Reprinted from the Archives of Environmental Health
September 1963, Vol. 7, pp. 337-340

Pulmonary Function in Welders Exposed to Ozone

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Introduction

The American Conference of Governmental Industrial Hygienists recommend a maximum permissible concentration of ozone of 0.1 ppm in industrial plants.[1] We recently demonstrated that the passengers and crews of commercial jet airliners are exposed to mean concentrations of 0.3 to 0.4 ppm during flight.[2] It is therefore important to determine if any impairment of pulmonary function can be demonstrated in subjects repeatedly exposed to similar concentrations of ozone over a period of some years.

Kleinfeld et al described three cases of severe pneumonia in men who had been exposed to a concentration of approximately 9 ppm of ozone for periods of 3 to 14 days while engaged in argon-shielded electric arc welding.[3,4] Other welders chronically exposed to 0.3 to 0.8 ppm had a high incidence of complaints of pharyngeal irritation and chest constriction, while those exposed to concentrations below 0.3 ppm had no symptoms.[4] No studies of pulmonary function were carried out.

Griswold et al demonstrated a decrease in vital capacity and timed capacity following an experimental exposure to 2.0 ppm of ozone for two hours.[5] Clamman and Bancroft reported a fall in the pulmonary carbon monoxide diffusing capacity following exposures to 1.2 to 6.0 ppm for one to two hours.[6] Bennett has recently reported a fall in one-second forced expiratory volume ($FEV_{1.0}$) in a group of six subjects exposed to 0.5 ppm of ozone, three hours per day, six days per week for 12 weeks.[7] A similar exposure to 0.2 ppm of ozone produced no change. Challen[8] has reported symptoms of upper respiratory tract irritation in 11 of 14 welders, on exposure to ozone concentrations of 0.8 to 1.7 ppm. These symptoms no longer occurred when the concentrations of ozone were reduced to approximately 0.2 ppm.

Charr has described three welders with progressive respiratory illness of several years' duration.[9] Lung biopsy was performed in two of these cases, of which one revealed a necrotizing bronchiolitis, epithelialization of alveolar walls, extensive fibrosis, and infiltration with neutrophils and macrophages. These pathological findings are similar to those described in the lungs of animals exposed to ozone.[10,11] They are not sufficiently specific, however, to exclude other irritants.

It is evident that men engaged in argon-shielded electric arc welding constitute a group of subjects likely to have frequent exposures to ozone and that any pulmonary damage done might be detected by tests of pulmonary function presently available.

Submitted for publication Sept 19, 1962.

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This work was supported in part by the Defence Research Board of Canada, grant No. 9310-84, and in part by the Medical Research Council of Canada.

TABLE 1.—*Clinical Data*

Subject	Age, Yr	Ht, In	Wt, Lb	Cigarettes per Day	A-SW,* Yr	Cough	Sputum	Abnor. Phys. Signs
A	48	68	170	20	10	Yes	No	No
B	45	66	125	30	4	Yes	No	Yes
C	51	68	206	20	5	Yes	Yes	Yes
D	46	68	134	12	10	No	No	No
E	36	70	172	12	10	No	No	No
F	35	66	153	½ oz †	3	No	No	No
G	46	66	125	20	2	No	No	No

* Argon-shielded electric arc welding.
† Pipe tobacco.

Methods

The complete staff of seven full-time welders of an electronics factory were selected as subjects. All these men worked together in a room approximately 40 by 20 by 15 ft subdivided into cubicles by canvas partitions about 6 ft high. Ventilation was only occasionally provided by opening one small window. At least one or two of the men were doing argon-shielded electric arc welding at all times throughout the working day. This task was shared about equally by the seven men who have done this type of welding for two to ten years. The mean concentration of ozone in the shop was found to be 0.2 to 0.3 ppm by the rubber cracking method.[2]

A full clinical history was taken with particular reference to illness involving the respiratory tract, and a thorough physical examination was performed. Each subject had had a chest x-ray reported as normal in the previous six months. The following measurements of pulmonary function were carried out: vital capacity, functional residual capacity (FRC), maximal midexpiratory flow rate, indirect maximum breathing capacity ($FEV_{0.75} \times 40$), and carbon monoxide diffusing capacity at rest and on exercise. Normal values for all the resting measurements were calculated from the formulae of Bates et al[12] and those for the exercise diffusing capacity from the formula of Donevan et al.[13]

Results

The clinical data are shown in Table 1 and the laboratory measurements in Table 2. Only one individual (D) had symptoms which might be attributable directly to the presence of ozone. He complained that whenever he did argon-shielded electric arc welding, the work area soon became pervaded with an irritating odor like that of burnt felt. He then developed soreness of the eyes and dryness of the mouth, throat, and trachea. His appetite, and his taste for cigarettes, disappeared until late in the evening.

Subjects A and B coughed only when smoking, while subject C reported a morning cough productive of one-fourth cup of whitish sputum. In addition, subjects B and C gave a history of two attacks of pneumonia in earlier life.

Examination of subject B revealed an occasional medium-pitched rhonchus over the

TABLE 2.—*Laboratory Data*

	VC, Liters		FRC, Liters		MMFR, L/Sec		$FEV_{0.75} \times 40$L/Min		D_{CO} (Rest), Ml/Mm Hg/Min		D_{CO} (Exercise), Ml/Mm Hg/Min	
Subject	p	a	p	a	p	a	p	a	p	a	p	a
A	4.0	4.2	3.2	3.5	3.52	6.36	112	146	17.2	16.8	32.8	43.6
B	3.8	3.1	3.3	2.4	3.53	2.85	99	93	17.7	13.4	37.6	28.5
C	3.9	3.7	2.9	3.0	3.40	3.16	118	100	16.4	22.9	33.0	37.1
D	4.1	3.2	3.5	3.3	3.60	3.50	101	105	17.8	22.0	27.3	33.3
E	4.7	4.5	3.5	3.6	4.11	3.90	134	128	21.0	15.1	40.2	36.5
F	4.1	3.1	3.0	2.0	3.94	4.12	124	106	20.5	22.6	36.3	31.7
G	3.8	4.0	3.3	4.0	3.49	4.82	97	133	17.4	13.1	27.3	24.4

p indicates predicted; a, actual; VC, vital capacity; FRC, functional residual capacity; MMFR, maximal midexpiratory flow rate; $FEV_{0.75} \times 40$, indirect maximum breathing capacity; D_{CO}, carbon monoxide diffusing capacity.

right anterior chest, cleared by cough, while in subject C transient rhonchi and a few fine basal rales were heard bilaterally. The remainder were free from signs and symptoms.

Three subjects (B, D, and F) showed a reduction in vital capacity. In one subject (D), this was an isolated finding. In a second (F), it was accompanied by a diminished FRC, the significance of which is uncertain in the presence of normal findings in other respects. The third (B) showed a reduction in all measurements. Subject E showed a reduction in the diffusing capacity at rest. However, as all other measurements, including the exercise diffusing capacity, were within normal limits, this finding is probably of no significance.

Comment

The only man with symptoms possibly attributable to ozone had no abnormal physical signs but did have a reduced vital capacity. Of the three men with cough (A, B, and C), two (B and C) had a history of previous pneumonia, and these were the only subjects who had abnormal physical signs. Only one of these (B), however, had abnormalities of pulmonary function. The pneumonia preceded the occupational exposure in all cases.

The abnormalities in the measurements of pulmonary function show no relationship to the number of years spent at argon-shielded electric arc welding; this finding suggests that the abnormalities were not caused by the occupational exposure to ozone. There is no obvious relationship to age or smoking habits, but this is difficult to assess in such a small number of individuals of similar age and habits.

In three of the men (B, D, and E) the measured vital capacity was below the predicted value by 0.7, 0.9, and 1.2 liters respectively. This measurement has a wide range in normals, and it is, therefore, difficult to attach any precise measurement of significance to this observation. It cannot be dismissed entirely, however, in view of Griswold's observation of a fall in vital capacity following an experimental ozone exposure.[5]

The finding of some deviation from predicted values in measured pulmonary function in three of seven smokers between the ages of 35 and 51, with an occupational exposure to dusts and fumes, is not unusual. Also there is the suggestion that in one case (B) the abnormalities are due to pre-existing chronic pulmonary disease. Our data demonstrate no major consequences on pulmonary function of repeated exposures to very low concentrations of ozone.

Some reservations are necessary in drawing conclusions from these data. First, the group of subjects is a rather small sample. Second, there may exist some degree of automatic selection of subjects, whereby particularly ozone-sensitive individuals may tend to transfer from argon-shielded electric arc welding to another type of work. The complete elimination of symptoms of upper respiratory tract irritation by reduction of ozone levels from 0.8 to 1.7 ppm to 0.2 ppm, as reported by Challen, is, however, in agreement with our viewpoint.[8] Bennett's recent work showing that chronic experimental exposure to 0.2 ppm of ozone produces no impairment of vital capacity or one-second forced expiratory volume also is in agreement with our findings.[7] The fact that a similar exposure to 0.5 ppm did produce a significant fall in the $FEV_{1.0}$ suggests that the welding shop we have studied is operating close to the limit of safety.

Summary and Conclusions

1. Seven men engaged in argon-shielded electric arc welding were examined clinically and by measurements of pulmonary function for evidence of respiratory damage attributable to ozone. The mean concentration of ozone in the welding shop was 0.2 to 0.3 ppm.

2. One man had symptoms associated with the argon-shielded electric arc welding. Three had a slight cough associated with smoking, and two of these, both of whom gave a past history of pneumonia, had abnormal physical signs in the chest.

3. The measurements of pulmonary function were: vital capacity, functional residual capacity (FRC), maximal midexpiratory flow rate, indirect maximum breathing capacity, and carbon monoxide diffusing capacity at rest and on exercise. Four of the seven showed no abnormality in any of the pulmonary function measurements. Three men had vital capacities below those predicted. In one, the only subject with symptoms possibly attributable to ozone, it was an isolated finding. In a second it was accompanied by a diminished FRC and in a third by a slight-to-moderate diminution in all the other measurements. However the history and physical examination suggested that this man had some pre-existing pulmonary disease.

4. These findings indicate that exposures to these low concentrations of ozone do not cause impairment of air flow or of pulmonary diffusion. However, recent evidence that slightly higher levels of ozone produced a decrease in the one-second forced expiratory volume suggests that they are approaching the limit of safety.

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Printed and Published in the United States of America