily be discovered. There are, for example, some three thousand products which currently utilize this valuable material. To the present, lack of recognition of the potential of non-occupational exposure has led to the absence of appropriate safeguards during industrial use. Demolition of buildings, often rich in asbestos-containing materials, is suspect, while sheer carelessness and ignorance may explain its

TABLE 3
Asbestos Bodies in New York City
Dwellers Without Occupational Exposure—1,975 Cases

Male				Female			
Age	Total cases	Total with asbestos bodies	Percent with asbestos bodies	Age	Total cases	Total with asbestos bodies	Percent with asbestos bodies
0-1	40	0	—	0-1	31	0	—
2-9	2	1	—	2-9	2	0	—
10-19	4	0	—	10-19	8	3	38%
20-29	22	4	18%	20-29	12	4	33%
30-39	50	14	28%	30-39	16	7	39%
40-49	178	83	47%	40-49	58	18	31%
50-59	290	149	51%	50-59	85	38	45%
60-69	359	205	57%	60-69	147	57	39%
70-79	321	180	56%	70-79	151	65	43%
80-89	105	62	59%	80-89	80	40	50%
90+	7	4	57%	90+	12	8	66%
Total	1368	702	51%	Total	607	240	39%

TABLE 4
Asbestos Bodies in Shipyard
and Construction Workers: Quantitative Results

	Blue Collar (no Shipyard or Construction)	Shipyard and Construction	White Collar Males
Total Number of Workers	246	129	206
Workers Having Asbestos Bodies	121 (49%)	90 (70%)	87 (42%)
Workers Having No Asbestos Bodies	125	39	119
Workers Having Five or More Asbestos Bodies	20 (8%)	29 (23%)	10 (5%)

recommendation and use as playroom material in kindergartens or in toy cosmetics.

Indirect occupational exposure is suggested by recent observations in our laboratory. We find, for example, that while approximately 30 percent of housewives examined at post mortem had asbestos bodies demonstrated, comparable figures are 40 percent for white collar males and 60 percent for blue collar males. This rises to over 70 percent for men with any history whatsoever of having worked either in the construction industry or in shipyards. The latter, moreover, are much more likely to have numerous asbestos bodies than any of the other groups.

A special hazard may be present as the result of an interaction recently described. We have found that there is a special risk of cigarette smoking among asbestos workers. Since January 1, 1963 we have observed 87 experienced insulation workers who were not regular smokers or who were pipe or cigar smokers; we have yet to see a death from bronchogenic carcinoma among these men. On the other hand, of 283 equally experienced workmen who smoked cigarettes and who were watched for the same period of time, we have seen 24 such cancers, although only three were expected. We have calculated that the risk of dying of lung cancer is approximately 92 times greater for an asbestos worker who smokes cigarettes than a man of the same age who neither works with asbestos nor smokes cigarettes.

These observations naturally raise the question whether the inhalation of asbestos fibers among individuals in the general population might carry a special risk among the cigarette smokers in that population.

Wagner's observations have been extended by two recent studies. Muriel L. Newhouse investigated 76 consecutive cases of mesothelioma examined at the London Hospital, the majority within the last several years. She found, to no one's surprise, that

31 had been asbestos workers. Much more unexpected, however, was her discovery that nine had merely lived in the household of an asbestos worker, raising the serious question of a special hazard of family exposure. Then, of the 36 who neither worked with asbestos nor had relatives who worked with asbestos, eleven had lived within a half mile of an asbestos plant. The latter cases, of course, suggested a special risk associated with neighborhood contact with asbestos.

Similar findings were reported by Jan Lieben last year in Pennsylvania. Of 42 cases of mesothelioma reviewed by the Pennsylvania State Department of Health, twenty had identifiable occupational exposure. In addition, eight individuals had lived within the vicinity of an asbestos plant and three had had family contact.

We have confirmed that family contact is a distinct possibility. Sampling of both ambient air and settled dust in the homes of New York insulation workers has shown characteristic asbestos particles to be present, presumably brought home on the clothes of the workmen.

It is obvious that the spectrum of asbestos disease has considerably widened in the last several years. In addition to lung cancer and asbestosis, a number of other tumors are now recognized as constituting a hazard. Rather than a small number of heavily exposed workmen, a large number are involved in less intense direct occupational exposure or even in indirect occupational exposure. The latter may represent a real problem: there are, for example, 3,500,000 men employed in the construction and shipbuilding industries, or five percent of the total employed population in the United States. Significant exposure may even extend to the families of these men and perhaps to residents in the neighborhoods surrounding their places of employment. Finally, this ubiquitous material may well be found to permeate the community at large. What such exposures may bring is not now known. Disease associated with asbestos is rarely seen sooner than 20 to 30 years from the onset of exposure. Thus, among our insulation workers in New York, I have yet to see a mesothelioma in a man who began work after 1930 or a case of lung cancer in an asbestos worker who had worked in that industry less than twenty years. Our concern is with the future.

At the outset of this presentation, I questioned whether asbestos might properly be included as an *unanticipated* environmental hazard. Yet the less iconoclastic among us might have countered with: it must have been—we could not have knowingly allowed this to come to pass! Surely the explanation lies in the length of the period that elapses between exposure and disease which kept us from really knowing the problem until now.

Perhaps so. Indeed, I do not share the demonological theory of industrial hygiene history, in which industry is seen as callously exchanging human disease for profits. Rather, I would say industry shared the ignorance and lethargy of us all.

The answer will come in these next several years. We have some reason for optimism. First, we have the law of averages on our side—sooner or later, *some* important environmental hazard is bound to be controlled. I suspect asbestos will be one. We have reason to believe that the dose-disease relationship is on our side—to the present, disease has been uncommon except with significant exposure. And our data suggest further that such significant exposure can be prevented, that we can learn to live and work with asbestos. All it will take is the doing—but herein lies another tale. □

NOTES

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November 2, 1970

Mrs. M. Manning, Executive Director
Girl Scouts of Metropolitan Detroit
153 E. Elizabeth
Detroit, Michigan

Dear Mrs. Manning:

This is to call your attention to a potentially hazardous activity suggested in the Girl Scout Manual entitled: "Moreabouts for Brownie Girl Scout Leaders." On page 43 of this manual is a suggestion for making candlestick holders from asbestos dough and on page 64 is a recipe for the asbestos modeling material.

There is extensive literature in medical and environmental health journals which indicate a relationship between exposure to asbestos dust and serious lung diseases. I am attaching copies of two articles that you may find interesting which survey some of these findings: (1) Paul Broder, "A Reporter at Large — The Magic Mineral," The New Yorker, October 17, 1968; and (2) I. J. Selikoff, "Asbestos," Environmental Health Perspectives, March 1969. Incidentally, I have brought these passages to the attention of Prof. Selikoff's attention. Prof. Selikoff, who is Director of the Environmental Sciences Laboratory of the Mount Sinai School of Medicine of the City University of New York, is a recognized worker in the field of asbestos disease epidemiology.

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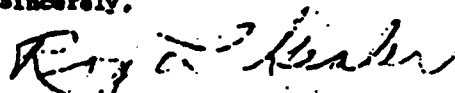
Mrs. M. Manning

-2-

November 2, 1970

In view of the possible danger of exposure to asbestos dust, you may wish to recommend that reference to asbestos modeling material be deleted from future printings of this (or any other) manual, and that cautionary information be disseminated to appropriate Girl Scout leadership.

Sincerely,



Roy L. Gealer, Ph.D.
Chemical Engineering Department
Scientific Research Staff

RLG:red

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