AIRWAY OBSTRUCTION IN ASBESTOSIS STUDIED IN SHIPYARD WORKERS

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ABSTRACT

Airway obstruction was measured by spirometry in 296 boilermakers with shipyard exposure to asbestos for 15 or more years. Percent of predicted was used to adjust each worker's pulmonary function values for height (mean 174 cm.), age (mean 52.5 years) and cigarette smoking (mean 23.3 years). Mean values were significantly ($P<0.05$) below predicted for FVC 4.23 L 94.2%, FEV_1 3.06 L 89.3%, FEF_{25-75} 2.51 L/sec 82.3%, and FEF_{75-85} .574 L/sec 77.8%. Corresponding values for the 106 men with pulmonary asbestosis (ILO profusion of opacities 1/0 or greater) were below these levels. Correlation coefficients for pulmonary functions with ILO categories of asbestosis (profusion of irregular opacities) were: FVC −.2381, FEV_1 −.2494, FEF_{25-75} −.2403 and FEF_{75-85} −.1629. All were significant $P<0.05$. The subgroup with radiographic asbestosis (ILO 1/0 or greater) had more functional loss. Data on this large cohort of asbestos exposed workers establish that airway obstruction occurs with the slightest profusion of asbestosis scarring in the lungs of shipyard workers and progressively worsens with greater profusions of irregular opacities.

INTRODUCTION

The pattern of pulmonary functional impairment due to asbestosis, particularly the nature and severity of airway obstruction during the development of pulmonary fibrosis, is unclear despite many published studies.[1] Although small airways, the terminal and respiratory bronchioles, were identified as the locus of asbestos damage by Gloyne[2] half a century ago, in the past decade or two standard practice has been to attribute the airway obstruction observed in asbestos workers to cigarette smoking. In fact, the presence of airway obstruction, even in the presence of radiographic evidence of pulmonary asbestosis manifested by fine irregular opacities, has been considered as evidence against asbestosis.[3,4] In an earlier study of nonsmoking asbestos insulators we showed evidence for airway obstruction.[5] In contrast, active and retired shipyard workers[6] showed less convincing evidence for airway obstruction after adjusting for the effects of cigarette smoking. The relatively small group of nonsmokers showed little functional impairment, but they were older men who had less severe asbestosis. A subsequent review of the literature showed that too few nonsmokers with asbestosis had been studied to generalize about the effects, except in clinically advanced pulmonary asbestosis with its classic triad of decreased lung volume, vital capacity and diffusing capacity.[1] The age ranges were different in these studies and the shipyard study population contained older survivors. This study was designed to measure the degree of airway obstruction in asbestos exposed workers in ship construction, and repair and to relate it to the signs of asbestosis on chest radiographs.

METHODS

Measurements of pulmonary function were made in 296 male boilermakers who had been exposed to asbestos for more than 15 years. They were members of a local union of the International Brotherhood of Boilermakers, Iron Shipbuilders, Blacksmiths, Forgers and Helpers. The 296 men were or had been employed mostly in ship repair but some had built new ships as well. The presence and profusion of irregular pulmonary opacities of asbestosis was graded numerically using the ILO[7] criteria applied to posteroanterior radiographs taken at full inflation of the chest. Lateral chest radiographs were also obtained at full inflation to examine for dorsal pleural plaques and retrocardiac disease, and to measure lung volume.[8]

To express the severity of asbestosis in usable numbers for calculation, the ILO profusion categories were converted to integers as follows: 0/0=1, 0/1=2, 1/0=3, 1/1=4, 1/2=5, 2/1=6, 2/2=7, 2/3=8, 3/2=9, 3/3 and greater =10. These numbers were used as independent variables in regression equations to calculate correlation coefficients.

An occupational diary and exposure questionnaire was completed and questions answered with interviewer assistance on chronic bronchitis, wheezing, and shortness of breath

which were adopted from DLD-78.[9] The questionnaire inquired about pneumonia, respiratory illness, work loss and chest pain or heaviness and about workshift symptoms including feverishness, chills, thirst, taste, headaches, hoarseness or sore throat along with chest tightness, cough, phlegm and wheezing. Trained interviewers assisted the welders in answering questions and checked questionnaires for completeness.

Spirometry was done by recording sequentially forced expiratory flows on rolling seal spirometers until a pair agreed within 5%. Spirometers were calibrated repeatedly with a 3 liter syringe during the study. Spirometry was measured with subjects standing wearing a nose clip and following the American Thoracic Society Standardization.[10] The best effort was digitized and values computed for forced vital capacity, forced expiratory volume in 1 second, FEF_{25-75} and FEF_{75-85}. These values were compared to those from a stratified random population of Michigan, and comparisons were made smoking specific to current and ex-smokers of cigarettes and non-smokers.[11] This meant that a smoking duration (years) adjustment of $\underline{-0.0094}$ for FEV_1. Total lung capacity was measured $\underline{-0.0052}$ for log FEF_{25-75} from posteroanterior and lateral chest X-rays at full inflation and $\underline{-0.0112}$ for log FEF_{75-85} using the method of Harris et al.[8]

The individual pulmonary function values were summed and means calculated from individual data for comparisons of values and percentage of predicted. Data was entered into a Hewlett-Packard 9816 computer and standard statistical analyses and regression analysis done with the HP statistical library. A p value of 0.05 or less was considered statistically significant.

RESULTS

The mean age of the 296 southeastern shipyard workers was 52.5 years. They were 68.5 inches in height, had smoked an average of 23.3 years and averaged 23.6 cigarettes per day. They had 27.3 (mean) ± 11.6 (s.d.) years of exposure to asbestosis, Table I. Chronic bronchitis was diagnosed in 22.6% and 13.5 had a history of intermittent wheezing relieved spontaneously or by medications which was classified as asthma. They had reductions of FVC to 94.2% of predicted, FEV_1 to 89.3% of predicted, and FEF_{25-75} and FEF_{75-85} to 82.3% and 77.8% of predicted, respectively. All reductions were significant. The 106 men with asbestosis had lower FVC and flows than the entire group and these differences were all significant. Thus in the men with pulmonary asbestosis (ILO profusion 1/0 and greater) percent of predicted for vital capacity averaged 8% lower, for FEV_1 was 11% lower, midflow was 19% lower and terminal flow was 14% lower than in those without asbestosis. All differences were significant, $p<0.05$. There was a 12 year age difference from this division, but comparisons based on percentage predicted adjusted for the difference.

The correlation coefficients of pulmonary functions as dependent variables against ILO category score as the independent variable showed −.2381 for FVC, −.2494 for FEV_1, −.2403 for FEF_{25-75} and −1629 for FEF_{75-85} were all significant, Table II. The r^2 values showed that 2.65% to 6.22% of the variance in vital capacity and flows was accounted for by asbestosis. The regression lines with their confidence intervals show a continuum of change in each function from no asbestosis to severe asbestosis, ILO profusion 3/3, Figure 1 panels A-D. When the correlation coefficients were calculated for the 106 subjects with asbestosis, ILO profusion 1/0 and greater the respective coefficients were for FVC −.1919, for FEV_1 −2832, for FEF_{25-75} −.2918 and for FEF_{75-85} −.2483. Again all were significant. The two sets of regression equations for all shipyard workers and those with asbestosis produced similar estimates of reduction of vital capacity and of flows at $1/0^3$ and 2/1.[6]

DISCUSSION

By collecting spirometric measurements of pulmonary function by uniform protocol on 296 asbestos exposed shipyard workers and adjusting the data for height, age, ethnicity and cigarette smoking duration, we found that pulmonary parenchymal asbestosis is correlated with worsening airway obstruction. Increasing profusions of irregular opacities of asbestosis are associated with more obstruction. This observation was anticipated in 1933 by the original observation of the pathologic findings of peribronchiolar cuffing by Gloyne. However, the physiologic impairment of late or advanced disease has focused on restriction, i.e. reduced lung volume (TGV), FVC, and diffusing capacity. In contrast, the airway obstruction in workers with a high proportion of cigarette smokers has been attributed to smoking. By adding a standard adjustment for duration of smoking to the regression equations for FVC, FEV_1 and flows this confounding was removed.[11] Thus, the effect of asbestosis on FEV_1 and flows was clearly revealed.

This approach may lend itself equally well to analysis of the effect of other occupational and environmental exposures when the population lacks sufficient nonsmokers to make it possible to analyze effects in them alone. It appears to adjust effectively for the contribution of smoking. One reality which may interfere with such analyses is the age dependence and thus, concordance between age, years of smoking and years of exposure. By using a quantitative effect estimate of asbestosis based on the profusion of irregular opacities in the lung fields, the estimate of effect was independent of years and thus avoided this problem.

These findings establish an observational continuum between observations of airway obstruction especially of small airways in insulators who were heavily exposed to asbestosis but had not yet showed asbestosis on chest radiographs[5] and advanced asbestosis[6] in which FVC and diffusing capacity are reduced. It also avoids the survivor bias which was observed when elder men, most nonsmokers were studied earlier.[6,12] It appears that as increasing numbers of small airways are permanently obstructed FVC progressively decreases. As the process worsens, so many small airways are obstructed that the slowly ventilated space is lost and VC is small but quickly emptied so FEV_1 is restored together with flows. Diffusing capacity is closely tied to alveolar volume and remains so until it is critically reduced.

There may be a greater functional pulmonary impairment of shipyard workers compared to construction workers.[13] This was evident when the two segments of a midwestern

Table I

Comparison of Pulmonary Functions, Means and Standard Deviations for 296 Southern Shipyard Workers and the Subgroup with Asbestosis Compared as Percentage Predicted to the Michigan Male Population Sample

	ALL SHIPYARD	ASBESTOSIS	NO ASBESTOSIS
Number	296	106	190
Age - years	52.5 + 11.0	57.9 ± 8.2	49.5 ± 11.2
Ht - cm.	174.0 ± 7.1	173.7 ± 6.8	174.2 ± 7.4
Smoked - years	23.3 ± 16.7	27.7 ± 16.8	20.8 ± 16.1
Cig per day	23.6 ± 18.1	25.5 ± 16.7	22.6 ± 18.8
Asbestos - years	27.3 ± 11.6	31.3 ± 10.7	25.0 ± 11.5
Ch. Bronchitis	22.6	26.4	20.5
Asthma history	13.5	15.1	14.2
FVC L	4.23 ± .93	3.91 ± .90	4.42 ± .89
% pred	94.2 *	89.4 *+	96.9 *
FEV_1 L	3.06 ± .91	2.73 ± .82	3.25 ± .90
% pred	89.3 *	84.0 *+	92.3 *
FEF_{25-75} L/sec	2.51 ± 1.53	2.00 ± 1.23	2.79 ± 1.60
% pred	82.3 *	71.8 *+	88.2 *
FEF_{75-85} L/sec	.57 ± .46	.42 ± .34	.65 ± .50
% pred	77.8 *	71.0 *+	81.6 *
TGV L	7.69 ± 1.03	7.63 ± 1.04	7.72 ± 1.02
% pred	98.0	97.0	98.3

* Significant difference $p<0.05$ compared to predicted.
\+ Significant difference $p<0.05$ compared all shipyard workers.

Table II

Correlation Coefficients, R^2 and Regression Analysis for Pulmonary Function Against Category of Asbestosis (ILO) for 296 Southeastern Shipyard Workers and for the 106 with Asbestosis, ILO Category 1/0 or Greater

Percent Predicted	EXPOSED ASBESTOS		ASBESTOSIS	
Number	296	r^2	106	r^2
FVC	-.2381 *	.0567	-.1919 *	.0368
FEV_1	-.2494 *	.0622	-.2832 *	.0818
FEF_{25-75}	-.2403 *	.0577	-.2918 *	.0852
FEF_{75-85}	-.1629 *	.0265	-.2483 *	.0616

* $P<0.05$

REGRESSION EQUATIONS FOR ILO PROFUSION CATEGORY OF ASBESTOSIS AND PULMONARY FUNCTION AS PERCENT PREDICTED FOR 296 SOUTHEASTERN SHIPYARD WORKERS.

			ILO CAT 3/6
Percent pred. FVC	=	98.87 - 02.11 x ILO category	= 92.5/86.2
Percent pred. FEV_1	=	95.50 - 02.78 x ILO category	= 87.2/78.8
Percent pred. FEF_{25-75}	=	94.63 - 05.56 x ILO category	= 78.0/61.3
Percent pred. FEF_{75-85}	=	87.54 - 04.39 x ILO category	= 74.4/61.2

REGRESSION EQUATIONS FOR ILO PROFUSION CATEGORY OF ASBESTOSIS AND PULMONARY FUNCTION AS PERCENT PREDICTED FOR 106 SHIPYARD WORKERS WITH ASBESTOSIS.

			ILO CAT 3/6
Percent pred. FVC	=	96.27 - 01.64 x ILO category	= 91.4/86.4
Percent pred. FEV_1	=	96.35 - 02.98 x ILO category	= 87.4/78.5
Percent pred. FEF_{25-75}	=	96.02 - 05.83 x ILO category	= 78.5/61.0
Percent pred. FEF_{75-85}	=	95.95 - 06.01 x ILO category	= 77.9/60.0

population were compared to this southeastern group. The usual causes of differences including methodological ones have been eliminated. The ambient environments were urban and varied only slightly and all groups had equal exposure to welding. It is suggested that the greater impairment in shipyard workers reflects the hours of work in a container of limited volume into which are generated effluents of welding, insulating, metal grinding and polishing (still including sandblasting), painting, and other surface coating. These within ship and shipyard shops atmospheres appear richer and potentially more harmful to pulmonary function than is the outdoors or the interior of most buildings during construction.

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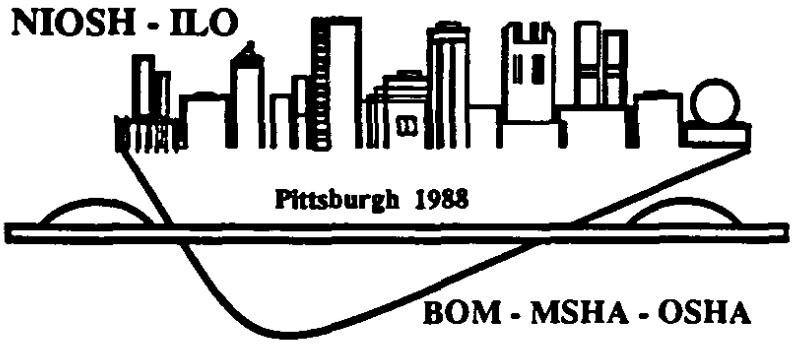

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