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Silicosis in Shipyard Sandblasters[1]

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In sandblasting, a stream of sand is projected under pressure of 60 to 120 psi to prepare metallic surfaces for the application of paint or other coatings. This method of abrasive cleaning which is employed in shipyards to treat the external and internal surfaces of ships and boats produces high concentrations of respirable free silica within the working atmosphere. Despite its prohibition in the United Kingdom in 1949 (1) after earlier demonstration that it was associated with an accelerated course of silicosis (2), sandblasting has been extensively used in the United States since World War II. The average duration of exposure to silica reported in fatal silicosis in sandblasters was 10 years as opposed to an average of 40 years for all occupations related to the fatal disease (2).

Previous data on dust levels for sandblasting within enclosed spaces are limited. Hanemann investigated a small shipyard in Louisiana in which there were four fatal cases of silicosis after an average duration of exposure to sand of 4 years (3). The work was performed in a building 60 by 130 ft with a ceiling 30 ft high which was open at both ends, but ventilated only by a moveable fan. The workers alternated as blasters and wore air-supplied masks only while blasting. Particles less than 10 μm were sampled with a midget impinger. The highest concentration, 71 million particles per cubic foot (mppcf) was sampled in an area within the blasting building where the air velocity was almost zero. The air 25 ft from the sandblaster contained 29 mppcf of dust.

In this paper, silica dust measurements from two open steel fabrication yards in which sandblasting is steadily employed are presented. They were obtained under conditions strongly resembling external operations in a shipyard. The protective capacity of several devices in current use was studied by internal sampling with the MSA gravimetric respirable dust sampler. The clinical, roentgenographic, and physiologic features of silicosis in a group of shipyard workers studied by the author also will be presented.

Figure 1 shows an external sandblasting operation: The blaster wears an air-supplied hood and a sampling pump as he faces the dust cloud. Table 1 shows the effect of increasing degrees of blasting activity upon the dust concentrations sam-

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FIG. 1. Sandblaster wearing air-supplied hood in silica dust cloud.

pled outside and inside the blaster's hood. All sampling was continuous and included both blasting and nonblasting periods. X-ray diffraction analysis was performed on samples in the range of 500 μg–7 mg. All other samples were analyzed by a colorimetric method using a Beckman DU spectrophotometer. Table 1 shows the large amounts of respirable dust, 37.25 mg/m^3, with high silica concentration generated during busy sandblasting. It also compares the protection offered by non-air-supplied hoods with that furnished by air-supplied hoods. The concentrations of respirable dust exceeded the Threshold Limiting Value (TLV) in all operations from slow to busy.

Figure 2 shows a recently introduced air-supplied hood worn by a blaster who also carries two samplers for determining dust concentration within and outside the hood. Table 2 illustrates the protective effect of the new hood. In a slow operation, dust concentrations within this hood during the blasting period were below TLV, but during unhooded intervals respirable dust exposure was excessive, producing a daily accumulation which exceeded the TLV.

Twenty-two sandblasters with silicosis, who worked in shipyards, were studied by the authors from their hospital and consultation practices (Table 3). Seventeen worked in the area around New Orleans; five worked in Jacksonville, Florida. Of the total group, half are dead. The average age at death was 48.5 years for the fatal group and is 43.6 years for the survivors. The average silica exposure was 9.7 years for fatal cases and 12.4 for the survivors. It was noted that almost half of the dead men worked without air-supplied hoods. Four of the fatal cases worked in en-

TABLE 1

RESPIRABLE DUST AND FREE SILICA CONCENTRATIONS DURING SANDBLASTING OPERATIONS[a]

Position of sampler	Level of sandblasting activity: Slow operation (2–2½ hour blasting) Mean respirable dust (mg/m³)	Slow operation Silica (%)	Moderate operation (2½ hr blasting) Mean respirable dust (mg/m³)	Moderate operation Silica (%)	Busy operation Mean respirable dust (mg/m³)	Busy operation Silica (%)
Outside hood	2.0 (6)	67.4	6.9 (6)	54.5	37.2 (7)	83.6
Inside non-air-supplied hoods[b]	1.3 (12)	30.8	2.8 (7)	42.2	11.0 (4)	70.3
Inside air-supplied hoods	0.6 (11)	39.3	2.6 (2)	77.2	5.9 (5)	55.1

[a] Number of samples is given in parentheses.

[b] 35% of sandblasters did not wear respirator under hood.

FIG. 2. Sandblaster wearing modern air-supplied hood and pumps for sampling inside and outside hood.

TABLE 2
SEPARATE SAMPLING OF RESPIRABLE DUST DURING HOODED AND UNHOODED INTERVALS[a]

Position of sampler	Sampling period	Slow operation (2–2½ hr sandblasting) Average Concentration of dust (mg/m^3)	Free silica (%)	TLV
Inside hood	Sandblasting Intervals	0.4 (4)	5.75	1.3
Breathing zone unhooded	Nonblasting intervals	0.8 (4)	32.0	0.3
Hooded and unhooded	Total working day	0.8 (4)	25.5	0.4

[a] Number of samples is given in parentheses.

closed spaces. Massive lesions were demonstrated on chest roentgenograms in eight fatal cases and seven survivors. One fatal case had silico-proteinosis. Fourteen of the sandblasters had mycobacterial infections; eight were caused by atypical mycobacteria, almost invariably *M. kansasii.*

RESULTS

The course of the disease was rapid and is illustrated by a survivor whose case was complicated by *M. kansasii* infection and systemic lupus erythematosus (SLE). The change in appearance of chest X-ray films within 32 months (Fig. 3) was associated with rapid deterioration of pulmonary function. In this case and all others, specific chemotherapy produced prompt conversion of sputum in both typical and atypical mycobacterial infections but did not halt the progression of the complicated silicosis. Figure 4 summarizes alterations of pulmonary function for the series. It was possible to obtain complete studies of 9 cases and spirometric studies in 15. The crosses represent the living cases; the squares are dead. The averages for all parameters of pulmonary function of the living were below 80% of predicted; in the fatal cases, the average reductions for all functions were severe. The percentage reduction of air flow rates was greater than for lung volumes, but both parameters of function fell together. The terminal picture was that of restriction of lung volume with reduction of total lung capacity, vital capacity, and pulmonary diffusing capacity associated with low air flow rates. In a larger series

TABLE 3
CHARACTERISTICS OF 22 SILICOTIC SHIPYARD SANDBLASTERS

Type of case	Number	Age at time of death/study: Average	Age at time of death/study: Range	Years of exposure: Average	Years of exposure: Range	Number with air supplied hoods	Number with massive disease	Number with mycobacterial infection
Fatal	11	48.5	28-68	9.7	3-20	5	10	9
Surviving	11	43.6	25-58	1.24	6-20	7	8	4

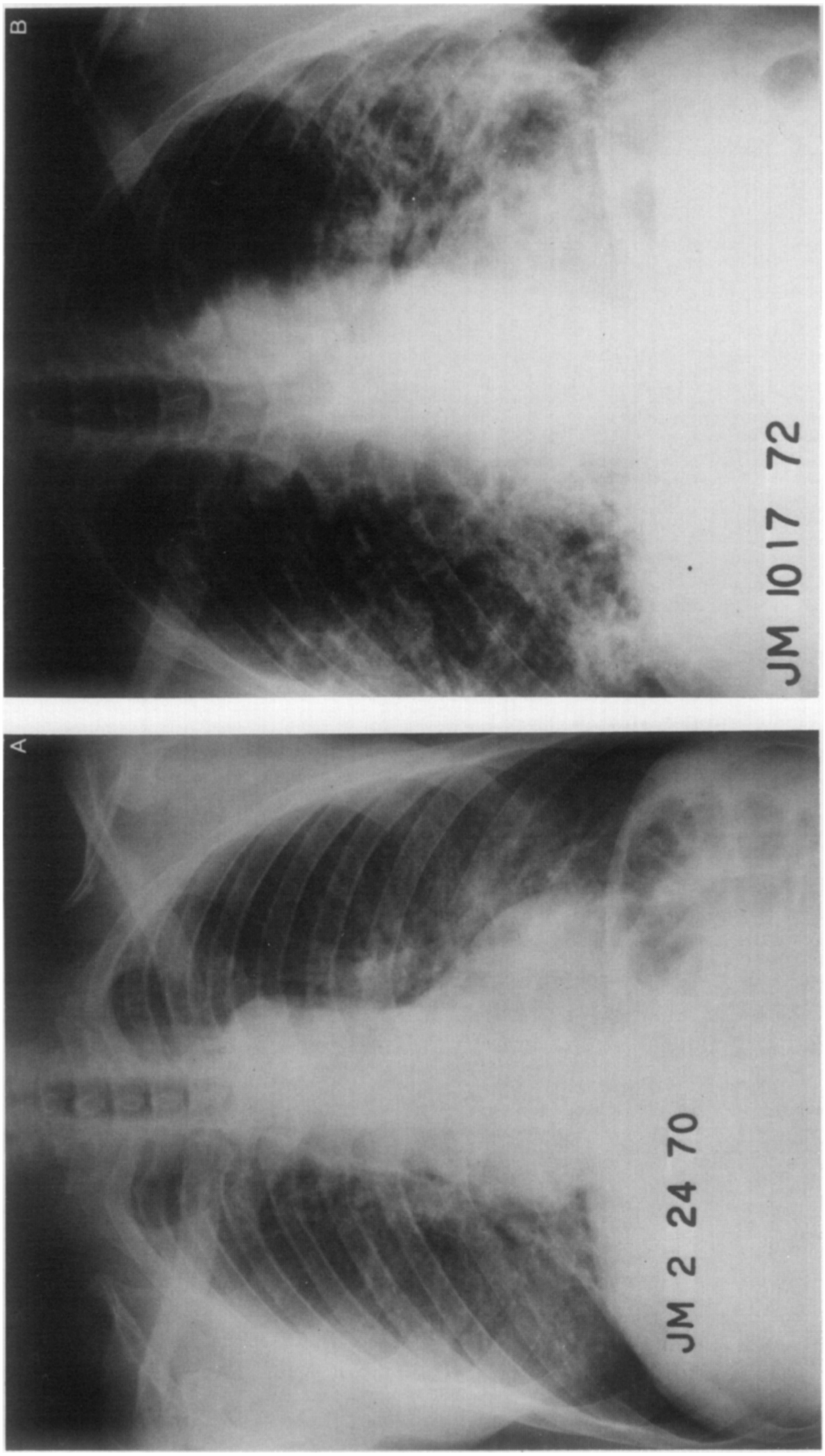

FIG. 3. Progression of disease in 32 months in silicosis complicated by (A) *M. kansasii* infection and (B) systemic lupus erythematosus.

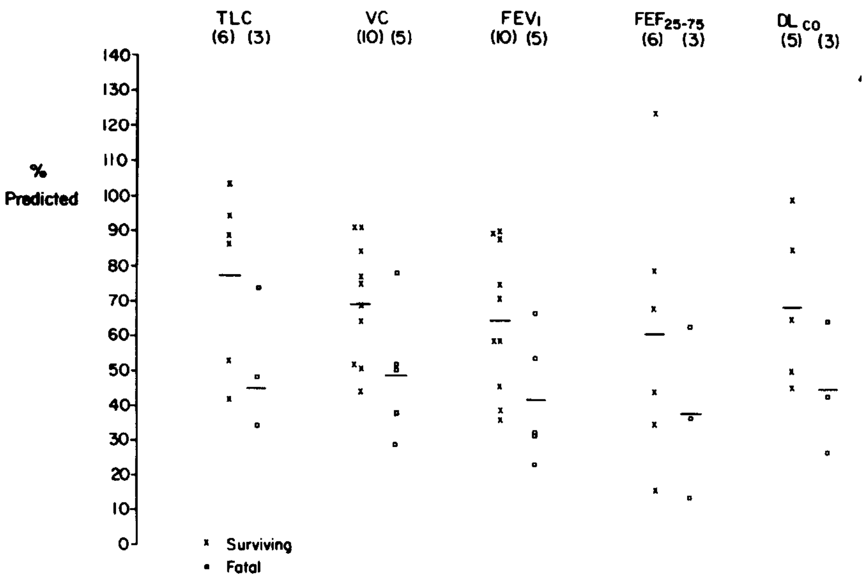

FIG. 4. Pulmonary function—surviving and fatal cases.

of 32 sandblasters, the progressively adverse effect of roentgenographic categories of increasing profusion and complication of disease upon pulmonary function was demonstrated (4).

Our results confirm the rapid course of fatal sandblaster's silicosis. The accelerated character of the disease appears to be the result of several factors, of which exposure to high concentrations of respirable silica with inadequate protection appear to be most important. Almost half of the workers did not have external air supply but even modern air-supplied hoods may not prevent a total daily exposure in excess of the calculated TLV. A second accelerating factor, the frequency of complicating mycobacterial disease and the high incidence of infection with opportunistic atypical mycobacteria, is consistent with previous experience with massive lesions in classical silicosis (5). In our larger series of 40 personally studied cases of silicosis, 20 have had sera analyzed for evidence of autoimmunity. In these workers, antinuclear antibody has been found in 40% of the cases (6) and the rheumatoid factor was found in the same percentage (7). This is a third factor promoting accelerated disease. In our larger series, two blasters have SLE, two have localized scleroderma, and two have rheumatoid arthritis.

The pathologic findings in sandblaster's silicosis are usually those of the more chronic disease with earlier onset of nodulation and complications. In the "acute" disease, which develops after less than 3 years of intense free silica exposure with faulty protection in enclosed spaces, nodulation may be obscured by rapid destruction of macrophages within alveoli, producing a pattern resembling alveolar proteinosis (8). This florid disease can produce fever and has benefited temporarily from corticoid therapy.

Information has been furnished which shows that exposure to respirable free silica was still excessive, although the worker wore a modern air-supplied hood during blasting intervals in an external environment. Respirable dust remains suspended after blasting and is dispersed from neighboring sources during the nonblasting intervals. Only prohibition of free silica as an abrasive can eliminate this hazard. There is increasing use of sand substitutes containing very low concentrations of free silica, but a number of shipyards continue to use sand because of its allegedly superior physical properties. Some of the operators state that their contractors require the use of sand in order to get a preferred surface before applying paint or coatings. Clearly, the exposure of workers in shipyards to free silica should be monitored and regulated. All cases of silicosis should be reported since they are the result of dangerous industrial operations that will lead to other cases of pneumoconiosis and to complication by tuberculosis, atypical mycobacteriosis, and a variety of opportunistic infections. Silicosis in sandblasters diminishes the working force and can lead to tuberculous contagion within the general community.

REFERENCES

1. Hunter, D. (1955). "The Diseases of Occupation," p. 864. Little, Brown, Boston.
2. Merewether, E. R. A. (1936). The risk of silicosis in sandblasters. *Tubercle* **17**, 385.
3. Bobear, J. B., Hanemann, S. J., and Beven, T. (1962). Silicosis in Louisiana: New or unrecognized industrial hazard. *J. Louisiana State Med. Soc.* 114, 391–398.
4. Ziskind, M., Weill, H., Bailey, W. C., Buechner, H. A., Brown, M., Waggenspack, C., and Samimi, B. (1973). Accelerated silicosis in sandblasters. (abs.), *Chest* **64**, 411.
5. Rosenzweig, D. Y. (1967). Silicosis complicated by atypical mycobacterial infection: Transactions of 26th VA-Armed Forces Pulmonary Disease Research Conference, p. 47. Washington, U. S. Government Printing Office.
6. M. Turner-Warwick. Personal communication.
7. O. Gum. Personal communication.
8. Buechner, H. A., and Ansari, A. (1969). Acute silico-proteinosis. *Dis. Chest* **55**, 274.