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ASBESTOS EXPOSURE, CIGARETTE SMOKING, AND PULMONARY FUNCTIONS

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ABSTRACT

A comprehensive health screening program consisting of standardized health and occupational history questionnaire, pulmonary function testing, chest x-ray, blood chemistry and urinalysis was organized for the Asbestos Workers Union Local No. 1 in St. Louis, Missouri. One hundred and thirty-five male caucasian workers or 44% of the membership volunteered to participate, and the pulmonary function testing data for all 135 workers were grouped according to their number of years of exposure to asbestos and their personal smoking habits. In general, smokers have significantly lower pulmonary functions than non-smokers and ex-smokers. When the number of years of exposure to asbestos is greater than 20, the pulmonary functions of non-smokers are significantly better than the ex-smokers. This study also shows that working in the asbestos industry can cause decreases in pulmonary functions regardless of whether or not a worker smokes.

INTRODUCTION

Asbestos is a general name for a large group of hydrated silicates that, when crushed or milled, separate into flexible fibers. The group is a continuous solid solution series of minerals that represents a small part of a larger mineral group of fibrous materials, the amphiboles. Since this class of minerals is a solid solution series, the chemical composition varies considerably from locality to locality and from one class member to another.

Asbestosis was recognized as a respiratory disease very early and led to the development of some of the first standards regulating dust levels in the workplace (Levin 1978). Asbestos in man involves diffuse interstitial fibrosis, calcification and fibrosis of the pleura, broncogenic carcinoma, and mesothelial tumors (Selikoff and Lee 1979). All amphiboles appear to be capable of initiating symptoms in man and the organization of the surface of the fibers, their lengths, and their diameters seems critical in the production of biologic effects (Becklake 1976, Churg and Warnok 1979).

Bronchogenic carcinoma and mesothelial tumors in man rarely occur less than 30 years after exposure to asbestos. Other environmental and personal habits exposing workers to other carcinogens or potential carcinogens further complicate the interpretation of the incidence of cancer following asbestos exposure. Smoking appears to enhance bronchogenic cancer and mesothelial tumor production (Selikoff et al. 1968, Burrows et al. 1977, Buist et al. 1979).

We determined to study the relationship between years of asbestos exposure and cigarette smoking on the pulmonary health of asbestos workers. To accomplish this, a comprehensive health screening program consisting of standardized health and occupational history questionnaire, blood chemistry profile, complete blood count, urinalysis, chest radiographs, and pulmonary function testing was organized for the Asbestos Workers Union Local No. 1 in St. Louis, Missouri. The program was made available on a voluntary basis to all members in the Union.

MATERIALS AND METHODS

During the evenings of the first week of June, 1981, 140 members of the Asbestos Workers Union Local No. 1, St. Louis, Missouri, volunteered to participate in a comprehensive health screening program. The other 170 members elected not to participate for personal reasons. According to Union records, these 170 individuals appeared to represent a cross-section of the Union similar to the 140 participants. They were fairly well distributed according to age, time span of asbestos exposure, and history of illness. On the surface, there appeared to be nothing remarkable in this group which would tend to skew the obtained study results.

The 140 participants consisted of 1 caucasian female, 4 black males, and 135 caucasian males. To assure a homogeneous study population, only the data for the 135 caucasian males were used in this study. Each participant was interviewed and completed a standardized health and occupational history questionnaire. All participants received the same battery of screening tests which included blood chemistry profile, complete blood count, urinalysis, chest x-ray, and pulmonary function testing.

Ventilatory function was assessed by recording the forced vital capacity using a Collins Eagle One Spirometry System, which was periodically and routinely checked for calibration. This unit, which meets all ATS-OSHA performance standards (Gardner et al. 1979), consists of a modified Stead Wells spirometer with kymograph and potentiometer, micro-processor, and a line printer. As the subject inhales and then exhales maximally, the resulting forced vital capacity curve is graphically displayed on the spirometer kymograph. The potentiometer signals are fed into the micro-processor, which calculates the flows and volumes, corrects them for BTPS (body temperature, pressure, saturated) conditions, and displays the test parameters on the digital screen. In addition, these parameters are printed out by the line printer.

The flow range of the Collins Eagle One Spirometry System is 0.0 to 20.0 L/sec, with an accuracy of 0.1 L/sec. The volume range is 0.0 to 9.99 L, with an accuracy of 0.025L. The parameters measured include the actual forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), the ratio of forced expiratory volume in one second to forced vital capacity (FEV_1/FVC), peak expiratory flow rate (PEFR), and forced expiratory flow during the mid-portion of the

FVC curve (FEF_{25-75}). Predicted normal equations based on age, height, sex, and race for adults (Knudson et al. 1976) are internally programmed. The predicted value and % predicted for the measured values are automatically generated and printed out at the completion of the study.

Each participant was carefully coached as to the proper method for performing an acceptable FVC curve, and the procedure was demonstrated (Cissik and Salustro 1977). Each participant then produced at least three acceptable FVC curves. Acceptability was defined and based on OSHA standards for cotton dust (USDOL 1978). Among others, these standards include continuing expiration for at least 6 seconds or until an obvious plateau in the volume-time curve occurs, and variation between the two largest FVC and FEV_1 measurements not exceeding 10% or 100 mL, whichever is larger. With each curve, a permanent alphanumeric record of the test results was printed out. The micro-processor also summarized the results and the summary results were used in the statistical analysis.

RESULTS

Data were collected from 135 male caucasian workers. Chemistry profile and urinalysis indicate that all test results were within normal limits. Pulmonary abnormalities were seen on 22 of the 135 chest radiographs. Eighteen of these, demonstrating such abnormalities as overinflation, tubular shadows, prominent linear markings, oligemia, and/or pulmonary hypertension, were consistent with a chronic airflow obstruction (Fraser and Pare 1979, Guenther and Welch 1977). The other four, showing differing extent and profusion of small opacities, pleural thickening, and pleural calcification, were consistent with varying grades of asbestosis according to the ILO U/C 1971 classification system (Fraser and Pare 1979). The remaining 113 chest radiographs demonstrated no significant pulmonary abnormalities.

The pulmonary function testing results for all 135 asbestos workers were separated into two subgroups based on their work history. Because Fraser and Pare have indicated that development of clinical manifestations requires a minimum of 20 years after onset of exposure to asbestos, we elected to look at our subjects in terms of those with less than 20 years of continuous work exposure (66 members) and those with more than 20 years of continuous work exposure (69 members). Within each group, the workers were further divided into three subgroups according to their smoking history, namely, non-smokers, ex-smokers, and smokers. A non-smoker was a person who had never smoked regularly. An ex-smoker was a person who had abstained from smoking for at least one year. A smoker was a person who had smoked regularly. The 66 workers with less than 20 years of continuous asbestos exposure consisted of 19 non-smokers, 11 ex-smokers, and 36 smokers, and among the 69 workers with more than 20 years of continuous work exposure: 17 non-smokers, 23 ex-smokers, and 29 smokers.

It was interesting to note that of the 22 workers with abnormal chest x-rays, there was only one non-smoker, but there were 10 ex-smokers and 11 smokers. The average period of time for ex-smoking was 10 years with the range being from 4 to 22 years. Analysis of the data from these 22 individuals showed this group to consist of middle-aged (50.68 $\pm$ 8.73 years) men with a mean work history in the asbestos industry of 29.37 $\pm$ 8.89 years.

The means and standard deviations of pulmonary functions of the 6 sub-

groups were calculated and are presented in Table 1. Table 2 shows the coefficient of correlation among pulmonary function measurements in the 135 workers. Significant correlation was observed between various pulmonary function parameters at the $p < 0.01$ level. Multiple-regression equations relating pulmonary function parameters to age, smoking habit, and years of asbestos exposure are given in Table 3 for all workers in the study, Table 4 for non-smokers, Table 5 for ex-smokers, and Table 6 for smokers. The correlation coefficients are all significant at the $p < 0.05$ level.

DISCUSSION

Asbestos, a natually occurring fibrous material, is an example of a material uniquely useful, because of its physical and chemical properties, and at the same time hazardous to man. Evidence linking inhalation of asbestos fiber to the development of a group of diseases is generating ever-growing concern. The demonstrated risks include the worker exposed in manufacture and use of asbestos, residents of neighborhoods of asbestos manufacturing plants, and the members of households of the asbestos worker.

In the group of workers with less than 20 years of asbestos exposure, the FVC, FEV_1, PEFR, and FEF_{25-75} were all within normal limits for the non-smokers and ex-smokers. Normal limit was defined as 80% or better of the predicted value. While the smokers in this group had normal FVC, FEV_1, and PEFR, their FEF_{25-75} had a mean value of 67.58% which was considered to have mild impairment. Mild impairment was defined as 65-79% of the predicted value.

In the group of workers with more than 20 years of asbestos exposure, the FVC, FEV_1, and PEFR were also within normal limits. The FEF_{25-75}, however, had a mean value of 68% for the non-smokers, indicating mild impairment, while the ex-smokers and smokers had mean values of 49.17% and 51.10% respectively. These workers were considered to have moderate impairment which was defined as 50-64% of the predicted value. In both groups, the smokers demonstrated lower forced mid-expiratory flow rates than the non-smokers and ex-smokers. However, with more than 20 years of asbestos exposure, the forced mid-expiratory flow rates were significantly much lower than the predicted normal values for all three sub-groups of workers. This seems to indicate that long-term asbestos exposure has a deleterious effect on lung function. The large change in the FEF_{25-75} would particularly suggest that asbestos not only causes a fibrotic condition but also can affect the small airways (Fraser and Pare 1979).

It has been suggested that with the cessation of cigarette smoking, ventilatory functions improved over a period of at least 6 to 8 months (Buist et al. 1979). Our data showed that while there were no significant differences in the ventilatory functions between the non-smokers and ex-smokers in the group with less than 20 years of asbestos exposure, the ex-smokers in the group with more than 20 years of asbestos exposure showed a statisitically significant difference in FVC, FEV_1, and FEF_{25-75}. It would appear that a significant improvement cannot be expected for a person who has worked for more than 20 years in the asbestos industry. It appears that cessation of cigarette smoking is particularly beneficial to those workers with less than 20 years of asbestos exposure. Recently, it has been suggested that smokers might even be better off than the ex-smokers who had damaged their lungs by long exposure to cigarette smoking, asbestos work, or a combination of

both (Corbin et al. 1979). The smoker exhales as a result of his smoking habit, while the ex-smoker no longer exhales as deeply. As a result, potentially harmful asbestos fiber is not exhaled by the damaged lung. The smoker in the process of smoking might exhale more of the asbestos fibers that penetrated into the lungs. If this hypothesis was correct, then deep breathing exercise performed regularly can certainly help the workers to eliminate some of the harmful asbestos fibers from the respiratory system.

CONCLUSIONS

In conclusion, our data show a significant difference in pulmonary function between smokers and non-smokers/ex-smokers in general, and ex-smokers do not have significantly better pulmonary function tests than do the smokers when both have more than 20 years of asbestos exposure. In all workers, the FEF_{25-75} ranges from the low end of the normal limit to moderate impairment depending on the smoking habit and the number of years of asbestos exposure for the individual. The pulmonary functions are significantly different when the older and younger non-smokers are compared, yet there are fewer functions that had significant differences when the older smokers, ex-smokers, and non-smokers are compared. Our data also show that working in the asbestos industry can damage the lung functions regardless of whether or not the worker smokes.

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Table 1. Means and Standard Deviations of Pulmonary Functions of 135 Male Asbestos Workers Based on Exposure and Smoking History.

Parameters	All Workers	20 Years of Exposure			20 Years of Exposure		
		Non-Smokers	Ex-Smokers	Smokers	Non-Smokers	Ex-Smokers	Smokers
Age (Years)	42.63	28.89	32.73	35.69	50.65	50.26	50.86
	12.44	6.90	9.36	10.49	7.77	6.22	6.80
Asbestos Exposure (years)	19.91	8.89	10.45	11.39	29.76	30.48	27.14
	10.83	5.52	5.89	5.03	6.73	6.32	6.32
Smoking (Packs/day-year)	21.77	0.00	11.82	20.81	0.00	32.57	45.59
	21.92	0.00	13.21	13.83	0.00	18.98	19.41
FVC (L)	4.70	5.53	5.55	4.87	4.25	4.17	4.35
	0.95	0.74	0.68	0.96	0.51	0.76	0.92
% Pred. FVC	98.93	108.32	109.18	99.47	94.82	92.87	95.45
	13.78	11.73	8.16	13.81	9.86	13.00	14.65
FEV_1 (L)	3.56	4.50	4.42	3.66	3.29	3.03	3.07
	0.85	0.62	0.74	0.80	0.42	0.47	0.74
% Pred FEV_1	93.10	107.42	108.64	92.42	92.47	85.65	84.97
	15.96	14.16	12.82	15.53	9.36	11.32	15.20
FEV_1/FVC (%)	75.67	81.53	81.09	75.14	77.76	73.26	71.74
	8.32	4.39	7.61	8.43	5.03	6.76	9.74
PEFR (L/sec)	8.45	10.23	8.47	8.02	8.10	8.64	7.87
	2.07	1.65	2.41	2.14	1.95	1.55	2.02

% Pred PEFR	92.65	108.11	89.09	86.14	91.88	97.65	88.45
	22.53	20.59	22.18	22.77	23.44	16.87	23.13
FEF_{25-75} (L/sec)	3.15	4.60	4.50	3.34	3.04	2.16	2.34
	1.43	0.95	1.46	1.35	1.00	0.87	1.25
% Pred FEF_{25-75}	65.91	89.84	89.91	67.58	68.00	49.17	51.10
	27.41	20.16	27.69	25.84	20.54	20.45	24.05

FVC: forced vital capacity
% Pred. FVC: % predicted forced vital capacity
FEV_1: forced expiratory volume in 1 second
% Pred. FEV_1: % predicted forced expiratory volume in 1 second
PEFR: peak expiratory flow rate
% Pred. PEFR: % predicted peak expiratory flow rate
FEF_{25-75}: mid-portion forced expiratory flow

These abbreviations will be used in the subsequent tables.

Table 2. Coefficients of Correlation Among Pulmonary Function Measurements in 135 Male Asbestos Workers.

	Age (years)	Exposure (years)	Smoking (P/day-yr)	FVC (L)	FEV_1 (L)	FEV_1/FVC (%)	PEFR (L/sec)	FEF_{25-75} (L/sec)
Age (years)	1.000	0.866	0.529	−0.680	−0.733	−0.324	0.299	−0.620
Exposure (years)		1.000	0.401	−0.615	−0.643	−0.222	−0.178	−0.542
Smoking (Packs/day-year)			1.000	−0.336	−0.487	−0.444	−0.250	−0.537
FVC (L)				1.000	0.885	0.067	0.376	0.534
FEV_1 (L)					1.000	0.515	0.487	0.829
FEV_1/FVC (%)						1.000	0.350	0.802
PEFR (L/sec)							1.000	0.354
FEF_{25-75} (L/sec)								1.000

Table 3. Multiple Regression Equations Relating Pulmonary Function Parameters to Age, Smoking Habit, and Asbestos Exposure for all Asbestos Workers.

Parameter	Equation	Correlation Coefficient	Std Error of the Estimate
FVC (L)	$y = -0.046A + 0.001P - 0.009W + 6.822$	0.682	0.702
FEV_1 (L)	$y = 0.041A + 0.006P - 0.005W + 5.542$	0.743	0.578
FEV_1/FVC (%)	$y = -0.171A - 0.139P + 0.113W + 83.756$	0.462	7.461
PEFR (L/sec)	$y = -0.084A - 0.010P + 0.057W + 11.092$	0.350	1.963
FEF_{25-75} (L/sec)	$y = -0.046A - 0.019P - 0.011W + 5.735$	0.663	1.096

A: Age of worker in years
P: Smoking habit in packs/day-year
W: Asbestos exposure in years

Table 4. Multiple Regression Equations Relating Pulmonary Function Parameters to Age and Asbestos Exposure for Non-smokers.

Parameter	Equation	Correlation Coefficient	Std Error of the Estimate
FVC (L)	$y = 0.006A - 0.067W + 5.937$	0.816	0.539
FEV_1 (L)	$y = 0.008A - 0.064W + 4.817$	0.838	0.455
FEV_1/FVC (%)	$y = 0.030A - 0.173W + 81.842$	0.348	4.837
PEFR (L/sec)	$y = 0.047A - 0.128W + 9.788$	0.478	1.876
FEF_{25-75} (L/sec)	$y = 0.028A - 0.097W + 4.595$	0.697	0.940

A: Age of worker in years
W: Asbestos exposure in years

Table 5. Multiple Regression Equations Relating Pulmonary Function Parameters to Age, Smoking Habit, and Asbestos Exposure for Ex-smokers.

Parameter	Equation	Correlation Coefficient	Std Error of the Estimate
FVC (L)	$y = -0.069A + 0.005P + 0.005W + 7.566$	0.772	0.629
FEV_1 (L)	$y = -0.035A - 0.005P - 0.022W + 5.748$	0.826	0.511
FEV_1/FVC (%)	$y = -0.352A - 0.164P + 0.082W + 74.579$	0.517	7.060
PEFR (L/sec)	$y = -0.105A - 0.014P + 0.082W + 11.152$	0.265	1.859
FEF_{25-75} (L/sec)	$y = -0.016A - 0.024P + 0.082W + 4.759$	0.705	1.149

A: Age of worker in years
P: Smoking habit in packs/day-year
W: Asbestos exposure in years

Table 6. Multiple Regression Equations Relating Pulmonary Function Parameters to Age, Smoking Habit, and Asbestos Exposure to Smokers.

Parameter	Equation	Correlation Coefficient	Std Error of the Estimate
FVC (L)	$y = -0.053A + 0.008P - 0.008W + 6.776$	0.592	0.801
FEV_1 (L)	$y = -0.052A - 0.002P - 0.005W + 5.471$	0.673	0.623
FEV_1/FVC (%)	$y = -0.310A - 0.109P + 0.313W + 84.229$	0.398	8.633
PEFR (L/sec)	$y = -0.091A - 0.008P + 0.077W + 10.624$	0.366	1.976
FEF_{25-75} (/sec)	$y = -0.072A - 0.013P + 0.026W + 5.878$	0.625	1.113

A: Age of worker in years
P: Smoking habit in packs/day-year
W: Asbestos exposure in years