December 27, 1948

Dr. Charles H. Sullivan, Jr.
Armstrong Cork Company
Lancaster, Pa.

Filed in
Dr. McConnell
folder

Dear Dr. Sullivan:

I am enclosing a short sketch of my services taken from "Who's Who in Industrial Medicine." You may use any part you wish in my introduction.

Very truly yours,

W. J. McConnell, M.D.
Assistant Medical Director
Director, Industrial Health Section

Enc.

Health Hazards Associated With The Use Of Solvents

H. J. McConnell, M.D.*

* * *

As striking as the advances have been in new methods and new technique in determining the volatile solvents in the working environment during recent years, our knowledge of their toxic action has not kept pace with the ever-mounting number of newly perfected solvents used in increasing quantity and variety in nearly every manufacturing industry today. Further difficulties frequently are experienced in identifying their chemical compositions which are concealed by the use of various trade names.

Although it is quite well known that the inhalation of concentrated vapors of certain of the volatile solvents is dangerous, it too often happens that the danger inherent in some of the newer solvents has not been suspected until fatal poisoning has occurred or instances of sickness have been traced back to their exposure. Poisoning from the industrial use of such solvents can be avoided only by more scientific and, incidentally, less expensive methods of determining their toxic properties before use.

Despite the fact that the cellular reactions of small laboratory animals are not strictly analogous to those of man, exposure of these animals to varying concentrations of solvent materials provide relevant evidence of the probable effects of these substances on man. While naturally there may be differences, both qualitative and quantitative, in behavior between man and laboratory animals, sufficient manifestation of the type of toxic action

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usually can be predicted. However, it must be remembered that the toxic threshold of solvent vapors for man can only be roughly approximated from exposure of animals, even when dosage is carefully correlated with body weight.

Aside from individual variations in the physiological responses to toxic vapors in man, these vapors present a special risk to those individuals who are either unusually sensitive to poisons or are suffering from organic diseases which increase their susceptibility. Therefore, as desirable as some measure of the harmfulness of an atmosphere in which men work undoubtedly is, our current tables of minimum toxic concentrations must be accepted with due consideration for these variable factors affecting the individual.

After inhalation, solvent vapors enter into the general circulation and are rapidly distributed to the heart and to the central nervous system, where they can produce either a general toxic action without harm to the respiratory passages or a mixed effect, being generally toxic and locally irritant. Seldom do these exert their influence on a single organ. Changes usually are found in the blood, kidneys, liver, heart, nervous system and other organs. A few irrespirable vapors act so violently in the upper respiratory tract and lungs that their general toxic effects are secondary considerations.

Other avenues of entrance into the body are by way of the gastrointestinal tract and by absorption through the intact skin. Substances ingested, if they do not initiate vomiting, may be absorbed by the intestine in varying amounts, depending on their properties and their reaction on the stomach contents. A major portion of the absorbed material passes into the portal circulation and may be satisfactorily handled by the liver. The intact skin is vulnerable to some of the more toxic fat soluble vapors. Dermatitis may result from the continual

handling of almost any of them, either because of their fat solvent properties or because of sensitivity, natural or acquired. A few may affect the eyes and any optic neuritis or paralysis in workers exposed should give rise to investigation.

With few exceptions, all volatile solvents, if inhaled in sufficient concentration for a sufficient length of time, may be said to be detrimental to health, although their early effects on the body frequently go unrecognized or are regarded as minor ailments of no consequence, because of inadequate knowledge of their physiologic properties.

Of the hydrocarbon groups of solvents encountered in industry, the most important from the point of view of toxicity is the aromatic series. benzene (benzol), one of the most volatile of the group, is widely used in industry, not only as a solvent and diluent, but in large quantities in the blending of motor fuels and in the chemical industries. The central nervous system primarily is affected in acute poisoning in contrast to its destructive action on the blood, the blood-forming organs, and the blood vessels in chronic poisoning. High concentrations of benzene are rapidly fatal, whereas exposures of moderate concentrations cause symptoms similar to alcoholic intoxication at first, but are followed by symptoms of progressive depression, twitchings, convulsions, and paralysis with loss of consciousness. These are not common types of poisoning and usually result from an accident or in cleaning vats, tanks, etc. Repeated exposures to small concentrations of benzene cause gastrointestinal disturbances, headaches, vertigo, weakness and frequently nosebleeds, and small hemorrhages of the gums and conjunctive. The blood undergoes changes as a result of destruction of the blood-forming organs. Anemia is an important diagnostic factor.

While there may be at first a slight leukocytosis, a erythropenia and a leukopenia with a slight reduction in hemoglobin content follow. These findings are supported by Dr. P. A. Davis, who has been observing and examining between 6,000 and 7,000 men and women whose work involved contact with various compounds containing benzene for the last twenty years.

A recent number of the Journal of Industrial Hygiene and Toxicology, however, has published a series of five papers on chronic industrial benzene poisoning which challenge, more or less positively, our present conception of the diagnosis, clinical course and pathology of chronic benzene poisoning. While time will not permit a discussion of the controversial factors presented in these papers, it is obvious that the toxic effects of benzene must be restudied in the light of these new observations.

The action of the higher homologues of benzene are also somewhat controversial, perhaps because in industrial use benzene frequently is present in the mixture. Under ordinary circumstances, they are obviously less dangerous than benzene because they are less volatile. Toluene, though more irritant and exerting a slightly more narcotic effect on animals, at least does not appear to have a destructive effect on the blood and the blood-forming organs so characteristic of benzene. Xylene, which commercially is usually a mixture of three isomeric xylenes, is believed by some authorities, and denied by others, to be more toxic in producing narcotic effects than benzene and toluene. Like toluene, it does not cause characteristic changes in the blood or in the blood-forming organs.

The higher homologues of benzene, other than toluene and xylene, are seldom used in pure form, but in mixtures of varying composition. Although further

investigation may demonstrate toxic properties, at present they are considered much less dangerous than benzene.

The coal tar solvent naphthas should be distinguished from the petroleum distillate naphtha. Crude naphtha consists of a mixture of benzene, toluene, xylene, and other impurities. Coal tar solvent naphtha, though varying in composition, consists of xylenes and cumenes and small quantities of hydrocarbons of the paraffin series. Miflash naphtha, according to Dr. Alice Hamilton, is a mixture of toluene, xylene, and the still heavier cumenes. Solvent naphtha consists of xylene and higher homologues, and is relatively free of benzene and toluene. These may produce acute poisoning in man when severely exposed and death may result from respiratory paralysis.

Naphthalene, a distillation product of coal tar, causes local irritation as well as systemic toxic effects. It may cause a rather severe eczema of the skin and injury to the cornea. Naphthalene has been suspected by a number of observers as a cause of cataract. Gastrointestinal disturbances and irritation of the kidneys have been observed from inhalation of its hot vapors.

The coal tar distillates also furnish us with an interesting group of pharmaceuticals, such as aspirin, novocaine, and sulphanilamide, to mention only a few. Sulfapyridine, which has been so successful in the treatment of pneumonia, is produced from the tar base pyridine. Of current interest is the utilization of coal tar derivatives in the production of nylon from which textile fibers are now being made.

The chlorinated hydrocarbon group of compounds are important because of their anesthetic and poisonous action. The member of this group with which we are all familiar is chloroform which possesses high solvent powers, but does not find wide application in industry because of its well known anesthetic

properties. The most widely used solvent of this group is carbon tetrachloride. It is used extensively as a cleaning fluid, in fire extinguishers, and as a solvent for fats and oils. It has been used medically as a vermifuge in hookworm disease. It is very much like chloroform but is more toxic. Its narcotic effects are less marked than those of chloroform, but its toxic effects on the liver, heart, and kidneys are much more rapid.

Trichlorethylene, because of its solvent action on fats and oils, is used as a thinner for coatings and varnishes, and as a solvent for rubber, replacing carbon tetrachloride in many instances. It is marketed under many different trade names. Its chief industrial application, however, is in the metal industry and is used in degreasing of metal parts. It is claimed to be even less narcotic than carbon tetrachloride, but its toxicity is in the same order. Because of the mild feeling of intoxication it creates when inhaled in low concentrations a craving for such exposure may develop leading to anemia and nervous sequelae.

Tetrachlorethane, which has a wide field of application on account of its high solvent power, is the most poisonous of the chlorinated hydrocarbons. Its toxic effects have restricted its use in the aviation industry, where it was widely used during the World War in aeroplane "dope". In acute poisoning the symptoms develop rapidly resulting in atrophy of the liver, jaundice, and fatty changes in other viscera, especially the heart. In chronic cases the blood changes are marked, leading to secondary anemia, hemoglobinuria and hemolysis.

Dichlorethane (ethylene dichloride) used in the chemical industry and as a cleaning fluid and in the extraction of oils and fats, has irritant and narcotic properties but so far has not given rise to chronic symptoms from prolonged exposure. It is frequently mixed with either trichlorethylene or carbon tetrachloride.

Dichlorethylene is used as a solvent for cellulose acetate and as a dry-cleaning agent, as well as in the rubber and perfume industries. It has narcotic and irritant properties, but only one fatal case noted by Hamilton in 1934 has been reported from its industrial use.

Tetrachlorethylene (perchlorethylene) used as a constituent of solvent soaps and as a fat solvent in the printing industry and sometimes as a dry cleaning agent, is a relatively new solvent and is less narcotic than the majority, although symptoms of drowsiness, headache, giddiness, and a narrowing of the visual field have been reported from repeated daily inhalations.

Chlorinated naphthalene products, used, among other ways, as a form of wax in insulating, have been clearly shown to cause severe skin troubles and have caused symptoms of hepatic disturbances with fatal cases showing signs of toxic jaundice. It is interesting that these substances appear relatively harmless when cold, but when heated, despite the fact that they do not decompose, their vapors cause acne more or less characteristic of wax or tar acne.

Among the many toxic chemicals resulting from the introduction of sulphur into aliphatic hydrocarbons, carbon disulfide deserves special mention because of its varied effects on the body following repeated exposure to small concentrations. It is used as a solvent for fatty bodies in essential oils and resins as well as in insecticides, and in the rayon, rubber and chemical industries. In high concentrations, carbon disulfide fumes affect a person somewhat like chloroform and, if exposure is continued, the fumes cause complete insensibility, coma and death.

Chronic poisoning is characterized by persistent headache, somnolence during the day, sleeplessness at night, and weakness of the limbs. It is extremely

damaging to the nervous system, causing polyneuritis, visual disturbances, tremors, mental confusion, severe psychosis and ataxia. An important characteristic of this poison is its effect on the higher nerve centers. Not only is it damaging to the eyes, but the disappearance of the corneal reflexes is said to be an early sign of poisoning. Recovery is slow and if the poisoning is severe, there may be permanent injury. One attack may predispose to a second.

Petroleum hydrocarbons, which represent mixtures of hydrocarbons having no definite composition, are difficult to appraise physiologically because of inadequate knowledge of the source of their product and the process of manufacture. They have an extensive use as an automobile fuel and as solvents in the rubber, electrical, paint and varnish, glue, shoe, and printing industries. They have a mildly anesthetic action and in high concentration cause headache, dizziness, blurred vision and even loss of consciousness. Generally speaking, it appears that their toxicity increases with their specific gravity and boiling point. Any chronic blood damage from this group is controversial and, if found, probably points to the advisability of looking for some other exposure or cause, particularly the admixture of one of the more dangerous types of solvents in the substances handled. Like all the other solvents, these can readily cause a dermatitis.

Turpentine, which is a mixture of terpenes, closely related to the aromatic hydrocarbons, is used chiefly as a solvent or thinner in the paint and varnish industry and to some extent for cleaning type and rollers of printing machines and for cleaning fabrics. Turpentine vapors have a direct action on the skin, causing reddening and not infrequently an obstinate eczema. Irritation of mucous membranes and burning of the eyes are experienced with comparatively low concentrations. The inhalation of heavy fumes of turpentine induce symptoms of a mild narcotic poisoning. Nephritis has been reported from prolonged exposure.

A larger group of newer solvents which is rapidly coming into greater prominence is the alcohol-ethers, or oxides. Often these may be compounded with phthalic acid, oxalic acid or formic acid, thereby introducing the possibility of a second toxic agent. Our knowledge of this group is largely based on animal experimentation, though a number of cases of industrial poisoning have been reported. As a group, they are narcotic or anesthetic, and some of them are known to be toxic in the higher concentrations, but there is little information concerning their chronic effects or the maximum permissible concentration.

The alcohols and their ethers are perhaps the most widely used of the various classes of solvents. As a group, they are narcotic and injurious to the nervous system. The toxicity of the alcohols generally increases with the molecular weight, but the decreased solubility and volatility of the higher alcohols partially offset the danger of exposure to concentrated vapors. The exception to this rule is wood alcohol which includes methyl alcohol and methanol and is prepared both by distillation of wood and synthetically from carbon monoxide and hydrogen. Although its narcotic effect is less marked than that of ethyl alcohol, it is a powerful nerve poison which specifically affects the optic nerves and may result in blindness and death. Owing to the accumulation within the body tissues of its oxidation products, formaldehyde and formic acid, and its slow excretion from the body, its effects on the nervous system are hazardous from continued exposure to even small concentrations.

While propyl, isopropyl, and butyl alcohol share with the other alcohols a narcotic effect on the central nervous structures, their industrial use has carried little danger for workers. Inhalation experiments with the secondary and tertiary alcohols are required to indicate their danger from an industrial point of view.

The most important group of solvents for nitrocellulose and cellulose acetate are found among the esters, ketones, glycols and their ethers, and furfurals. All are narcotic in sufficiently heavy concentrations and in proportion to their volatilities. Whereas fatal accidents are very rare, evidence is beginning to appear pointing to their physiologic effects as being more dangerous than they were formerly considered.

Although the most common effect noticed from the inhalation of amyl acetate, known as "banana oil" and readily recognized by its sweet, sickening odor, is merely that of irritation on mucous membranes, one death which was attributed to amyl acetate intoxication is reported in the literature. A fatal case of acute poisoning by ethyl acetate also has been reported in a workman who was painting the interior of a tank with paint containing 30% ethyl acetate.

Methyl, propyl, and butyl acetate are other members of the ester group of solvents. Ethyl and butyl acetates are the most widely used of all the solvents of nitrocellulose.

Of the ketone group of solvents, acetone, a highly volatile, inflammable fluid, is probably the most widely used, and it is one of the most powerful solvents known. Absorption takes place through all mucous membranes and is excreted through the lungs and urine. It has a narcotic action more pronounced than that of chloroform and is irritant to the conjunctiva and skin.

Several ketones of higher molecular weight have been recommended as good solvents for nitrocellulose, but they have little or no effect on cellulose acetate. The more important members of this group are methyl-propyl ketone, or pentanone, and methyl-butyl ketone, or hexanone. Their acute effects on guinea-pigs were studied by Yant and his colleagues, who report that death results from a state of narcosis rather than from the irritation of the lungs. All fatalities occurred during the exposure. Men momentarily exposed to approximately 1.3 and 5 per cent of pentanone vapor and to 1.23 and 2 per cent of hexanone vapor complained

of irritation of the eyes and nasal passages and of a characteristic strong odor.

The glycols and their derivatives are used in a variety of industrial processes. Ethylene glycol is sometimes used as a constituent of anti-freeze mixtures and the drinking of such a mixture has occasioned the only serious cases of poisoning. Death appears to be due to respiratory failure and convulsions.

The mono-ethyl ether of ethylene glycol, or "cellosolve" is an important solvent of this group. It is a stable liquid and it is used as a solvent for nitrocellulose and resins. It offers the advantage over most other solvents of similar boiling point in being nearly odorless in low concentrations. It was found by Waite, Patty and Yant to be toxic for animals. During the investigation two men, who breathed air containing 0.6 per cent by volume of the vapors for a few seconds, reported the atmosphere to be irritating to the eyes and of a disagreeable odor. However, very little evidence of any injury to health from the use of cellosolve has been reported. Under certain conditions, the mono-methyl ether of ethylene glycol was found by Greenburg, Mayers and Goldwater to cause or, at any rate, contribute to abnormal blood changes, with or without neurological findings, among a small group of workers in a shirt factory, where this substance was used in conjunction with denatured alcohol as a solvent for cellulose acetate.

The inhalation of glycol chlorohydrin, or ethylene chlorohydrin, causes local irritation of surface mucous membranes and deeper lung tissue. Its toxic effect is that of a severe nerve and metabolic poison. Fatal cases of acute poisoning have been reported.

The second ether of ethylene glycol, or dioxan, is a solvent of numerous products such as fats, wax, oils, gums, etc. It is used in the lacquer, celluloid and textile industries and in the manufacture of polishes, shoe creams, glue, cosmetics and pharmaceutical preparations. At low concentrations, aside from a

workers who have been exposed to silica dust in these various occupations and are now engaged in other forms of work.

OTHER INDUSTRIES OFFERING EXPOSURE TO SILICA DUST

The silica hazard, to be sure, is known to be present in mining and manufacturing industries other than those we have mentioned and in the building industry as well. Our survey would not be complete without giving some consideration to the large number of workers exposed to the hazard in these industries. Bituminous coal-mining, for example, employs 458,000 men, among whom, as among anthracite miners, are a number who are engaged in rock-drilling, and consequently are exposed to silica dust. Among the manufacturing industries with a more or less definite silica hazard, should be mentioned the making of abrasive wheels, sandpaper, hones and mineral earths, employing together about 8,000 men; the manufacture of mineral fertilizers, 21,000 men; of clay products (other than pottery manufacture) 93,000 men; and of cordage and jute-goods manufacture, 19,000 workers.

Many branches of construction work require considerable drilling in siliceous rock. In New York City alone, about 2,000 men at one time were employed at drilling, excavating, and blasting in rock containing a high percentage of free silica. Laborers and helpers in general construction and in road and street construction, together with steam- and street-railroad laborers and laborers in public service, many of whom are engaged in construction work, numbered 1,337,000 in 1930. It is in this class that most of the workers in the building trades exposed to silica dust will be found.

The combined total of workers included in this group of miscellaneous industries numbers about 2,000,000. In all probability only a small fraction of this total is exposed to the silica hazard. If, however, we apply even the conservative figure of 5 per cent as our estimate of those who would be exposed to the hazard, we shall have to add 100,000 workers to our general estimate.

Our very rough, but obviously conservative, estimate of the number of workers exposed to silica dust to a harmful degree in the United States is, therefore, upward of 500,000. We recognize that the data upon which this estimate is based leave much to be desired. Only the known exposures could be taken into account. The interesting thing about silicosis, which must not be overlooked in considering its importance in the nation's toll of ill health, is the unexpected places in which it is being

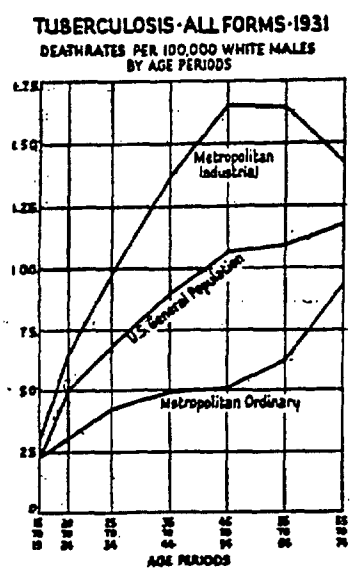
uncovered, and the revelations as to the extent of the damage being done in industries in which the hazard is known to exist.

What does the exposure of more than half a million workers to silica dust mean in terms of the prevalence of silicosis and of tuberculosis? We cannot, for the reasons given at the beginning of this paper, give a direct answer to these questions. Many competent students of industrial hygiene, as you are well aware, have shown an extremely high incidence of silicosis in those industries in which they have carried out thorough investigations. They have shown, too, how frequently silicosis terminates in tuberculosis. It is not our purpose to review these findings. Valuable as they are, they are entirely inadequate to serve as the basis for anything like a countrywide estimate of the number of silicotics. We think, however, that their findings, taken in conjunction with our estimate of persons exposed to silica dust, amply justify the assumption that, first of all, a very large number of industrial workers are the victims of silicosis, and second, that these silicotics contribute to the high incidence of tuberculosis and the high death-rates of industrial workers from this disease, especially at the later ages of life.

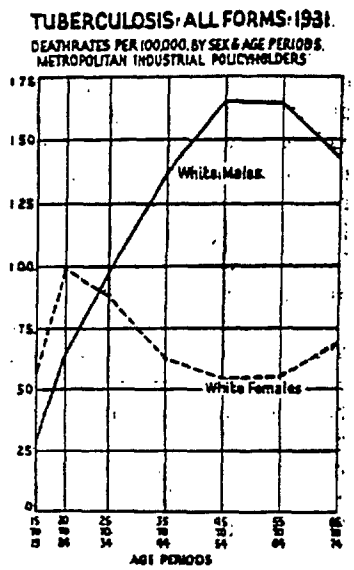
This latter statement finds partial support in certain mortality statistics made available to us. We present two graphs which are interesting in that they show the age-curve of tuberculosis death-rates for several population groups with varying degrees of industrial exposure. The graph on the right shows how the rates for female industrial policyholders compare with those of male industrial policyholders. The maximum female mortality, it will be noticed, occurs in the age-period 20–24 years, after which it rapidly declines to the age-period 55–64 years. The male mortality, on the other hand, rises rapidly with age, reaching its maximum in the period 45–64 years. At these ages, the death-rate for males is three times that of females, whereas at ages 20–24 the female rate exceeds the male rate by about one-third.

Graph 1, on the left, compares the mortality of three groups, all males, namely industrial policyholders, representing the greatest industrial exposure; ordinary policyholders, representing the least, with the males of the general population midway between. At each age, the lowest death-rate is shown for the ordinary policyholders, and the highest rate for the industrial policyholders. The disparity in rates increases rapidly with age from 15 to 19 years, when the rate for industrial policyholders is 28 per cent in excess of that for ordinary policyholders, to ages 45–54, when the industrial rate is three and one-quarter times the ordinary rate.

These differences may not be ascribed to industrial influences alone, least of all do they measure the effect of silicosis. The classes vary as to economic status for one thing, which, as has been well established, has an important bearing on tuberculosis mortality. The very considerable differences do, however, suggest the influence of silicosis, especially as the greatest disparity in the mortality rates occurs in those ages in which silicosis is most likely to show its effects.



GRAPH 1



GRAPH 2

More indicative of the direct influence of silicosis on the general death-rate from tuberculosis is a comparison of the mortality from this cause for men engaged in specific occupations with exposure to silica dust with the mortality for those not exposed. There is, unfortunately, in the United States no complete analysis of occupational mortality by which we might measure the effect of exposure to silica dust on the health and longevity of workers. The most recent data are found in an investigation made by twelve life-insurance companies into the occupational mortality of persons insured under ordinary policies (policies of $1,000 or more, payable quarterly or at longer intervals). The study

analyzes the mortality experience of those insured under policies issued during the years 1915 to 1926. The method employed was to compare the actual number of deaths, which occurred among men engaged in specified occupations, with the expected number of deaths on the basis of death-rates prevailing among standard lives of the same ages and for the same duration of insurance. Although these figures have been presented before, they will bear repetition.[3] It must be kept in mind that the standard of comparison is a group of selected lives insured under ordinary policies and not the general population. To offset this, however, it should be pointed out that the men in these specific occupations also underwent insurance selection. Table 1 shows the figures for occupations exposed to silica dust.

Table 1 bears out in a striking way what is implied by exposure to silica dust in terms of tuberculosis. The occupations showing the highest ratios of actual to expected deaths were, in order: underground lead- and zinc-miners, granite- and sandstone-cutters, copper-miners and gold- and silver-miners. All of these workers are exposed to a serious silica hazard. The ratios of actual to expected deaths from tuberculosis were, respectively: 1,833 per cent, 976 per cent, 913 per cent, and 804 per cent. There were actually 60 deaths from tuberculosis among these men as against six expected. Tuberculosis was responsible for one-half of all the deaths of lead- and zinc-miners, 29 per cent of the deaths of copper-miners, and 20 per cent of those among gold- and silver-miners. In all of these three mining classes, deaths from tuberculosis exceeded deaths from accidental injuries. Among iron-miners, who are, on the whole, probably less exposed to silica dust than are the other mining classes mentioned, we find that the ratio of actual to expected tuberculosis deaths is only 260 per cent. The ratio among quarry-workers was 263 per cent, 7 of the deaths being due to tuberculosis, while 2.7 might have been expected. It was impracticable to separate the granite-quarriers from the limestone-quarriers. If we had the figures, we should probably find that the excess among granite-quarrymen exposed to silica was much greater than among the limestone-quarrymen. This is clearly indicated by the difference in the mortality ratios for the granite- and sandstone-cutters and the marble- and limestone-cutters. The ratio of actual to

[3] *The Prevalence of Tuberculosis in Industry with Observations on the Dust Hazard:* a digest of a paper by Doctor A. J. Lanza and R. J. Vane, read before the Industrial Hygiene Conference, Chicago Tuberculosis Institute, April 25, 1930. Published in *Statistical Bulletin, Metropolitan Life Insurance Company, July, 1930, xi, no. 7.*

expected deaths among the former was 976 per cent, while among the latter it was but 294 per cent. There were 16 deaths from tuberculosis among the granite-cutters, as against 1.7 expected; whereas, there were 3 deaths in the limestone group, against 1 expected.

TABLE 1

Number of deaths expected from specified causes and number which actually occurred among persons engaged in certain occupations exposed to silica dust

Ordinary department mortality experience of twelve life insurance companies 1915–1926*

Occupation	All causes			Respiratory tuberculosis		
	Actual Deaths	Expected Deaths	Percentage, Actual of Expected	Actual Deaths	Expected Deaths	Percentage, Actual of Expected
Metal mine operatives (underground)						
Gold and silver mines	44	9.77	450	9	1.12	804
Lead and zinc mines	22	5.03	437	11	0.60 (1)	1,833 (1)
Copper mines	83	22.63	367	24	2.63	913
Iron mines	33	12.12	272	4	1.54	260
Quarry operatives	37	22.45	165	7	2.66	263
Stone-cutters						
Granite and sandstone	29	17.47	166	16	1.64	976
Marble and limestone	12	10.55	114	3	1.02	294
Metal industries						
Grinders	46	28.65	161	7	3.33	210
Buffers and polishers	89	64.10	139	10	6.80	147
Chippers (not ship)	38	14.14	269	8	1.30	615
Core-makers and sand-moulders	32	22.65	141	3	2.92	103
Iron and steel foundries						
Moulders, founders and casters	202	130.14	155	24	13.38	179
Other operatives						
Drillers, flask-makers, machine-hands, mixers, etc.	45	31.03	145	7	3.03	231

* Compiled from *Joint Occupation Study—1928, Actuarial Society of America and Association of Life Insurance Medical Directors.*

(1) Approximate figure.

In grinding, another occupation in which there is exposure to silica dust, the ratio of actual to expected deaths was 210 per cent and there were 7 deaths from tuberculosis, as against 3.3 expected. Here, again,

we have a mixed hazard, for the sandstone-grinders, who work in the cutlery trade, have extreme exposure to silica dust and undoubtedly suffer inordinately from tuberculosis, whereas those who work on the composition wheels do not have this exposure and probably do not have very high rates from this disease.

The only other large occupational groups suffering exposure to silica dust included in the study are certain selected foundry occupations. An interesting feature of the foundry operatives' mortality, not brought out in the table, is that, while deaths from tuberculosis were higher than expected, extreme ratios are found for influenza and pneumonia rather than for tuberculosis. Dust is probably an important factor also in the high nontuberculous respiratory-disease rate. Chippers of metal had very high death-rates from both influenza-pneumonia and tuberculosis. There were 8 actual deaths from tuberculosis, as against 1.3 expected; and 17 deaths from influenza and pneumonia, as against 2 expected. Moulders, founders and casters had 24 actual deaths from tuberculosis, as against 13.4 expected; while there were 61 deaths from influenza and pneumonia, as against 23 expected.

There is another important question: What is the effect of silicosis in terms of tuberculous infection among other members of the family? Here again we are confronted by a lack of conclusive information. It has always been the impression among metal-mining communities, where silicosis is prevalent, that there is little tuberculosis among miners' families, even when the miner has died of silicotuberculosis. McFarland, and also Russell of the Public Health Service, have commented that in Barre, Vermont, the amount of tuberculosis among families of silicotics was less than among the general population, which they attribute in part to the comparatively late age at which the silicotics become tuberculous. However, Cummings of the Saranac Laboratory has kindly permitted me to quote some conclusions from as yet unpublished studies which he has made.

He studied a number of families of individuals with silicosis plus tuberculosis, the diagnosis being made by X-ray. He divided these into two groups, namely, sputum-positive and sputum-negative by guinea-pig inoculation. Over 50 per cent of the children under sixteen, living in contact with a silicotic having a positive sputum, showed X-ray evidence of tuberculous infection. Less than 30 per cent of the children under sixteen, living in contact with a silicotic having negative sputum, showed X-ray evidence of tuberculosis. All of the tuberculous infection shown

in these children was of the primary type; there were no cases of adult-type tuberculosis. Probably the situation with respect to tuberculosis among families of silicotics varies with the gravity of the silicosis hazard. When silicosis is severe and the individuals become tuberculous at a period when their children are small, there is naturally more likelihood of contact infection than when the silicosis hazard is less severe and becomes manifest at a later period in life when the children are older and have perhaps left the family circle. The whole field of family tuberculosis, in relation to silicosis, awaits careful investigation and study.

To sum up: There are sufficient data to indicate that, in the United States, the number of industrial workers exposed to the silica-dust hazard is in excess of half a million; that the mortality from tuberculosis among these persons is appreciably affected thereby; and that the extent of family infection due to silicosis plus tuberculosis is uncertain; but under some conditions, at least, it may be a serious problem.

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