Causes of Death Among Employees of a Synthetic Abrasive Product Manufacturing Company

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Analysis of the causes of death among workers employed in the manufacture of synthetic abrasive products was performed using data from death certificates and employment records. The subjects were 1,030 white male workers who had been employed by a single manufacturer and who were identified through a review of death certificates issued during a 20-year period. Proportional mortality analysis revealed excess digestive cancer and respiratory disease deaths. Personnel records permitted refinement of the study population to 968 with classification of each individual into one of seven employment categories. Case-control analysis of workers ever employed in the individual categories revealed elevated odds ratios for respiratory disease deaths among those most exposed to the synthetic abrasive dust. The excesses were greatest in those exposed 20 or more years. Excesses of esophageal and rectal cancers were noted in two manufacturing areas although the numbers were small. Review of the literature further supports the finding that synthetic abrasive exposures may be associated with elevated respiratory morbidity and mortality.

The manufacture of synthetic abrasive materials has been commercially successful for more than 50 years. These materials have largely replaced natural abrasives (such as sandstone) because they have better properties as abrasives and because studies of short-term health effects have shown them to be less hazardous. However, there is little published information evaluating the effects of long-term exposure to the components of synthetic abrasives. A cause-of-death analysis of former employees of one large producer of synthetic abrasive products was undertaken to investigate possible health effects of this work process.

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Supported by Grants 5 T 15 OH-070-6 from the National Institute for Occupational Safety and Health and SSS 5 P30 ES0002 from the National Institute of Environmental Health Sciences

Methods

Study Group. — In Massachusetts death records are maintained by city and by year of death. Because the manufacturer of interest is a large employer in a small city, it was possible to select a study population directly from the death records. Records from the city where the manufacturer was located were examined for a 20-year period beginning with 1954. A record was chosen only if this employer's name was noted in the location marked "industry or business." Date of death, age at death, sex, usual occupation, underlying cause of death, name and ethnic origin of the individual were abstracted. Since few non-whites or females were found, this report will discuss white males only.

After results of an initial proportional mortality analysis were presented to the company, they granted access to their personnel records. A search of these records provided adequate work histories for most of the subjects. These files also contained records of subjects who had not been included in the original study group, but who had died between 1954 and 1973. They had not been identified in the search of state death records because they had died in other than the study city, or had died out of state, or because the company name did not appear on the death certificate.

Work Histories. — The plant manufactures abrasives, beginning with the synthetic abrasive grains, in the following production groups: (1) Abrasive Preparation — preparation of the synthetic abrasive grains, including crushing, washing, sizing, and grading; (2) Production — manufacture of aluminum oxide or silicon carbide abrasive wheels, using a clay bonding material (vitreous bonding) or an organic bonding material (organic bonding). The process involves mixing of grains and bonding material, wheel molding, kiln firing, truing (to assure parallel sides), grading (for hardness and soundness) and inspection. Production of synthetic diamond abrasive wheels using either an organic or a metal bonding material is done similarly but on a much smaller scale; (3) Refractories Process — manufacture of light and heavy refractory materials; and (4) Packing and Shipping — packing and shipping

areas are located in each production department but are substantially removed from the production area.

Each personnel record was abstracted without knowledge of cause of death, recording date of first employment and first job, date of entry into each subsequent job (up to a total of six jobs), total years worked in each job and year terminated. A job had to be worked for at least six months to be tabulated. Persons who worked more than 50% of the time in clerical or office jobs were considered clerical workers and excluded from most analyses.

The employer records of work history were excellent and reasonably detailed. The company has been under the same management for over 50 years and has kept records for that entire period. Records for those still working after 1945 showed specific job titles. The work history for persons who retired before 1945 was limited to major categories, generally departments. Since the number of subjects studied would not permit subdivision into more than five to ten groups, the limited data available before 1945 were considered detailed enough.

A walk-through survey of the entire plant provided the information necessary to identify 26 unique jobs which were grouped into seven "departments": (1) Abrasive Preparation; (2) Vitreous Bonding; (3) Organic Bonding; (4) Packing and Shipping; (5) Nonabrasive (includes Machine Tool Division, Small Machine Manufacture and Maintenance); (6) Service, and (7) Miscellaneous (includes Diamond and Refractories Abrasive Manufacture as well as other unclassifiable production jobs).

Analysis

Data were analyzed first in aggregate and then within each department by proportional mortality analysis (PMR) using the computer program of Monson[1] updated in 1978. This program uses cause-, time-, sex-, and race-specific data for the population of the United States from 1925 to 1975. In this analysis the 7th revision of the International Classification of Diseases[2] was used.

The proportional mortality analysis approach is recognized to have shortcomings. It examines proportions, not rates, of deaths so it does not examine the actual force of mortality. It also, as in this case, commonly employs the national mortality proportions as the standard and thus cannot consider regional environmental or socioeconomic differences.

In response to some of these shortcomings an odds ratio approach was also utilized in this investigation. With this approach the expected ratios are derived from the death experience of members of the same work force. Furthermore, a reasonable assumption can be made that, properly selected, the referent diseases have the same rates in the exposed group as in the unexposed group. To the extent that this is valid, the odds ratios are the same as rate ratios, which do indicate the relative force of mortality for the diseases under study.[3]

Those work areas showing unusual proportional mortality therefore were also examined by case-referent comparison using as comparison diseases all diseases except cancer and nonmalignant respiratory disease. Subjects and controls were grouped according to whether they had never or ever worked in the department under study. Those who had worked in the department were also stratified by duration of work. Odds ratios were calculated between the groups of never and ever exposed.

Age and ethnicity were specifically considered. With respect to age, odds ratios were adjusted using three age categories (younger than 65, 65 to 74, 75 years and older) and associations were estimated and tested by the Mantel-Haenzel method.[4]

Since Scandinavians, predominantly Swedes, make up 40% of the work population, an effort was made to determine whether this ethnic group was overrepresented in any disease category. A log linear model[5] was used to describe the data in terms of department of longest job, ethnicity, cause of death, and the interactions between these three variables.

Results

Death records for 1,030 males were identified in the state records for the years 1954 through 1973. Digestive cancer (five out of six sites), Hodgkin's disease and nonmalignant respiratory disease categories were notably elevated in a proportional mortality analysis.

Over 170 different occupation titles were included on the 1,030 death records. These were grouped and examined for group-specific excesses but none was found.

Expanded Study Group. — Job histories were available for 82% of the 1,030 decedents. Of these, 90 had worked as clerical or office workers and were excluded. Employment and death records were obtained for an additonal 229 employees who were identified in the personnel file as having died in Massachusetts but who had not been identified through the city's records of death. All had either died in other cities or had death certificates on which the company's name did not appear. Sixteen had worked as clerical or office workers and were excluded.

Since these two sets of deaths had been identified from different sources, PMR analyses of the two sets were compared. The differences noted were that decedents identified through company records had a deficit of nonmalignant respiratory disease (8 observed, 12.0 expected) and a younger mean age at death (60.3 vs. 69.6). PMR analysis of the clerical workers revealed a normal pattern. The one excess observed was for digestive cancer, but the ratio was not significant and the elevation was accounted for by an excess of liver cancer (2 observed, 0.47 expected). There was no excess of nonmalignant respiratory disease and no death due to pneumonia. The two groups, excluding clerical workers, were combined to provide 968 final study subjects.

Table 1 shows distribution by department of the final study subjects and the numbers who worked ten or more years. Of the decedents who had worked in departments engaged with abrasives, the largest group had worked in the Vitreous Bonding department.

Proportional mortality analyses for the final study group and for those who had worked in each of the three departments in which significant PMR excesses were seen (Abrasive Preparation, Vitreous Bonding, Organic Bonding) are shown in Tables 2 to 5. Since the PMR analysis uses the U.S. population as a standard, each of these department groups was also examined for selected diseases by the case referent method. The referents come from the

Table 1. — Number of Persons Ever Employed and Employed for Ten or More Years According to Department.*

	Ever Employed	Employed 10 or More Years
Abrasive preparation	155	98
Vitreous bonding	322	188
Organic bonding	140	56
Packing/shipping	117	59
Factory (nonabrasive)	495	332
Service	270	114
Miscellaneous	171	67

*Subjects are counted more than once if they worked in more than one department

same group as the cases thus avoiding the use of the U.S. population as the comparison group, as in the PMR. Since nonmalignant respiratory disease or malignancies occurred in excess in one or more of these departments, the referents selected for all three were those who died from causes other than nonmalignant respiratory disease (ICD/7 460-519) or malignancy (ICD/7 140-209).

Abrasive Preparation. — The authors were informed by a plant representative that the greatest dust exposures had occurred in the Abrasive Preparation department. Those who had worked in that department and who died with nonmalignant respiratory disease (ICD/7 460-519) as the underlying cause were compared with those who died from all other nonmalignant causes (Table 6). The odds ratio was found to be elevated and especially so for the group which had worked in Abrasive Preparation for 20 or more years. A trend in the ratio is apparent with increasing duration of employment in the department.

Cause of nonmalignant respiratory disease death for those in this department was predominantly pneumonia or bronchopneumonia (11 deaths). There were four who died with diseases of special interest. One died of pneumoconiosis, one of chronic interstitial pneumonitis, and two with silicosis (in one case as the underlying cause and in the other as an associated cause of death). If those who died of silicosis were excluded, the elevated odds ratio for those with 20 or more years experience was still significant.

Five of the 26 jobs identified were placed in the Abrasive Preparation category. These were crushing silicon carbide, crushing aluminum oxide, sizing abrasive grains, storing and inspection of grains and miscellaneous. The two workers with silicosis had worked for 28 and 29 years, respectively, in the crushing of silicon carbide. The silicon carbide was known to contain a low level of free silica but never more than 1%.

Vitreous Bonding. — Focus was next directed to those who had been employed in Vitreous Bonding and had died from nonmalignant respiratory disease or esophageal cancer (ICD/7 = 150). There is an elevated odds ratio for nonmalignant respiratory disease for those 17 subjects employed 20 or more years (OR = 1.7; $p < 0.1$). In contrast to the findings for Abrasive Preparation, no trend was apparent. Employment in specific jobs within this department did not distinguish those who died of nonmalignant respiratory disease from those who died of all other causes.

As in Abrasive Preparation, for most of those with nonmalignant respiratory disease pneumonia was the cause of death. There were 6 other causes of death of interest (3 with pneumoconiosis unspecified and 3 with silicosis). Those with pneumoconiosis unspecified had worked 17, 22 and 25 years, respectively, in those jobs which prepared the vitreous bonded wheels for firing in the kiln (mixing, molding, and shaving). One of those with silicosis had worked ten years firing the kilns. The second had

Table 2. — Proportional Mortality Analysis on Final Group.

	Observed	Expected	PMR	95% CI*
All infection	13	10.9	119	
All cancer	193	173.7	111	
Digestive organs	92	55.5	166	136 - 202
Esophagus	11	4.2	264	150 - 465
Stomach	19	12.0	158	101 - 246
Large intestine	27	16.8	160	111 - 232
Rectum	16	6.8	234	146 - 376
Liver	9	4.3	209	111 - 395
Pancreas	8	10.1	79	
Respiratory system	42	51.2	82	
Lymphopoietic and hematopoietic	21	16.0	131	
Lymphosarcoma and reticulosarcoma	3	3.9	77	
Hodgkin's	6	1.7	346	163 - 732
Leukemia and aleukemia	8	7.0	114	
All nervous system	83	95.3	87	
All cardiovascular	494	476.2	104	
All respiratory	73	56.7	129	103 - 161
Asthma	4	2.7	147	
Pneumonia	45	24.0	188	142 - 249
Emphysema	13	16.9	77	
All digestive system	37	40.7	91	
Cirrhosis	16	16.7	96	
All external	44	59.1	74	53 - 103
Accidents	31	41.9	74	
Residual	31	55.4		

*95% confidence intervals.[4] For brevity only those significantly elevated or depressed are listed on this table
n = 968 white males with work histories

Table 3. — Proportional Mortality Analysis on Those Ever Employed in Abrasive Preparation.

	Observed	Expected	PMR	95% CI*
All cancer	20	28.1	71	
Digestive organs	12	9.0	134	
Esophagus	2	0.7	291	
Stomach	3	1.9	156	
Large intestine	3	2.7	111	
Rectum	2	1.1	181	
Liver	1	0.7	143	
Pancreas	1	1.6	61	
Lymphopoietic and hematopoietic	0	2.6	0	
Lymphosarcoma and reticulosarcoma	0	0.6	0	
Hodgkin's	0	0.3	0	
Leukemia and aleukemia	0	1.1	0	
All cardiovascular	81	76.6	106	
All respiratory	20	9.4	212	139 - 324
Asthma	1	0.4	225	
Pneumonia	11	3.7	294	169 - 512
Emphysema	2	2.8	72	
Remainder	34	40.9		

*95% confidence intervals.[4] For brevity only those significantly elevated or depressed are listed on this table
n = 155 white males

worked 30 years firing, as well as 8 years in other jobs in this department. The third had worked only one year in this department but had worked 29 years in Abrasive Preparation.

The odds ratio analysis for esophageal cancer was elevated (OR = 2.5; $p < 0.1$). Two of the six who died of esophageal cancer and had been employed in this department had worked predominantly in mixing, molding, and shaving of vitreous wheels (22 and 36 years) and a third had worked one year at that location and 36 years at crushing and sizing of silicon carbide. The other three had worked predominantly at the kilns. Two of the three had worked exclusively at the vitreous wheel kilns (28 and 35 years, respectively) and one had worked eight years at those kilns and 22 years at the refractory material kilns (a separate department).

Organic Bonding. — Lastly, subjects were distributed according to employment in the Organic Bonding department and deaths due to nonmalignant respiratory disease or digestive cancer (ICD/7 = 150-159). There is an elevated odds ratio for the 17 who died with nonmalignant respiratory disease, which is significant (OR = 1.9; $p < 0.05$). No trend, however, was seen for increasing length of employment with 13 employed less than 10 years.

When digestive cancer as a whole was examined, an excess was noted for those 8 subjects employed 10 or more years (OR = 1.6; $p < 0.05$). The only specific type of digestive cancer appearing in more than two subjects was rectal cancer, with five cases. The odds ratio is significantly elevated for those four employed more than ten years (OR = 8.4; $p < 0.01$). Two of the five subjects who died of rectal cancer and had worked in this department had worked predominantly in mixing, molding, and shaving of organic bonded wheels (10 and 44 years, respectively); one had worked predominantly in truing and post-firing inspection and two had worked in unspecified jobs in this

Table 4. — Proportional Mortality Analysis on Those Ever Employed in Vitreous Bonding.

	Observed	Expected	PMR	95% CI*
All cancer	64	58.2	110	
Digestive organs	28	18,8	149	104 - 213
Esophagus	6	1.4	424	204 - 883
Stomach	6	4.1	148	
Large intestine	7	5.7	123	
Rectum	2	2.3	86	
Liver	2	1.5	137	
Pancreas	5	3.4	147	
Lymphopoietic and hematopoietic	8	5.1	1.6	
Lymphosarcoma and reticulosarcoma	1	1.3	80	
Hodgkin's	3	0.5	616	
Leukemia and aleukemia	2	2.3	88	
All cardiovascular	152	161.2	94	
All respiratory	30	20.4	147	102 - 207
Asthma	2	0.9	223	
Pneumonia	15	8.1	185	113 - 302
Emphysema	6	5.9	102	
Remainder	76	87.2		

*95% confidence intervals.[4] For brevity only those significantly elevated or depressed are listed on this table
n = 322 white males

Table 5. — Proportional Mortality Analysis on Those Ever Employed in Organic Bonding.				
	Observed	Expected	PMR	95% CI*
All cancer	35	25.1	140	104 - 188
Digestive organs	16	8.1	196	123 - 313
Esophagus	3	0.6	488	
Stomach	1	1.8	56	
Large intestine	3	2.4	124	
Rectum	5	1.0	494	225 - 1,086
Liver	2	0.7	309	
Pancreas	2	1.5	137	
Lymphopoietic and hematopoietic	8	2.3	353	185 - 671
Lymphosarcoma and reticulosarcoma	1	0.5	180	
Hodgkin's	4	0.2	1,710	
Leukemia and aleukemia	2	1.0	210	
All cardiovascular	59	69.8	85	
All respiratory	17	8.5	200	114 - 300
Asthma	2	0.4	460	
Pneumonia	9	3.5	259	139 - 481
Emphysema	5	2.4	207	
Remainder	29	36.6		

*95% confidence intervals.[4] For brevity only those significantly elevated or depressed are listed on this table
n = 140 white males

department. Four of the 968 subjects had rectal cancer as a secondary cause of death. All had worked exclusively in jobs involving nonabrasive materials.

The proportional mortality analysis (Table 2) indicated an excess of Hodgkin's disease. Four of the six with Hodgkin's had worked in Organic Bonding, three in the same job category (mixing and molding of the abrasive products) for 1, 29 and 33 years, respectively. The fourth had worked three years in the truing and inspection of the kiln-fired products. Three of these four had worked also in their same job categories in Vitreous Bonding. The two who had never worked in Organic Bonding had worked in maintenance or machining.

Ethnicity. — Since the incidence of certain diseases differs by ethnic group, the proportion of Scandinavians was examined by work group and cause of death. Jobs were grouped into the seven departments, ethnicity was grouped as Scandinavian or other and cause of death was grouped into digestive cancer, other cancer, nonmalignant respiratory disease and all other. Log linear analysis was performed to evaluate the significance of the interaction between cause of death and ethnicity. The interaction was found to be not significant ($p = 0.65$), which suggests that Scandinavians were represented proportionately within each of these major categories of death. The excess of nonmalignant respiratory disease or digestive cancer is unlikely to be explained by overrepresentation of Scandinavians.

Discussion

Natural abrasives for sharpening, grinding, and polishing have been used for over 3,500 years. These abrasives, mostly derived from siliceous rock, unfortunately have also been responsible for pulmonary disease, especially silicosis. At the end of the 19th century, the first commercial synthetic abrasive, silicon carbide, was made. Shortly afterward, aluminum oxide, which had been available naturally, was first prepared synthetically.

The greater hardness of these two synthetic abrasives compared to sandstone, as well as the belief that they were physiologically inert, led to their replacement of sandstone by the 1920s.

Review of the literature concerning health effects of silicon carbide and aluminum oxide indicates that only a minor effort has been made to evaluate long-term health effects of these two materials. The earliest report comes from a government study in Great Britain published in 1923.[7] Those performing metal grinding with abrasive wheels containing silicon carbide or aluminum oxide had much less pulmonary disease then those exposed to silica. However, there was "clinical" fibrosis in 18 subjects.

Investigations among aluminum producers and china

Table 6. — Case Comparison Analysis for Nonmalignant Respiratory Disease in Those Ever Employed in Abrasives Preparation.			
	Nonmalignant Respiratory Disease No.	Comparison Disease* No.	Age-Standardized Odds Ratios† (95% CI‡)
All ages			
Never worked	58	578	
Ever worked	20	115	1.9 (1.1 - 3.2)
< 10 years	5	47	1.2 (0.5 - 3.0)
10-19 years	4	24	1.6 (0.5 - 4.8)
≥ 20 years	11	44	2.7 (1.3 - 5.5)

*See text (p 8)
†Odds ratios are compared to never worked group
‡95% confidence intervals[4]

workers, both exposed to finely powdered alumina, showed no x-ray or clinical evidence of pneumoconiosis.[8 9 10] However, significant fibrogenic activity of relatively heavy exposures to gamma aluminum oxide has been reported from animal studies.[11 12]

In a study which examined workers exposed to silicon carbide during its production, early x-ray changes and mild symptoms, accompanied by a rather marked loss in vital capacity (mean of 520 mls over 6 years), were found in 32 workers.[13] Animal experiments with silicon carbide suggested it might promote tuberculosis but that it produced little, if any, fibrosis.[14 15]

A report in 1925[16] from the same plant examined in the current study summarized the experience of 137 persons exposed from 10 to 42 years. Forty-two showed no x-ray evidence of silicosis, 12 showed slight changes, 77 showed first-stage and 6 (four with no prior silica exposure) showed second-stage silicosis.

An earlier report from this same plant[16] included causes of death reported by the mutual benefit association between 1892 and 1924. Tuberculosis was not elevated as might have been expected if silicosis were prevalent. However, 20 out of 91 deaths were due to pneumonia, excluding epidemic influenza. A review of Massachusetts mortality records (between the years 1890 and 1925) for males 20 years or older showed that 9 to 12% of deaths were due to pneumonia. The proportion of deaths due to pneumonia reported for employees therefore is twice the expected number. The later report[15] updated the study of deaths and found that three of the seven subsequent deaths were due to pneumonia.

Another artificial abrasive plant[17] received aluminum oxide and silicon carbide to dry, crush, size and bag for shipment. Of the 52 employees who were x-rayed, 15 showed some pulmonary fibrosis, which in seven cases was classified as "the nodular type resembling silicosis." Five of the seven had had no other inorganic dust exposure. Dust exposures were extremely high (in processing, 12 of the 13 samples were over 50 mppcf, 7 were over 100 mppcf).

This review and results of the current study suggest that exposure to synthetic abrasives may be associated with adverse effects on the lung. Although cases of pneumoconiosis were reported in the current study, the predominant cause of death under All Respiratory Disease was pneumonia. This is not a very reliable death certificate diagnosis.[18] Review of the individual death certificates did not reveal any miscoding but pertinent clinical information which may have changed the underlying cause of death may not have been recorded. In addition, death assigned to pneumonia as the underlying cause may be over-reported in Massachusetts,[19] resulting in an elevated PMR compared to national proportions. Regardless, if the pneumonia excess were simply a matter of misclassification of cause of death, the excess odds ratio in Abrasive Preparation should not have shown a trend with increasing employment. Furthermore, the excess pneumonia reported in the literature for this company supports a possible work association.

No prior published report of cancer excess in a work population was found. The digestive cancer excesses identified in this study may have resulted from possible confounding factors.

Cigarette smoking and alcohol consumption are potential confounders for which almost no data were available. Smoking might contribute to pneumonia susceptibility and is a known risk factor for esophageal cancer. Alcohol intake is also a risk factor in esophageal cancer. No systematic information on smoking habits of the deceased is available. Cancer of the respiratory system, however, is not excessive, which suggests against differential smoking habits as an explanation of the excesses.

Mortality rates for esophageal and rectal cancer are 20% and 14% higher, respectively, than U.S. rates in the county where this manufacturing plant is located. This difference may account for some of the excess revealed in the PMR analysis. The use of the internal control in the case referent study, however, uses persons from the same area to estimate an expected proportion of deaths. Using cancer mortality experience in a city near the one under study to calculate an expected value similarly did not alter the findings of an excess.

In summary, the potential confounding factors considered do not appear sufficient alone to explain the excesses of observed cases of cancer of the esophagus and rectum. The excesses are based on small numbers and further comment is not indicated.

The excess of nonmalignant respiratory disease found in this study and the association of the excess with duration of employment suggests a work-related health effect on the lung. This is supported by the few reports on health effects in those exposed to similar synthetic abrasives. It should be noted that the excesses in this study population reflect exposures of many years ago when levels may have been less well controlled. Of particular concern, however, is the fact that these abrasives have many and varied uses throughout modern industry. Abrasive grinding and polishing, for example, are found in all metal machinery and foundry operations, in many parts of transportation equipment fabrication and in maintenance and mechanical repair occupations. Therefore, if present, such a risk could be widespread.

The cancer excesses noted are based on numbers too small to permit interpretation of their relationship to this work environment. The limitation of the proportional mortality analysis approach and the assumptions necessary to accept the odds ratio analysis have been noted. Further evaluation by respiratory morbidity studies, as well as mortality studies which expand the study population in time or size and use a standardized mortality analysis, should be informative.

We wish to thank Kim Schmidt Duir, Robbie Lauter, Lucille Pothier, Donna Spiegelman and Susan Sheridan for their assistance in carrying out this study.

References

1. Monson RR: Analysis of relative survival and proportional mortality. *Comput Biol Med* 7:325-332, 1974.

2. World Health Association Manual of the International Statistical Classification of Diseases, Injuries and Cause of Death, 1955, Revision, 1957.

3. Miettinen OS and Wang JD: An alternative to the PMR. *Am J Epidemiol* (in press).

4. Mantel N and Haenzel W: Statistical aspects of the analyses of data from retrospective studies of disease. *J Natl Cancer Inst* 22:719-748, 1959.

5. Bishop YMM, Fienberg SF, and Holland PW: Discrete Multivariate Analysis. Theory and Practice. Boston: MIT Press, 1975.

6. Cutler SJ and Young JL: Third National Cancer Survey: Incidence Data, Monograph 41, DHEW Publication No. (NIH) 75-787, March 1975.

7. Macklin EL and Middleton EL: Report of the Grinding Metals and Cleaning of Castings. Her Majesty's Stationery Office, 1923.

8. Sutherland CL, Meiklejohn A, and Price FNR: An inquiry into the health hazard of a group of workers exposed to alumina dust. *J Ind Hyg Toxicol* **19**:312-319, 1937.

9. Meikeljohn A and Jones WW: The effect of the use of calcined alumina in china biscuit placing on the health of the workmen. *J Ind Hyg Toxicol* **30**:160-165, 1948.

10. Meikeljohn A and Posner E: The effect of the use of calcined alumina in china biscuit placing on the health of the workman. *Br J Ind Med* **14**:229-231, 1957.

11. Klosterkotter W: Effects of ultramicroscopic gamma-aluminum oxide on rats and mice. *AMA Arch Ind Health* **21**:68-82, May, 1960.

12. Stacy BD, King EJ, Harrison CV, et al: Tissue changes in rats' lungs caused by hydroxides, oxides, and phosphates of aluminum and iron. *J Pathol Bacteriol* **77**:417-426, 1959.

13. Bruusgaard A: Pneumoconiosis in silicon carbide workers. Proceedings of the Ninth International Congress on Industrial Medicine, London 13-17, September 1948.

14. Gardner LV: Studies on the relation of mineral dusts to tuberculosis. III. The relatively early lesions on experimental pneumoconiosis produced by carborundum inhalation and their influence on pulmonary tuberculosis. *Am Rev Tuberculosis* **7**:344-357, 1923.

15. Studies on experimental pneumoconiosis. V: The reactivation of healing primary tubercles in the lung by the inhalation of quartz, granite, and carborundum dusts. *Am Rev Tuberculosis* **20**:833-875, 1929.

16. Clark WI: The dust hazard in the abrasive industry: Second study. *J Ind Hyg* **11**:92-96, 1929.

17. Clark WI and Simmons EB: The dust hazard in the abrasive industry. *J Ind Hyg* **7**:345-351, 1925.

18. Smith AR and Perina AE: Pneumoconiosis from synthetic abrasive materials. *Occup Med* **5**:396-402, 1948.

19. Engel LW, Strauchen JA, Chiezze L, and Heed M: Accuracy of death certification in an autopsied population with specific attention to malignant neoplasms and vascular diseases. *Am J Epidemiol* **111**:99-112, 1980.

20. Monson RR: Cause of Death in Boston. *J Chronic Dis* **33**:21-28, 1979.

Training Humane Physicians

If we are to train humane physicians, we must begin to address ourselves as a society to the basic general education toward ethical and moral values from infancy onward. For those who are comfortable with training that is rooted in traditional religious values, the solution is relatively easy, although all too often formalistic religion slights ethics. For the large proportion of Western society that is secular, a substitute for religion in ethical training is an urgent societal need. Much of the ethical behavior of even our secular societies is still routed in the religious heritage of a generation or two ago. As each generation moves further away from these traditional habits and mores, many of the basic axioms of societal morality are held up for critical inquiry and often rejected. We are too frequently left adrift without an accepted ethical consensus, and we witness in our youth what sociologists call alienation. Our societies must come to grips with this problem, because the problem transcends medicine; it threatens the very fabric of Western societal structure and its future.

— From "Sounding Board: Humanistic Medicine in a Modern Age," by S. M. Glick, M.D., in *The New England Journal of Medicine*, April 23, 1981.