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INDUSTRIAL DUST

Hygienic Significance, Measurement and Control

BY

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TABLE 4.—NUMBER OF DEATHS EXPECTED FROM SPECIFIED CAUSES AND NUMBER WHICH ACTUALLY OCCURRED AMONG PERSONS ENGAGED IN CERTAIN OCCUPATIONS EXPOSED TO SILICA DUST[1]

Occupation	Deaths from all causes			Deaths from respiratory tuberculosis		
	Actual	Expected	Percentage actual of expected	Actual	Expected	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	1833
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-molders	32	22.65	141	3	2.92	103
Iron and steel foundries:						
Molders, founders, and casters	202	130.14	155	24	13.38	179
Drillers, flask makers, machine hands, mixers, etc.	45	31.03	145	7	3.03	231

[1] Ordinary department mortality experience of twelve life insurance companies, 1915–1926 (159).

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fully that, if he has silicosis, he got it in Canada and Canada will take care of him.

In the case of a first-stage silicotic without tubercular infection, there is no method, as yet, for determining his lessened efficiency. The lung change, if any, caused by his dust inhalation is too small to measure.

McCann and Hurtado (177) have estimated disability from lung fibroses of various origins but have not yet succeeded in developing a method that is simple enough for routine use by compensation boards, insurance examiners, and the like. Their procedures involve various simple measurements of respiratory function but they have not established any methods of detecting fibrosis which are more sensitive than the present X-ray examination.

Of course, industry would welcome a diagnostic routine for detecting early harm from dust or, better, for forecasting harm. No such method is available and it seems fruitless to expect such help. The pneumoconioses are not diagnosed until harm is demonstrable by X-ray.

Collis (42) has stressed the difference between the age groupings in pulmonary tuberculosis and in silicosis, with or without tubercular infection superimposed. Tuberculosis is most prevalent between the ages sixteen to twenty-four, while silicosis rarely becomes disabling until later in life. Inasmuch as a good many years of dust exposure are required before the average case of silicosis (or asbestosis) becomes evident, it is obvious that it is not likely to be acquired early in life. However, there is an epidemiological side to the question which is of great importance and it is that which Collis especially emphasized. The average healthy individual has acquired a fairly effective immunity or resistance to tuberculosis. His chest X-ray is likely to show one or more healed scars or fibrosed areas from tuberculosis. Such an individual, Cummings emphasizes (211), is a better silicosis risk than the man who shows no evidence of past exposure to pulmonary tuberculosis. Consequently it is wise to select for dusty jobs men who are past the age of forty and not young men just taking up a trade.

Asbestosis. The X-ray picture of the typical asbestotic chest is confusing to the layman. The effect is described as a diffuse fibrosis. Characteristic silicotic nodules are absent and even

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the expert roentgenologist withholds his diagnosis until the case history is complete.

The pathology produced by asbestos is not like that of silicosis. The asbestos fibers group about the neck of an alveolus and shut it off, causing what is known as *atelectasis*. There is no definite migration or transportation of the dust particles to the lymph nodes and no formation of the fibrous nodules as shown in Fig. 13. As the atelectatic areas increase, the reduction in lung

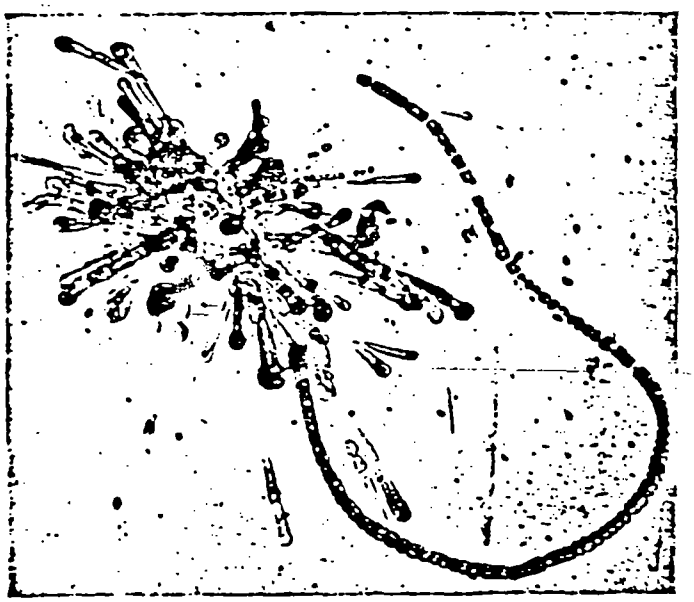

FIG. 15.—Asbestosis bodies in sputum. (*After Ellman* (75); *courtesy J. Ind. Hyg.*)

area causes serious dyspnea or labored breathing. Lanza (158) suggests that the enlarged hearts noted frequently in his cases of second-stage asbestosis may be the result of the increased work of the heart resulting from this condition; it takes more work to pump blood through the atelectatic than through the normal lung.

In silicosis it seems to be a general rule that, after a certain point, the victim's condition grows worse even if his dust exposure has ceased. But Wood and Gloyne (258) state that they have seen patients with asbestosis "whose condition appears to have

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remained stationary since stopping work in the factory," but they advise definitely that the asbestotic individual be removed from his dusty job. Merewether (183) and Lanza are less certain on this point.

Asbestosis Bodies. In the lungs of patients who died after prolonged exposure to asbestos dust and in the sputum of men with considerable asbestos-dust exposure are found what first were called *curious bodies* and later *asbestosis bodies* (Fig. 15) (75). While somewhat similar bodies occur in the lungs of coal workers and even of normal persons, it is admitted that asbestosis bodies in sputum are characteristic of asbestosis. Stewart (223) gives considerable diagnostic weight to their presence.

Pneumoconiosis in Animals. Silicosis has been produced experimentally by quartz dusting guinea pigs, rabbits, mice, cats, and domestic fowl, thus emphasizing the specificity of quartz dust to biological tissue. In like manner asbestosis has been produced in experimental animals.

The Equidae, such as horses and mules, apparently are not susceptible to ordinary pulmonary tuberculosis but there is no reason to believe they have any special immunity or resistance to silicosis. The normal silica content of horses' or mules' lungs is not known but it would seem wholly reasonable to use the lungs of animals which have worked 5 to 15 years underground in mines as physiological dust samples. A brief report on this subject was published by Haynes (121) but data such as one needs are singularly lacking.

TOXIC DUSTS

Poisoning from inhaling toxic dusts is much more likely than poisoning from swallowing them. Dusts that reach the lungs may pass directly into the blood stream, thence to the heart, and immediately be pumped all over the body. Distribution to all the body tissue is thus brought about rapidly and effectively. But dust taken in with the food goes to the stomach and the major part passes out in the feces. Some is picked up by the portal blood circulation and moves on to the liver. That portion which causes poisoning must first pass through the liver, which is an effective filter and detoxifier; only then can it enter the general circulation.

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The practical significance of this physiological distinction between the two ports of entry of dust is considerable. Hamilton (103) states,

> A great deal of money has been wasted by well-meaning employers who sought to protect lead furnacemen or oxide roasters or white lead grinders against poisoning, by providing baths and lunchrooms and clean overalls and mouth washes and such, instead of preventing the escape of lead into the air the men were obliged to breathe, and unfortunately this has sometimes been done under a physician's advice. It must never be forgotten that the great majority of industrial poisons enter the body with the inspired air and that while a workman eats only three times a day he breathes sixteen times a minute during the eight or ten hours of his working day.

Goadby (88) found that cats dusted with lead acquired lead poisoning more easily than did a control animal which was fed over ten times the total lead dosage for the dusted animals. Drinker and Shaw (61) showed how efficiently the liver removed foreign dust particles injected into the blood stream. Minot (188) emphasized the much greater danger of lead poisoning from inhaled than from swallowed dusts. Blumgart (24) showed that the absorption of lead directly in the nasal passages was rapid and might be "of a magnitude far in excess of the minimal dose by mouth."

All this experimental evidence confirms the view so often expressed by the late Sir Thomas Legge (162), but which is only now beginning to be accepted, namely, that there is far more danger of being poisoned by inhaling toxic dusts than by ingesting them in food or drink.

Metal-fume Fever. Of interest in connection with the breathing of dusts is metal-fume fever, a transient noncumulative malady that results from breathing rather heavy concentrations of metal fumes like zinc oxide, copper oxide, magnesium oxide, lead, probably lead oxide (227), and manganese dioxide.

About 2 to 8 hr. after a heavy exposure to the well-dispersed metallic or metallic-oxide fumes, the victim experiences chills followed by fever like that of malaria or the protein reaction following a typhoid inoculation. The patient's fever may reach uncomfortable heights (we have recorded 104°F.) and the next day he feels debilitated but generally can go to work. With

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the fever goes an increased white-blood-cell count or leucocytosis like that experienced in any infection. By the next morning the fever has abated but the leucocytosis persists (Fig. 16). Then the victim is fairly immune; generally he can take another inhalation without experiencing a second attack (70). In industry it is a commonplace that attacks on successive days are unlikely.

The cause of this peculiar malady is probably the absorption of protein material which results from the action of the inhaled

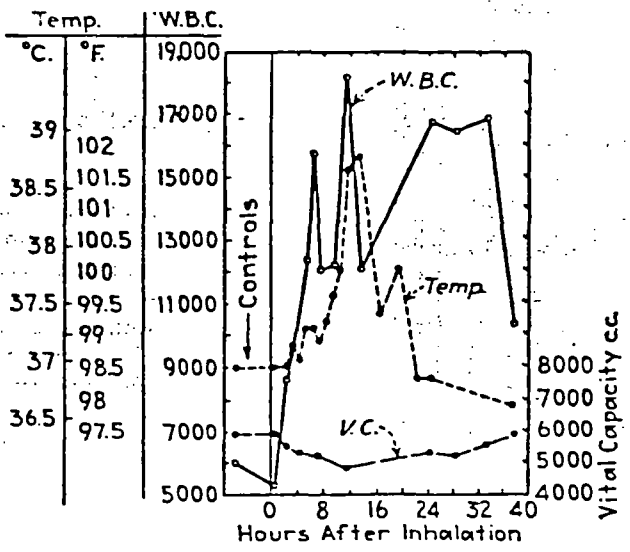

FIG. 16.—A typical attack of metal-fume fever, showing increase in leucocyte count, body temperature, and drop in vital capacity. Note that fever abates before the white count returns to normal. (*After Sturgis et al.; courtesy J. Ind. Hyg.*)

fume particles upon the tissue of the respiratory passages. In our own experiments we found that the chills and fever were acquired far more easily if one took a few deep breaths at a rate of say five breaths per minute than at the normal rate of twelve to fifteen breaths per minute. Slow, deep breathing insures penetration into the alveolar spaces.

Electric or acetylene welding in a confined space may generate metal-fume concentrations in excess of those used by Lehmann or Drinker in studying metal-fume fever. At present there are no data on the effects of breathing dense concentrations for

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