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Asbestos Exposure and Neoplasia

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Building trades *insulation* workers have relatively light, intermittent, exposure to asbestos. Of 632 insulation workers, who entered the trade before 1943 and were traced through 1962, forty-five died of cancer of the lung or pleura, whereas only 6.6 such deaths were expected. Three of the pleural tumors were mesotheliomas; there was also one *peritoneal* mesothelioma. Four mesotheliomas in a total of 255 deaths is an exceedingly high incidence for such a rare tumor. In addition, an unexpectedly large number of men died of cancer of the stomach, colon, or rectum (29 compared with 9.4 expected). Other cancers were not increased: 20.5 were expected, 21 occurred. Twelve men died of asbestosis.

ALTHOUGH PULMONARY CARCINOMA had been observed in the earliest studies of asbestosis, association between the two conditions was first suggested by Lynch and Smith in 1935.[1] Additional reports of such association followed. Perhaps the most striking data was presented in the annual report of the Chief Inspector of Factories of Great Britain for 1955.[2] Every death with asbestosis in the files of the Factory Department, from the first recognition of asbestosis as a disease entity, was studied. Altogether 365 such deaths were recorded (1924-1955). Sixty-five or 17.8% were found to be accompanied by cancer of the lung or pleura. Doll,[3] after reviewing the problem and adding data of his own, concluded that lung cancer was a specific industrial hazard of heavily exposed asbestos workers.

Nevertheless, some investigators have held that, while these observations might be suggestive, they did not establish an increased incidence of carcinoma of the lung in pulmonary asbestosis, and further, that the association was unproved.

The factor of selection was considered a potential weakness in evaluating reports of autopsy series. It was noted that complicated and unusual cases would be more likely to come to autopsy, thus raising the apparent frequency of associated lung neoplasms.[4] Further, it was argued that autopsy statistics, which dealt with particular groups of those who died, do not reflect total populations of asbestos workers.[5] Additional reservations were based on the frequent absence of data regarding exposure, smoking habits, and personal history, on the size of series, and on inadequate histological verification in some cases.

Within the last few years a number of additional problems connected with asbestos exposure have appeared, making clarification and resolution of the foregoing statistical uncertainty a matter of considerable concern. First, there has been greatly increased use of the various types of asbestos (a five-fold increase in world utilization of this group of minerals, from 500,000 tons to 2,500,000 tons per year in the last 30 years), as well as a greatly increased number and variety of industrial applications of asbestos (over 3,000 such uses now recorded). Second, suspicion has been growing that malignancy associated with asbestos exposure may include neoplasms other than carcinoma of the lung. Thus, a significant relationship has been claimed between diffuse mesothelioma of the pleura and peritoneum and asbestos exposure.[6]

This communication is concerned with investigations undertaken to study the following facts: (1) the incidence of deaths due to pulmonary carcinoma among a group of workers exposed to asbestos under United States industrial conditions in the past several decades, (2) whether or not such individuals would also be found to have increased risk of other neoplasms, and (3) whether such risks would be present in an industry other than the asbestos-producing or asbestos-products industries, with which most reports in the past have been concerned but which would not necessarily represent the most important risks of asbestos exposure at this time. Further, it was hoped that study of an industry with asbestos exposure of limited extent and intensity would throw some light on the potential problems associated with minimal exposure to asbestos.

Materials and Methods

Our investigations have been concerned with 1,522 members of the Asbestos Workers Union in the New York metropolitan area, members of New York Local 12 and Newark, NJ, Local 32 of the International Association of Heat and Frost Insulators and Asbestos Workers. Under this title of couples, these men are insulation workers. Although the union is considered one of the building trades unions, its members do insulation work in a variety of industries, including shipbuilding. Called "laggers" in Great Britain, they are often designated "pipe coverers," "insulators," or "asbestos workers" in this country.

The union is one of the oldest in the country.

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The New York local, as the "Salamander Association of Boiler and Pipe Felters," was the first union of insulation workers in the United States. It amalgamated with other locals as the current Asbestos Workers Union in 1912. The stability of this union has been reflected in the stability of its membership rolls. "Once a pipecoverer, always a pipecoverer" has been of epidemiological importance to us and has made this group of men particularly suitable for the study of long-term effects of asbestos inhalation. Unlike unskilled workers exposed to asbestos inhalation in poorly paid industries, there is little labor turnover among insulation workers. Accurate employment records are maintained by the union, which has also been concerned with health problems in the industry.

The trade was badly hit during the depression; some men had to drop out and very few were added during the 1930's. By the end of 1942 the union rolls consisted mainly of men with considerable experience, plus a few who joined in 1940, 1941, and 1942. Between 1946 and 1962 union membership increased substantially.

Source of Data.—From union records, a list was prepared of every individual who was a member of either of the metropolitan locals on Dec 31, 1942, or who joined between that date and Dec 31, 1962. No one was omitted, whatever his subsequent work history. The 1942 list included 632 men. 890 men joined after 1942.

Personal data were obtained from union records, and the work history of each man was detailed, including withdrawal from employment (war service, other employment, retirement, illness). These data gave the baseline for calculation of the onset and duration of exposure. For members who had died, records of the Health and Welfare Fund provided date and place of death. Copies of death certificates were obtained on all but one of them. Autopsy protocols, histological specimens, and hospital records were obtained and reviewed in those deaths approximately one half, in which the terminal illness had occurred in a hospital.

Statistical Analysis.—Previous studies have suggested that neoplasia associated with asbestosis seldom occurs until 20 years after first exposure to asbestos dust. Therefore, we decided to limit the present analysis to men with such an exposure history. Our complete records cover all members of the union (including active and retired members, both dead and alive) during the 20-year period from Jan 1, 1943, through Dec 31, 1962. However, with few exceptions, the only men with a history of 20 years or longer since first exposure to asbestos were the 632 men on the union rolls as of Jan 1, 1943. (The exceptions were a few men who joined the union after Jan 1, 1943, but who had been employed previously as asbestos workers elsewhere.) Of these 632 men, 255 died before Jan 1, 1963.

Table 1.—*Man-Years of Experience of 632 Asbestos Workers Exposed to Asbestos Dust 20 Years or Longer*

Age	Years 1943-1947	1948-1952	1953-1957	1958-1962
35-39	95.0	185.0	7.0	11.0
40-44	230.5	486.5	291.5	70.0
45-49	339.5	324.0	530.0	314.5
50-54	391.5	264.0	308.0	592.5
55-59	387.0	390.0	316.0	268.5
60-64	221.0	341.5	344.0	255.0
65-69	139.0	181.0	266.0	280.0
70-74	83.0	115.5	137.0	197.5
75-79	31.5	70.0	70.5	79.0
80-84	5.5	18.5	38.5	73.5
85+	3.5	2.0	6.0	13.5
Totals	1,912.0	2,478.0	2,336.5	2,011.0

Of these 632 men, 339 had been exposed to asbestos dust prior to 1924. In other words, as of Jan 1, 1943, 20 years or longer had elapsed since these 339 men were first exposed. The remaining 293 men reached the 20-years-since-first-exposure point at some time after Jan 1, 1943, and before the end of 1962. The 339 men who were first exposed prior to 1924 were counted in each of the 20 years (or up to the time of death of those who died). The 293 who were first exposed in 1924 or later were counted only after they reached the 20-years-since-first-exposure point (those who died being dropped at the time of death). When the statistics were completed, we found that we had records covering a total of 8,737.5 man-years of experience of men with a history of 20 years or longer since first exposure to asbestos dust.

Of the 8,737.5 man-years, 1912.0 were in the five-year period 1943-1947; 2,478.0 were in the period 1948-1952; 2,336.5 were in the period 1953-1957; and 2,011.0 were in the period 1958-1962. Table 1 shows the age distribution of the man-years in each of these five-year periods. Table 2 shows (1) the average age-specific death rates of all US white males during each of these periods, and (2) the average age-specific death rates from cancer of the lung, pleura, mediastinum, and trachea among US white males during each period, as reported by the US National Office of Vital Statistics.

The man-years were then multiplied by the corresponding reported US death rates to ascertain the *expected* number of deaths under the null hypothesis that the death rates of asbestos workers do not differ from death rates of all US white males (both age and date being taken into consideration). The results are summarized in Table 3.

Results

Total Deaths.—During the last five years (1943-1947) only 28 deaths occurred among the asbestos workers, whereas 39.7 deaths would have occurred had their age-specific death rates been the same as for all US white males during those years (Table 3). In other words, at the start of the study, the asbestos workers had below average death rates. This is by no means surprising. Indeed, such

Table 2.—Total Deaths and Deaths From Cancer of the Lung, Bronchus, Pleura, Mediastinum, and Trachea per 10,000 White Males per Year*

Age	1943-1947		1948-1952		1953-1957		1958-1962	
	Total	Lung Cancer†	Total	Lung Cancer†	Total	Lung Cancer†	Total	Lung Cancer†
35-39	30		30		26		25	1
40-44	55	1	48	1	43	1	42	1
45-49	86	2	78	3	72	3	70	3
50-54	133	4	124	5	115	6	116	7
55-59	196	5	189	8	178	10	178	11
60-64	295	7	281	10	276	14	272	17
65-69	435	7	417	11	416	17	418	21
70-74	634	6	605	11	586	16	607	21
75-79	977	6	915	10	876	14	896	17
80-84	1,453	5	1,353	8	1,346	12	1,363	14
85+	2,422	3	2,162	7	2,017	9	2,148	14

*Death rates as reported annually by the US National Office of Vital Statistics. A five-year average is given here for each period. Average for 1958-1962 is a projection of 1958-1960, since 1961-1962 rates are not yet available.

†Death rates include cancer of the lung, bronchus, pleura, mediastinum, and trachea, assigned to international list code No. 47[illegible] prior to 1949 and to No. 160-164 thereafter.

is almost always found in the first few years of a prospective epidemiological study of this type. The explanation is almost certainly as follows: The 632 men in this analysis were actively employed as asbestos workers in 1942. Since disability from illness or other causes precludes employment in a trade of this type, these men were presumably well (or at least not disabled) at the start of the study period. Almost any group so selected as to exclude the ill and disabled has a lower death rate during the ensuing few years than does the general population, since ill and disabled persons have extremely high death rates. A selective effect of this type gradually wears off with time and largely disappears within five to ten years from the time of initial selection.

During the second five-year period (1948-1952) the death rate of the asbestos workers was slightly higher than the death rate of all US white males, ie, 54 observed deaths compared with 50.8 expected deaths. In later periods, the death rate of the asbestos workers was proportionately higher. For the period 1953-1957, there were 85 observed deaths compared with 56.6 expected deaths, and for the period 1958-1962, there were 88 observed deaths compared with only 54.4 expected deaths.

Table 3.—Observed and Expected Number of Deaths Among 632 Asbestos Workers Exposed to Asbestos Dust 20 Years or Longer

Cause of Death	Years 1943-1947	1948-1952	1953-1957	1958-1962	Total, 1943-1962
Total, all causes	28	54	85	88	255
Observed (asbestos workers)					
Expected (US white males)	39.7	50.8	56.6	54.4	203.5
Total cancer, all sites	13	17	26	39	95
Observed (asbestos workers)					
Expected (US white males)	5.7	8.1	13.0	9.7	36.5
Cancer of lung and pleura	6	8	13	18	45
Observed (asbestos workers)					
Expected (US white males)	0.8	1.4	2.0	2.4	6.6
Cancer of stomach, colon, and rectum	4	4	7	14	29
Observed (asbestos workers)					
Expected (US white males)	2.0	2.5	7.6	2.3	9.4
Cancer of all other sites combined	3	5	6	7	21
Observed (asbestos workers)					
Expected (US white males)	2.9	4.2	5.4	5.0	20.5
Asbestosis	0	1	4	7	12
Observed (asbestos workers)					

Cancer of the Lung, Pleura, and Trachea. In each of the four five-year periods, far more deaths from cancer of the lung and pleura occurred among the asbestos workers than would have occurred had their death rates from these diseases been the same as for all US white males (Table 3). Altogether 45 of the 632 asbestos workers died of cancer of these sites, whereas only 6.6 such [illegible] would be expected from general US experience. Of these 45 deaths, 42 were recorded as due to bronchogenic carcinoma and 3 to neoplasms of the pleura. The pleural neoplasms were all recorded as mesotheliomas.

Thus it was found that the death rate from cancer of the bronchus and pleura was 6.8 times as high among these asbestos workers as in the general US white male population (both age and date being taken into consideration).

It may be asked whether the high rate of lung cancer among these asbestos workers could possibly be attributed to an unusually large proportion of cigarette smokers among them. We cannot answer this question directly, since we have not yet been able to ascertain the smoking habits of the men who died. However, the following pieces of evidence indicate that unusual smoking habits cannot account for the high death rate from lung cancer among these workers:

We have interviewed 320 of the 377 surviving members of the 1942 group. Table 4 gives a summary of the smoking habits in this group compared with a sample of men drawn from the general population of 1,121 counties in 25 states.[5] The union sample is somewhat inadequate since it does not include the men who died and does not include all of the present living members of the union. Nevertheless, it shows that a substantial proportion of asbestos workers never smoked cigarettes regularly. Certainly the 632 men in our analysis of death rates were not all heavy cigarette smokers.

In the general male population, lung-cancer death rates are about ten times as high among cigarette smokers as among nonsmokers; and the

Table 4.—Smoking Habits of 320 Abestos Workers Exposed to Ashestos Dust 20 Years or Longer Compared With Sample of Men From the General Population*

	Never Smoked Regularly		Smoked Pipe, Cigar, Never Cigarettes		History of Cigarette Smoking	
Age	Asbestos Workers, %	General Population, %	Asbestos Workers, %	General Population, %	Asbestos Workers, %	General Population, %
40-49	9.3	18.8	4.6	7.5	86.1	73.7
50-59	14.3	19.9	6.1	9.9	79.6	70.2
60-69	20.3	23.6	10.1	16.2	69.7	60.2
70+	25.5	37.1	11.8	23.9	62.7	39.0

*Sample of men from general population as reported by Hammond and Garfinkel.[7]

death rate from lung cancer increases greatly with the amount of cigarette smoking.[8] However, a large proportion of all men in the United States have a history of regular cigarette smoking. From data in a prospective study on smoking,[8] it may be estimated that if all men smoked a pack or more of cigarettes a day (ie, if all the nonsmokers, cigar smokers, pipe smokers, and light cigarette smokers had instead, been heavy cigarette smokers) the lung-cancer death rate would be approximately 3.4 times as high as it is at this time.

From this we may conclude that even if *all* our asbestos workers had smoked a pack or more of cigarettes a day (and, indeed, from our sample we know they did not), and if exposure to asbestos were of no significance, then their lung cancer [illegible]th rate would have been about 3.4 times as [illegible]h as the rate in the general US male population. Clearly, the smoking habits of the asbestos workers cannot account for the fact that their lung-cancer death rate was 6.8 times as high as that of white males in the general population.

Gastrointestinal Cancer.—Rather to our surprise, the death rate from cancer of the stomach and the death rate from cancer of the colon and rectum were higher among the asbestos workers than would be expected from the rates reported for the US white male population, calculated in the same way as for lung cancer. Twelve deaths from gastric cancer occurred among [illegible] compared with only 4.3 expected. Seventeen deaths from cancer of the colon and rectum occurred among the asbestos workers, as compared with 5.2 expected.

Cancer of All Other Sites.—The combined death rate from cancer of all sites other than lung and pleura, and stomach, colon, and rectum was not increased. Twenty-one such deaths occurred among asbestos workers, as compared with 20.5 expected.

Asbestosis.—Of the 255 deaths, 12 [illegible] due to asbestosis (pulmonary insufficienc[illegible] [illegible]

Comment

Carcinoma of the Lung.—[illegible] to carcinoma of the lung [illegible]

posure to asbestos by insulation workers, as studied here, results in a marked increase in the incidence of cancer of the lung, approximately six to seven times the expected incidence. Altogether, 45 (17.6%) of 255 men with more than 20 years elapsed since the onset of exposure died of cancer of the lung or pleura.

These data do not give the "incidence of cancer of the lung in asbestosis." They relate to the specific conditions of our investigation: to a group of men with only intermittent exposure to materials containing limited amounts (often 2% to 20%) of asbestos under working conditions varying from very dusty, as in extracting old insulation in closed quarters, to those with little dust exposure, as in building construction in open air. Moreover, they relate to the relatively recent past, in a trade with the shorter work week of the strong building trades unions, in an era when industry has been aware of potential asbestos hazard and the working population has had some consciousness of potential risk associated with dust exposure. These data would not necessarily apply to asbestos exposure in other industries, such as the factory production of asbestos products, the asbestos textile industry, etc, where conditions of employment might be quite different. Our results do not contradict the even higher incidence of lung cancer [illegible] other studies [illegible]

Diffuse Pleural and Peritoneal Malignant Mesothelioma.—Determining the incidence of diffuse pleural mesothelioma is complicated by the insecurity of its histological verification. [illegible] some pathologists will so categorize [illegible] tion of diffuse pleural tumors [illegible] others it is a very rare [illegible] iched by anaplastic [illegible] lung [illegible]

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growing number of reports of individual cases suggests that these tumors are perhaps becoming relatively frequent complications of asbestos exposure.

Our observations in this series are similarly suggestive. In three of the 255 deaths among the men who had worked for 20 years or more, the examining pathologist considered the death due to diffuse pleural mesothelioma, and in the two cases in which we have been able to review the histological material, the histological appearance was that often so categorized, and asbestos bodies were present. This incidence of more than 1% of deaths from pleural mesothelioma is strikingly high for a tumor which is generally considered to be extremely rare. In one case in our series pathological examination suggested diffuse peritoneal mesothelioma. This single experience is too fragmentary for evaluation.

Gastrointestinal Carcinoma.—Isolated instances of gastrointestinal carcinoma in the presence of asbestosis have been known, but there have been no data to indicate that these were more than coincidental findings. Among the asbestos workers studied here, cancer of the stomach, colon, and rectum was three times as frequent as expected. These data suggest that there may perhaps be an etiological relationship between industrial asbestos exposure and carcinoma of the gastrointestinal tract.

Environmental Asbestos Exposure.—The recent demonstration, by South African [9] and British [8] investigators of pleural and peritoneal neoplasms among individuals who had chance environmental exposure to asbestos many years before raises the very important question of possible widespread carcinogenic air pollution. The possibility of environmental exposure has long been known. Soon after the initial clarification of asbestosis as a clinic entity, Haddow [10] demonstrated asbestos bodies in a man not employed in the industry but living next door to an asbestos factory. This finding was later mirrored in the finding of chronic beryllium disease among residents of a community near a beryllium factory.[11] What is new, however, is an appreciation of the potential extent of the problem. Thomson and associates [12] have reported the frequent findings of asbestos bodies in the lungs of urban dwellers. Among 6,312 individuals x-rayed in an area about an asbestos mine in Finland, Kiviluoto [13] found 499 cases of pleural calcification of the type characteristically seen among asbestos workers, without obvious cause. In a comparable area without any asbestos mine, no cases were found among 7,101 persons x-rayed. It should be noted that these were not people who worked in the mine—none did—but, rather, were farmers, housewives, and others who lived in the general location. In one subject who came to autopsy, polarized-light microscopy demonstrated asbestos fibers in the lung. Similarly, the lung of a cow grazing near the mine also showed the presence of asbestos.

A particular variety of environmental exposure may be of even greater concern. Asbestos exposure in industry will not be limited to the particular craft that utilizes the material. The floating fibers do not respect job classifications. Thus, for example, insulation workers undoubtedly share their exposure with their workmates in other trades. Intimate contact with asbestos is possible for electricians, plumbers, sheet-metal workers, steamfitters, laborers, carpenters, boiler makers, and foremen; perhaps even the supervising architect should be included.

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MEDICAL INTELLIGENCE

ASBESTOSIS IN A WORKER ENGAGED IN AUTOMOBILE UNDERCOATING

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THE term pneumoconiosis applies to diseases of the lungs caused by inhalation of foreign material. A more specific diagnosis will depend upon correct identification of noxious substances in air samples, sputum and, whenever possible, lung tissues. Among the occupational hazards causing penumoconiosis, asbestos has been identified in exposed mine workers, workers handling insulating materials composed largely of asbestos, workers in the brake-lining industry and workers engaged in asbestos spinning, carding and weaving.[1] Although asbestos bodies have been found in the lungs or sputum of patients with no evidence of pulmonary disease or heavy exposure,[2] presence of asbestos bodies in the lungs of a worker in an occupation hitherto not connected with asbestos makes the following case worth reporting.

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CASE REPORT

F.T., a 41-year-old automobile mechanic, was admitted to a hospital in the vicinity of Boston on December 15, 1958, with an acute febrile disease. He complained of cough, shortness of breath and yellowish expectoration. He had been well until several weeks previously, when he had noted the gradual onset of productive cough, weight loss and excessive fatigue.

On admission he was febrile and coughing. The temperature was moderately elevated. On physical examination there was a congenital anomaly of the right arm and "cyanosis of the ears." Rales and rhonchi were present over both lungs. There was no clubbing of the fingers. Laboratory studies showed "yeastlike bodies" in the sputum. Gastric washings were negative for acid-fast bacilli, and a Papanicolaou smear was negative for malignant cells. Blood studies showed a hematocrit of 47 per cent, a white-cell count of 10,800, with 72 per cent neutrophils, and a sedimentation rate of 10 mm. per hour. A tentative admission diagnosis of pneumonitis was made.

X-ray films taken on the following day showed diffuse bilateral infiltration of the lungs by minute, discrete nodular densities and considerable accentuation of the peribronchial perivascular markings, particularly of the lower lobes. The cardiac contours were not clearly defined, being partially obscured by the infiltration. There was considerable bilateral adenopathy.

A bronchoscopy and bronchography performed on December 31 showed "tracheobronchitis." Thoracotomy in the region of the left lingula on January 5, 1959, revealed that lobes had a fine, granular appearance, suggesting pulmonary fibrosis. Two wedge biopsies from the lingula and the lower lobe showed, microscopically, many areas of foreign-body reaction as well as a few typical asbestos fibers. The foreign-body reaction appeared most marked around areas that stained blue and contained unidentifiable homogenous material. Marked connective-tissue reaction was present. There was no evidence of cancer, tuberculosis or Boeck's sarcoidosis.

The patient made an uneventful recovery, his fever subsided and he was discharged on January 9 greatly improved. He continued to have some greenish tenacious sputum and slight shortness of breath, with wheezing. He returned to work to the same company and was assigned as a parts distributor in the Service Department. On re-examination he still had a moderate cough, with slight dyspnea on exertion, 12 months later.

The occupational history revealed that this man was engaged in his present occupation at the same company as an automobile mechanic for ten years. Previously, he had worked for two years at a saw-manufacturing company, where he operated a cold press, and before this he had worked at a gasoline station. He had a complete physical examination, including a chest film, which was interpreted as normal, when he was first hired ten years before admission to the hospital.

His work consisted of the servicing of new automobiles, essentially from one American car manufacturer.

With the exception of fiberglass in small quantities, no material of significance in occupational diseases was used by this worker in his job.

In addition to his routine duties for the last six years he had been engaged on a contract basis in applying an asbestos-base undercoating on new cars.

This operation was performed in a wing of the garage measuring 15 by 26 feet and closed at both ends by wide sliding doors, which permitted the entrance and exit of automobiles. There were large windows at one side of this wing. The undercoating operation was carried out by means of a hand-type spray gun operated at a pressure of 110 pounds per square inch and directed against the underside of the raised car. No mechanical ventilation and no respiratory protective devices were in use during this operation. A considerable amount of the tarry asbestos-base material was dispersed through the work area and onto the worker's clothes and face. The undercoating material was pumped from a 55-gallon drum. Each car required approximately 4 gallons of the material and thirty to sixty minutes of spraying time. About 1 to 3 cars were sprayed daily by the patient during the first five years, and less during the last year.

Since the illness suggested the possibility that the undercoating of automobiles was responsible for or contributory to the acute pulmonary disease, it was deemed advisable to investigate the undercoating operation presently performed by many automobile sales and service agencies in Massachusetts. To obtain background information, contact was made with the industrial-hygiene units of the large automobile as-

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semblers and other state organizations responsible for the investigation of occupational health hazards. The case history was summarized and forwarded to these organizations with a request for information regarding whether similar cases had been observed in the past. None of these agencies had knowledge of any similar experiences. The information revealed that in plants outside of Massachusetts, an undercoating compound that contained a rubber or cork base was in use. In Massachusetts most of the automobile agencies are using an undercoating material that contains asbestos in an asphalt medium.

The undercoating compounds used by the dealers included in this survey were manufactured by 5 different companies. The material consisted of an asphalt base containing approximately 50 per cent of asbestos by weight. About 10 per cent of the constituents were fillers such as clay. The material was of a mastic consistence, with a petroleum distillate such as a high-flash naphtha or kerosene as the solvent.

Fifteen different types of American automobiles sold and serviced in Metropolitan Boston were selected for a cross-sectional survey to study the undercoating operation and its potential hazards. Fifty-five automobile sales and service agencies were visited to obtain information on the number of cars undercoated annually, the number of employees engaged in this operation, the methods of operation, the protective equipment worn by the operators and the composition of the undercoating compound being utilized.

The survey revealed that the number of cars undercoated by these establishments ranged from as low as 2 to as high as 600 per year. There was a recent tendency to reduction in the amount of undercoating as compared with previous years, since most of the new cars are delivered undercoated by the assemblers.

The undercoating operation was carried on as a rule by 1 or 2 employees at the close of the workday, when the operator performed the undercoating operation on his own time, with extra compensation for each car undercoated. The location of the operations varied at the different agencies. In most cases a section of the garage area was set aside for the operation, which might be performed in an open area or segregated from the main repair operations by means of canvas curtains used to prevent the material, during spraying, from lodging on other automobiles in the area. About 25 per cent of the agencies had separate workrooms used exclusively for this operation. The undercoating material was usually received in 55-gallon drums and dumped into 275-gallon storage tanks. Under a pressure of approximately 100 pounds per square inch the mastic material was forced through a spray gun of the type used in spray painting. Parts of the automobile that were not undercoated were protected during spraying by masking tape. The wheels were removed, and the automobile raised by means of hydraulic lift. The operator sprayed the underside of the automobile. The actual spraying operation required approximately fifteen to thirty minutes. There was considerable back splash of the material, striking all parts of the operator, including the face. This contamination resulted in a reluctance of workers to undertake this work as a full-time occupation and led to frequent rotation of workers in this particular job. General means of ventilation such as a wall fan were provided at a few locations. It was noted that no special precautions were taken to prevent the inhalation of the dispersed undercoating compound. Some of the operators were provided with plastic face shields or coated their faces with petroleum jelly.

Industrial-hygiene units that had surveyed the undercoating operations in the past were interested primarily in the solvents used in the undercoating material. Their analysis did not reveal any substantial hazard attributable to these solvents. Our survey was directed at determining whether asbestos constituted a hazard after the prolonged use of certain undercoating compounds. Atmospheric samples were obtained during the actual spraying of the undercoating material. The average concentrations varied from 500,000 to 1,500,000 particles of asbestos per cubic foot of air. Microscopical analysis showed that the asbestos was present in the form of dust particles, less than 10 microns in length. The present maximum allowable concentration for asbestos dust particles less than 10 microns long dispersed in the working atmosphere, as set by the American Conference of Governmental Industrial Hygienists, is 5,000,000 particles of asbestos per cubic foot of air. The maximum allowable concentration for long fibers greater than 10 microns in length has been tentatively set in Massachusetts as 1,000,000 fibers per cubic foot of air. The atmospheric contamination due to asbestos in the operations surveyed, based on available standards, was not excessive. It was noted that in some cases the air samples picked up asbestos particles surrounded by an asphalt coating.

Additional information about the health of certain individual operators was obtained. At establishments where at least 150 cars were undercoated annually it was recommended that an x-ray film of the chest (14 by 17 inches) be taken of each employee engaged in this operation. In some establishments arrangements were made with the local health units for free x-ray studies; in others, the expenses were underwritten by the employer. Twenty-five of the 75 employees involved had such films taken. All x-ray studies were interpreted as negative.

Discussion

The observations here recorded raise the question whether the use of an undercoating material containing asbestos had a direct relation to the clinical and

anatomic findings. The material that the patient used contained asbestos of various fiber sizes in an asphalt base and was held in a mastic form by a solvent of the petroleum-distillate type. Lung biopsy revealed the presence of asbestos bodies of characteristic appearance. The unusual finding in this case was the presence of numerous amorphous bluish-staining deposits fairly widely dispersed throughout the specimen and surrounded by inflammatory and connective-tissue reaction. The x-ray appearance of finely granular infiltration throughout both lower lobes and the possibility that the sputum also contained asbestos bodies ("yeastlike bodies") indicate that the lung process in this man was of a chronic nature consistent with pneumoconiosis and the direct local action of foreign material such as asphalt and asbestos. This is also suggested by some of the air tests obtained in the survey.

From the point of view of occupational health it is significant that the patient, in contrast to the workers observed in the survey, had been daily and continuously engaged in the undercoating operation for more than five years, that no history of previous lung disease or exposures to noxious material could be demonstrated and that the survey had shown that the spraying procedure was accompanied by considerable back splash into the breathing zone of the worker.

The cross-sectional survey here reported indicates that under prevailing methods of operation, the use of an asbestos-base undercoating, when limited to short exposures, has not resulted in an occupational hazard. However, if this operation is performed continuously without adequate ventilation and protective respiratory equipment it may expose the worker to the potential danger of pneumoconiosis.

As a protective measure, it is therefore recommended that workers using asbestos-base undercoating material over a period of ten hours weekly or more for periods longer than one year should observe the following rules:

Pre-employment and periodic x-ray studies of the chest, with the use of large-sized films (14 by 17 inches) should be made.

When possible the workers should be rotated.

Approved respiratory devices, with additional protection for the eyes, should be worn.

Mechanical ventilation should be provided in the immediate vicinity of the work area.

Summary

On the basis of a clinical and pathological study of a case of pneumoconiosis in a worker engaged in the undercoating of automobiles with an asbestos-base compound, a cross-sectional survey of this operation in the Commonwealth of Massachusetts was undertaken. Appropriate recommendations to protect workers with prolonged and continuous exposure to this operation are made.

We are indebted to R. E. Farrand, M.D., of Fitchburg, for permission to report this case, and to H. J. Spalding, M.D., pathologist, Burbank Hospital, Fitchburg, for review of the pathological specimens.

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NEPHROTIC SYNDROME CAUSED BY PROBENECID*

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THE nephrotic syndrome has been reported to occur in the course of therapy with a number of drugs including trimethadione,[1,2] paramethadione,[2] mercurial diuretics,[3] tolbutamide[5] and perchlorate,[6] as well as in association with sensitivity to poison oak[7] and bee sting.[8]

In the case reported below, the nephrotic syndrome appeared to be the result of a reaction to probenecid.¶ Although proteinuria and renal failure are known to occur in the course of chronic gout, the nephrotic syndrome has not heretofore been reported as a result of gouty nephritis or in association with probenecid therapy.

Case Report

A 52-year-old man was first seen at the Grace-New Haven Hospital in January, 1954, because of acute arthritis of the right knee. Physical examination was negative except for the painful swelling of the knee. The blood pressure was 118/85. Urinalysis was negative. The nonprotein nitrogen was 38 mg., the fasting blood sugar 100 mg., and the serum uric acid on 2 occasions 9.0 mg. and 9.9 mg. per 100 ml. The acute attack of gout responded in 24 hours without specific therapy, and he was discharged on probenecid, 1 gm. twice a day, and a low-purine diet. Eighteen days later he experienced urticaria, which subsided when probenecid was discontinued. He was then advised to stop probenecid and to take colchicine, 1.2 mg. daily, as prophylaxis against further acute attacks of gout.

In March, 1954, probenecid, 1 gm. daily, was restarted. When next seen in May, 1954, the patient had gained 10 kg. (22 pounds), but no evidence of edema was present. Two weeks later the weight had increased further, and gross pitting edema was noted. No urinalysis was done at that

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