PULMONARY FUNCTIONAL IMPAIRMENT FROM YEARS OF ARC WELDING

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ABSTRACT

Construction welders without shipyard exposure had vital capacity and flows measured by spirometry, chest radiographs classified for asbestosis using ILO criteria and were administered an occupational and respiratory questionnaire. Data on workers with asbestosis (ILO profusion 1/0 or greater) was deleted from analysis. The 226 men without asbestosis were 45 years old, 69.2 inches tall and had welded for 21.3 years (means). Expressed as mean percent predicted after adjusting for height, age and years of smoking mean FVC was 101%, FEV_1 was 98.7%, FEF_{25-75} was 94.0%, FEF_{75-85} was 91.8% and TGV was 104.2%. The 43 nonsmokers had similar reductions in flows. The regression coefficient for years of welding for FVC was −.0031, for FEV_1 was −.0035, for midflow (FEF_{25-75}) was −.0080, all significant ($p < 0.05$), and for FEF_{75-85} was −.0056 not significant. Calculated from regression equations 40 years of welding would reduce FVC to 95.2%, FEV_1 to 92.2%, midflow to 79.2% and flow at low lung volume to 81.3%. Chronic exposure to welding gases and fumes reduces flows in small airways in welders without asbestosis or shipyard exposures.

INTRODUCTION

Metal welding generates ozone, nitrogen oxides and metal oxide aerosols which cause respiratory tract irritation, cough and excessive phlegm and impair pulmonary function in welders.[1] Arc welders in shipyards have reductions in vital capacity and FEV_1[2-6] which have been considered permanent. Because many studies were done when 80 to 90% of the welders were also cigarette smokers, the degree of interdependence of effects of welding exposure and smoking is unclear. It follows that the chronic functional impairment in those welders who had never smoked is also poorly defined. Also earlier studies ignored the effects of asbestosis on pulmonary function.[7,8] Thus, this study of 226 welders, 43 of whom had never smoked, explored the chronic effects of welding on pulmonary function and interactions with cigarette smoking. We examined 226 welders, employed largely at construction sites and power plants, who had never worked in shipyards and had no evidence of asbestosis on chest radiographs. Pulmonary functions were expressed as percentage of predicted based on individual comparisons corrected for height, age and for cigarette smoking[9] to assess the contribution to impairment of years of welding.

METHODS

226 male welders from two midwestern locals of the International Brotherhood of Boilermakers, Iron Shipbuilders, Blacksmiths, Forgers and Helpers were studied during 1987 for asbestosis by questionnaires for occupational exposures and respiratory disease and for symptoms, a chest physical examination, posteroanterior and lateral chest radiographs and pulmonary function measurements. Informed consent was obtained after the nature of all procedures was fully explained to each welder. The questionnaire was adapted from DLD-78[10] as used in the study of the Michigan population[11] and inquired about chronic bronchitis, asthma, wheezing and shortness of breath as well as past respiratory and cardiovascular disease symptoms and illnesses. It was administered by trained interviewers. The mobility of welders doing construction jobs, variation in composition of base metal and rod, type of welding, and the uniqueness of each site for their lifetime made exposure measurement impossible. As an exposure surrogate, years of welding was used in analyses.

Spirometry was measured on Saturday or Sunday, at least 16 hours after a workshift on Ohio Rolling Seal spirometers which were repeatedly calibrated during the study with a 3 L syringe. Spirometry was repeated until 2 agreed within 5% with subjects standing, wearing a noseclip, and followed American Thoracic Society[12] criteria including that the origin of FEV, was established by back extrapolation. The best curve was digitized and FVC, FEV_1, FEF_{25-75}, and FEF_{75-85} and for each individual value as percentage predicted was adjusted for height, age and years of cigarette smoking. Values were compared to the Michigan male population sample in smoking specific groups.[9] Posteroanterior and lateral chest radiographs were made at full inflation. They were examined for evidence of asbestosis of lung or pleura using ILO classification criteria.[13] The 65 welders with signs of asbestosis of the 291 originally studied

were not analyzed further.

Statistical testing was performed in a Hewlett-Packard 9816 computer using the Hewlett-Packard library of statistical programs including calculation of confidence intervals, regression analysis and equation development. Analysis of variance using an SAS statistical program in an IBM computer was used to compare function values for caucasian male welders to the entire sample of Michigan men, not just those selected as normals for modelling pulmonary function.[9] What this means is that we restored to the group used for modelling "normal" predictive pulmonary function all subjects who had been excluded because of clinical abnormalities. Thus, the comparison group included all adult males studied in Michigan. A P value of < 0.05 was used to demonstrate significance.

RESULTS

The 226 electric arc welders were 45.0 years old, and 175.8 cm. tall (mean values) and they had welded an average of 21.3 years, Table I. Only 19.0% had never smoked cigarettes. Nearly 20% had chronic bronchitis diagnosed by phlegm production for 3 months per year for at least two years and 11.3% had a history of asthma. Nonsmokers prevalence of chronic bronchitis was 23% and of asthma was 13%; thus they were as symptomatic as smokers for these disorders. Pulmonary functions for the 226 men are presented as group means and percentages of predicted, adjusted for effects of height, age and for years of cigarette smoking,[9] Table I. There were no years of smoking adjustment for FVC, for FEV_1 it was –0.0094 for years of smoking for log FEF_{25-75} it was –0.0052 years of smoking and for log FEF_{75-85} it was 0.0112 years of smoking. The mean vital capacity of the group was 101.0%, FEV_1 was 98.7%, FEF_{25-75} was 94.0%, FEF_{75-85} was 91.8% and TGV was 104.2%. Although nonsmokers had slightly better function than the whole group, after adjustment for smoking only the difference in FEF_{75-85} was statistically significant, Table I.

To test for chronic effects of welding the critical analysis was to correlate years of welding, as the independent variable with the percentage of predicted values for FVC, FEV_1, FEF_{25-75}, and FEF_{75-85}, as dependent variables by regression analysis, Table II. This was done after each individual observation of pulmonary function was adjusted for height, age and for years of cigarette smoking. For the 226 welders the regression coefficients for years welding were –0.0031 for FVC, Figure 1a; –0.0035 for FEV_1, Figure 1b; –0.0080 for FEF_{25-75}, Figure 1c; and –.0058 for FEF_{75-85}, Figure 1d. All were significant ($P < 0.05$) except for FEF_{75-85}. Calculating the effects of 40 years of welding on percent predicted using the regression equations for all welders reduced FVC to 95.2%, FEV_1 to 92.2%, FEF_{25-75} to 79.2% and FEF_{75-85} to 81.3%, Table II. The 43 nonsmokers had larger regression coefficients for years of welding on mid and terminal flows. Thus for FEF_{25-75} the regression coefficient was –.0095 and it was –.0171 for FEF_{75-85}. Both were significant ($P < 0.05$). The smaller coefficients for FVC of –.0013 and for FEV_1 of –.0036 were not significant. (Figure 2) Thus, in nonsmokers the significant effects of duration of welding were limited to small airways. Perhaps the implications of these relationships for subjects who weld but have never smoked are best shown by calculation of the reduction in function expected from 40 years of welding using regression equations. Thus without exposure to cigarette smoke 40 years of welding would not reduce vital capacity but FEV_1 would fall to 93.3% of predicted. Midflow would fall to 77.6% of predicted and terminal flow would drop to 62.0%.

DISCUSSION

Chronic exposure to arc welding gases and fumes reduced flows including FEV_1. Such airway obstruction moderately impairs function of welders after 20 to 40 years. Cigarette smoking welders showed additional adverse effects which exceed standard adjustments for duration of smoking.[9] Thus, welders who smoked showed more than the sum of functional impairments from welding and from cigarette smoking. Initially, welding fumes and gases reduce flows in small airways as seen unequivocally in the 43 nonsmokers. When cigarette smoking is added vital capacity and FEV_1 are also reduced to an extent beyond the standard adjustment for smoking. If the effects of the cigarette smoke and welding smoke aerosols were equal and additive then 40 years of welding alone would resemble 20 years of welding in cigarette smokers. That the 40 year calculated effect of welding alone is to reduce flows including FEV_1 but not to reduce FVC suggests that the effects of welding and cigarette smoking are similar. Predictive regression equations for pulmonary function in these welders account stepwise for height, age and duration of cigarette smoking (years) by multiple linear regression, and isolate the effect of welding exposure. Horizontal lines would reflect decrements from occupational exposure to welding. These men all had some exposure to asbestos in insulating materials, gloves and blankets. Excluding those with signs of pulmonary asbestosis is a major step in removing its effects and goes beyond earlier studies of welders[2-6] but does not guarantee the absence of subtle effects.[7]

The additional decrement which occurred in smokers appears to be best explained by the synergism between the effects of particles, gases and chemicals adsorbed on particles in two complex aerosols.[14] One, cigarette smoke, is an exceedingly complex mixture of tobacco distillates and combustion products characterized as particles of complex hydrocarbons or tar containing over 2,500 chemical species and a mixture of gases including carbon monoxide, nitrogen oxides and aldehydes.[15] Welding fumes are oxides of metals with additives from flux, rod coating and surface treatments and gases, largely ozone and nitrogen oxides.[16] The particles from both cigarette combustion[17] and the welding arc[18] are poorly digestible by pulmonary macrophages, damage lining cells of distal airways and connective tissue during phagocytosis and disposal.[18] Thus, cigarette smoke, and welding of asbestos produce goblet cell metaplasia and mucous obstruction[19] in distal terminal bronchioles.[7] This is followed by peribronchiolar cuffs of cells and fibroblast proliferation, collagen production, and scarring with luminal narrowing and obstruction of terminal and respiratory bronchioles.[19,20,21] Functional loss of small airways by anatomic obstruction and obliteration removes respiratory units from ventilation and eventually reduces vital capacity. Although one might speculate that the welding particles would also stimulate digestion of lung at the alveolar level to destroy alveolar walls

Table I

Pulmonary Functions, Means (m), Standard Deviations (sd), and Percentage Predicted in 226 Midwestern Welders without Evidence of Asbestosis

	ALL WELDERS m	sd	NONSMOKING WELDERS m	sd
Number	226		43	
Age - years	45.0	± 10.4	44.9	± 10.7
Ht - cm.	175.8	± 6.5	175.5	± 8.9
Welding years	21.3	± 10.0	20.5	± 10.4
Smoking years	21.3	± 13.9	0	
Cig/day	25.1	± 18.9	0	
Asbestos exp. yrs.	22.6	± 10.8	19.9	± 9.8
Ch. Bronchitis %	19.9		23.3	
Asthma history %	11.3		13.2	
FVC L.	4.93	± .87	5.03	± .89
% pred.	101.0	± 14.6	103.9	± 15.6
FEV_1 L.	3.77	± .82	3.98	± .79
% pred.	98.7	± 16.6	100.4	± 15.8
FEF_{25-75} L/sec.	3.20	± 1.37	3.58	± 1.28
% pred.	94.0	± 34.3	96.2	± 28.3
FEF_{75-85} L/sec.	0.84	± .52	1.07	± .61 *
% pred.	91.8	± 46.7	95.6	± 45.3
TGV	7.60	± 1.05	7.19	± 1.10
% pred.	104.2	± 14.8	101.1	± 15.9

* $P < 0.05$

Table II

Correlations, R^2 and Coefficients from Regression Analysis of Effects of Years of Welding on Pulmonary Function as Percent Predicted of 226 Midwestern Construction Welders

Percent Predicted	ALL WELDERS Correlation	r^2	Regression Coefficient		NEVER SMOKED Correlations	r^2	Regression Coefficient	
Number	226				43			
FVC	-.2096	.0439	-.0031	*	-.0841	.0071	-.0013	
FEV_1	-.2114	.0447	-.0035	*	-.2351	.0553	-.0036	
FEF_{25-75}	-.2326	.0541	-.0080	*	-.3479	.1211	-.0095	*
FEF_{75-85}	-.1182	.0140	-.0056		-.3929	.1544	-.0171	*

* P<0.05

Percent pred. FVC = 107.50 - .0031 x (40) years welding = 95.2

Percent pred. FEV_1 = 106.25 - .0035 x (40) years welding = 92.2

Percent pred. FEF_{25-75} = 111.20 - .0080 x (40) years welding = 79.2

Percent pred. FEF_{75-85} = 103.68 - .0056 x (40) years welding = 81.3

Figure 1a–1d. Regression equations for all welders and effect of 40 years of welding on percentage predicted.

Percent pred. FVC = 106.44 - .0013 x (40) years welding = 101.4

Percent pred. FEV_1 = 107.74 - .0036 x (40) years welding = 93.3

Percent pred. FEF_{25-75} = 115.57 - .0095 x (40) years welding = 77.6

Percent pred. FEF_{75-85} = 130.63 - .0171 x (40) years welding = 62.0

Figure 2. Regression equations for non-smoking welders and effect of 40 years of welding on percentage predicted.

and increase total lung capacity as does cigarette smoke, the welders have no significant increase in total lung capacity after adjusting for the effects of cigarette smoking.[22]

Earlier studies of the effects of welding were of cigarette smoking shipyard welders who are frequently exposed, in addition to asbestos in insulating materials, to silica from sand blasting, paint fumes, metal particles, and other toxins in closed spaces of hulls or compartments. In shipyard welders Hunnicutt in 1968[2] anticipated the smoking-welders synergism by showing that smokers but not nonsmokers had reduced peak flow, FEV_1 and FEF_{25-75}. In another study the shipyard effect was amply underscored by finding that 61 shipyard welders and 63 shipyard pipefitters had reduced FVC, FEV_1, TLC, RV and DLCO compared to "new pipefittters" and to current standard populations.[3] In engineering shop welders in Finland only diffusing capacity was reduced,[4] again emphasizing the shipyard effect. After lumping ex-smokers and nonsmokers, a Swedish shipyard study showed welders had significantly lower FEV_1, FVC and TLC than nonwelders.[5] A Newcastle, Great Britain shipyard study of 209 welders and 109 controls matched 2:1 for age, height, smoking habits, residence and social class showed that in both smokers and nonsmokers FVC and FEV_1 were significantly lower than in controls and DLCO was significantly lower in nonsmokers.[6] The most comparable study to this one found significant reductions of FVC, FEV_1, FEF_{25-85} and DLCO in 72 nonsmoking shipyard welders.[23]

This study has eliminated confounding exposures from work in shipyards,[8] ruled out asbestosis recognized by irregular opacities on chest radiographs and adjusted for the expected effects of years of cigarette smoking. Thus, for the first time arc welding gases and fumes can confidently be regarded as causing airway obstruction. However, the magnitude of decrements is smaller than that from cigarette smoke alone[9] or from asbestosis.[8] Welding fumes and gases, especially particles to which are adsorbed cytotoxic molecules, appear to elicit cellular and tissue reactions which narrow and distort small airways. Studies of lung pathology in welders or experimental studies of chronic exposure are needed to elicit the mechanisms.

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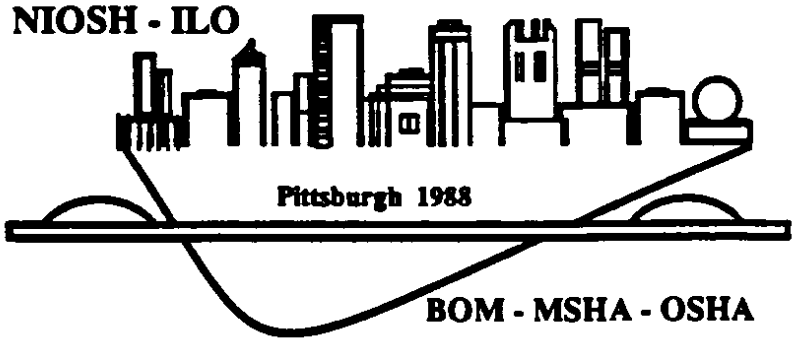
II



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