Thorax, 1980, **35**, 838–842

Smoke inhalation in firemen

KENNETH M UNGER, RICHARD M SNOW, JORGE M MESTAS, AND WARREN C MILLER

From the Pulmonary Division, Department of Medicine, The University of Texas Medical School at Houston, Texas, USA

ABSTRACT Thirty firemen were studied with pulmonary function tests immediately after a severe smoke exposure and then one and a half and 18 months later in order to evaluate acute and chronic changes in their spirometry. The results were compared with predicted values and with those from a group of closely matched control subjects. We found no significant differences between the acute post-exposure spirometry values and those recorded at six weeks and 18 months later. A trend toward an increased rate of volume loss in the FVC and FEV_1 was noted which is similar to other published observations. However, we did find a significant decrement in FVC compared with predicted value, and in FVC and FEV_1 compared with matched control subjects. This is further evidence that firemen may develop lung disease related to their occupational exposure.

Inhalation of toxic gases accounted for 11% of all reported injuries to firemen in 1976. Of the 161 662 firemen studied in the Fire Administration's Annual Death and Injury Survey, 152 (0·094%) were officially forced to retire or change jobs because of lung disease.[1] The hazard to firemen may be increasing because of the increased use of chlorinated hydrocarbons, such as polyvinyl chloride, in synthetic building materials. These substances may release large amounts of potentially toxic compounds, including hydrochloric acid, to the components of smoke as they decompose during the process of heating and burning.[2] After exposure, symptoms such as substernal chest pain, dyspnoea, and headache are commonly seen,[2] and hypoxia has been reported in a large percentage of firemen immediately after smoke inhalation.[3]

The acute and chronic effect of such exposure on pulmonary function, as measured by spirometry, is not clear. Small acute reversible changes in FEV_1 after the inhalation of dense smoke have been documented by Musk *et al.*[4] However, his group has been unable to document chronic changes in spirometry.[5]

We recently had the opportunity to study the pulmonary function of 30 firemen immediately after their exposure to dense smoke. Follow-up studies were performed six weeks and 18 months after the initial tests and are the subject of this report.

Address for reprint requests: Dr KM Unger, The University of Texas Medical School at Houston, 1270 Medical School Main Building, 6431 Fannin Street, Houston, Texas 77025, USA.

Methods

On 26 October 1987, a fire consuming a chemical warehouse on the outskirts of Houston, Texas was extinguished by 175 firemen. All the chemicals stored were chlorinated hydrocarbons, including pesticides, of complex organic structure. The fire was considered one of the most extensive and dangerous in Houston in the past five years by fire department spokesmen (fig 1).

Of the 175 men who responded to the alarm, 160 were seen in emergency rooms throughout Houston. Of these, 56 were brought to our emergency room for evaluation. Most complained of cough, substernal burning, shortness of breath, burning eyes, nausea, or headache. All patients seen in our emergency room were questioned specifically about symptoms related to the chest and eyes, and were asked about nausea and headache. Each had a physical examination with special attention to the chest.

We were able to study the pulmonary function of 30 of these men, including the five individuals admitted for observation. The spirometry was done the same evening as the incident by one of the investigators using a nine-litre water-filled spirometer (Warren E. Collins, Braintree, MA). Each subject performed three trials of both relaxed vital capacity and forced manoeuvres—the best result of each was taken for analysis. All results were corrected for body temperature and pressure, saturated with water (BTPS). Predicted values were calculated from age and

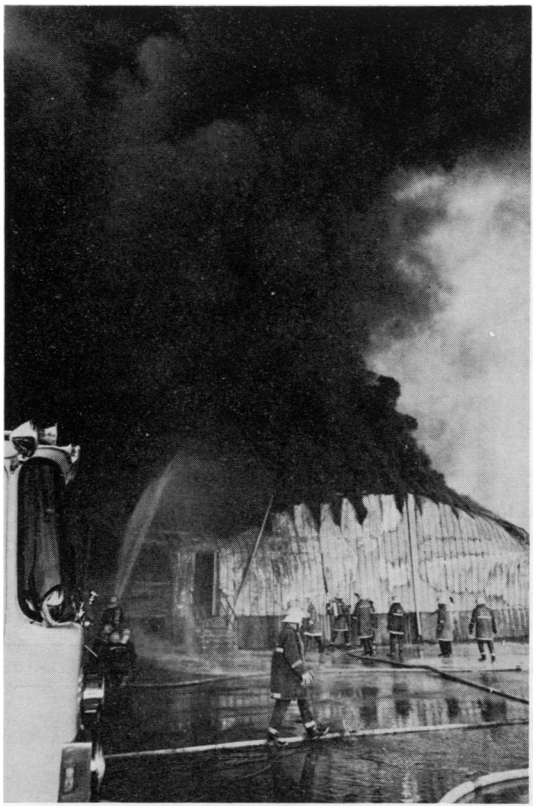

Fig 1 *Firemen extinguishing this fire, illustrating the close proximity of the men to the smoke. Published by kind permission of the Houston Post Company.*

height using the regression equations of Morris *et al*[6] for white subjects and of Stinson *et al*[7] for black subjects.

For comparison, a group of control subjects, closely matched for age, height, and smoking history, was selected sequentially from our pulmonary function laboratory files. Those known to have previous pulmonary disease or a systemic illness with pulmonary complications were excluded. They were selected without access to actual spirometric values. As a result, most of the selected control subjects had had their pulmonary function tested preoperatively for elective, non-thoracic surgery.

The follow-up studies were performed at six weeks and 18 months after the fire. They consisted of repeat pulmonary function testing and a questionnaire. The pulmonary function testing was performed using the same techniques and equipment and was done by the same investigators at the fire stations during the firemen's duty hours. The questionnaire which was administered at the time of the first follow-up study, requested information about the symptoms as recalled from the time of the fire and about residual symptoms at the time of this follow-up. Also, questions about proximity to the fire, duration of exposure, usage of an air pack during exposure, and cigarette smoking history were included.

The data collected were stored in a computer and analysed using the statistical procedures outlined by Nie *et al.*[8]

Results

Thirty firemen were studied initially with spirometry. The mean age was 28 years and these men had an average of seven years of service with the Houston Fire Department. Twenty-three per cent. had a history of five or more pack-years of smoking. When seen in the emergency room, 28 of these 30 men (93%) had some symptom attributed to their recent smoke exposure: 19 (63%) complained of substernal pain or burning, nine (30%) of headache, eight (27%) of burning eyes, eight (27%) of dyspnoea, and six (20%) had nausea (fig 2). None had wheezing at the time of their physical examination.

Of the initial 30 men studied, 24 were available for repeat studies at six weeks. The others either refused (three) or could not be located. There was no significant difference in age, length of service, or smoking history between the 30 who were initially studied and the 24 in the follow-up group. There was no significant difference in the prevalence of the presenting symptoms between those restudied and those not restudied. Of the firemen in this follow-up group, nine had used their air packs. All, however had removed them during the exposure period. More than 80% of the firemen were within 30 feet of the blaze, and all were in the unusually thick smoke. The median exposure time was 120 minutes with a range of 30 minutes to 15 hours. These men fought an average of 2·9 fires between the first study and the six-week follow-up.

The results of the pulmonary function tests (PFTs) of the 24 men restudied at six weeks are summarised in table 1. The mean forced vital capacity (FVC) was 4902+868 ml (mean ± SD) and the forced expiratory volume in one second (FEV_1) was 4046+772 ml. The FVC was significantly lower than the predicted values for this volume ($p<0·05$). The difference between observed and predicted FEV_1 did not reach

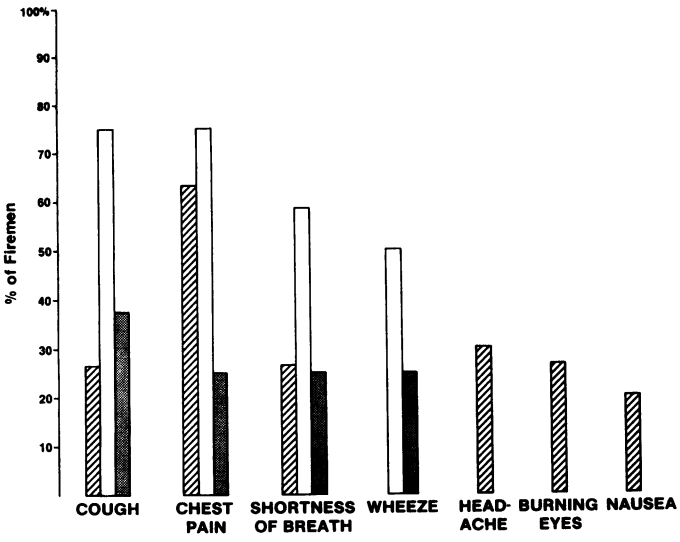

Fig 2 *Symptoms related to exposure to this fire as recorded in the emergency room (ER) record, as recalled at time of follow-up, and still present at time of the six-week follow-up. ▨=ER chart, □=Symptoms recalled from time of fire on questionnaire at follow-up six to eight weeks later. ▩=Symptoms still present at follow-up six to eight weeks later.*

Table 1 *Pulmonary function data from the 24 firemen studied immediately after smoke inhalation and six weeks later*

*Data**	*Immediately after*	*Six-week follow-up*	*Predicted values‡*
FVC (ml)	4902±868†	4897±875†	5263±683
FEV_1 (ml)	4046±772	4064±789	4198±445
FEV_1%	82·5±5·1	82·9±5·3	81·9±1·9
MMEF (l/s)	4·57±1·48	4·51±1·35	4·46±0·39

*Mean±SD.
†$p < 0.05$ when compared with predicted.
‡Predicted values from Morris[6] for whites and Stinson[7] for blacks.

Table 2 *Comparison of 24 firemen with matched control subjects*

*Data**	*Firemen†*	*Control subjects*	*p value*
Age (yr)	27·2±5·36	28·5±6·43	NS
Height (in)	70·6±2·4	70·5±2·7	NS
Smoking (pack-years)	5·75±10·0	6·63±8·2	NS
FVC (ml)	4902±868	5626±691	< 0·01
FEV_1 (ml)	4046±772	4490±592	< 0·05
MMEF (l/s)	4·57±1·48	4·34±1·09	NS
FVC (% predicted)	89±13	102±10	< 0·001
FEV_1 (% predicted)	93±16	104±11	< 0·05
MMEF (% predicted)	100±32	94±22	NS

*Mean±SD.
†PFTs done immediately after smoke inhalation.

significance. The FEV_1, expressed as a percentage of the forced vital capacity (FEV_1%) was 82·5 ±5·1%, and the maximum midexpiratory flow rate (MMEF) was 4·57±1·48 l/s. There was no significant difference between the FEV_1% or MMEF and the predicted values for these measurements.

Similarly, when the PFTs of this group were compared with those of the matched control subjects, there was a significant difference found for the FVC ($p<0.01$) and FEV_1 ($p<0.05$), but not for FEV_1% or MMEF (table 2). The significant difference seen for measurements of lung volume (FVC and FEV_1) and the lack of difference of the measurements of flow (FEV_1% and MMEF) remained when we compared the PFTs of the two groups expressed as a percentage of the predicted values.

When we compared the initial PFTs with those done six weeks later, we found no difference ($p>0.05$) (table 1). There was no difference between the PFTs of those men not restudied and the PFTs of the group as a whole. There was no difference between the PFTs of the smokers and non-smokers, or of whites and blacks.

Of the 30 men initially studied, 20 were available for repeat studies at 18 months. A comparison of the PFTs done initially and at 18 months revealed a decrease during this period of time of 122 ml in the mean FEV_1 and 62 ml in the mean FVC (table 3). The differences observed between the initial and 18 month follow-up PFTs did not reach significance by paired *t* test.

Table 3 *Pulmonary function data on the 20 firemen studied immediately after smoke inhalation and 18 months later*

Data	*Immediately after*	*18-month follow-up*
FVC (ml)	4862±717	4800±778
FEV_1 (ml)	4003±633	3881±515
MMEF (l/s)	4·47±1·15	4·20±0·99

Discussion

Despite the apparent risk from exposure to highly toxic fumes, the literature is not clear as to the nature or severity of chest disease which firemen develop as a result of their occupation. An increased prevalence of chronic cough and sputum production among experienced fire fighters, in comparison to recent recruits of the same age, has been noted.[9] Such nonspecific respiratory complaints were more common among those who reported the production of black mucus after exposure to fire and smoke, and correlated with indicators of accumulated lifetime episodes of severe exposure. A prospective study of 1430 Boston firemen found an annual loss of 77 ml from the FVC and 68 ml from the FEV_1, compared to only 25 to 30 ml in normal, non-firemen, control subjects. This loss of pulmonary function correlated with the frequency of exposure. There was no decrease over this year in $FEV_1\%$.[10] When the study of these firemen was extended over a three-year period however, this abnormally rapid loss of lung volume was not sustained.[5] The apparent discrepancy in these two reports might be explained by two factors. First, improved safety techniques introduced during the period of study, such as the use of air packs, may have reduced the severity of exposure to toxic fumes. Second, those men affected by lung problems may be the most likely to leave the fire department, leaving those less affected for study.[10 11]

Hypoxaemia was found in 19 of 21 Los Angeles firemen studied immediately after they were exposed to smoke, although most were asymptomatic.[3] When spirometry studies were done a month later and compared with a matched group of non-firemen, no statistical difference could be shown.[12] We studied our group of firemen in an attempt to delineate the effect on spirometry of a single severe, but uncomplicated, smoke inhalation. Our initial data revealed a significant decrement in lung volumes: FVC compared to predicted values, and both FVC and FEV_1 compared to matched control data. $FEV_1\%$ and MMEF were normal.

We had expected to find an increase in the FEV_1 and FVC at the six-week follow-up study, which would imply that the firemen had had an acute but reversible lung injury. We found no such difference. In fact, the results of the two measurements were almost identical (table 1). Musk *et al*[4] have shown that firemen can assess the severity of their smoke inhalation quite accurately. This was done by comparing their subjective assessment with objective criteria, either air samples obtained at the scene of the fire or by measuring the deposits collected in air filters. Since our firemen assessed their smoke inhalation to be severe enough to require medical evaluation (not only those seen at our hospital, but 160 of a total of 175 involved) and because of the high prevalence of related symptoms (93%), we cannot dismiss the lack of an appreciable change as implying that the smoke inhalation was mild.

There are two possible explanations for the decreased lung volumes seen in our firemen. These abnormalities could have predated their exposure to this fire, or the firemen could have sustained a pulmonary injury which resulted in a significant decrement in lung volume. We find the latter explanation improbable especially since there was no demonstrable flow obstructive component, nor any reversibility—that is, follow-up functions were not significantly different. Musk, by studying firemen many times before and immediately after exposure to smoke, found a small, but significant decrease in lung volumes, which was related to the severity of the exposure.[4] The decrement seen in our firemen, immediately after the fire, was much larger than the changes reported by Musk even for the most severe exposure, and is therefore unlikely to be the result of this single exposure. Further, the data of Musk strongly imply that the changes he observed were reversible as his firemen had no cumulative loss over the study period. We saw no reversibility. Therefore, it is most likely that this decrement was present before and was not specifically caused by, this exposure, despite its severity.

The decrease we have demonstrated is similar in degree to that found by Peters *et al*,[10] and the decrease in FVC and FEV_1 per year is similar to that which they found (though not reaching significance in our study, probably because of the small numbers).

This experience gives further support to the theory that seasoned fire fighters do have abnormal pulmonary function in addition to a high degree of nonspecific chest symptomatology, and is consistent with the findings of Sidor and Peters.[9 10] Further, our data strongly suggest that the reduction results from frequent, repeated insults, each of which causes a decrement difficult to quantitate with spirometry, but which have a cumulative effect over years of exposure.

Increased prevalence of chronic cough and sputum production has also been noted among workers exposed to nonspecific airways irritants

in the coal,[13] coke,[14] steel,[15] and other industries. Lapp has called this process industrial bronchitis.[16] Spirometry has been studied in subjects with bronchitis induced by occupational exposure and generally shows a 50 to 160 ml reduction in mean FEV_1 or FVC, if large numbers of workers are examined.[17] The similarity of the symptoms and findings in those with industrial bronchitis to those of firemen are apparent from our study and those of others.[8 9]

In summary, we have described a group of firemen who were exposed to the noxious products of combustion. They had a high prevalence of nonspecific complaints related to the chest similar to the symptoms of industrial bronchitis. We found a significant decrement in lung volumes (FVC, FEV_1), and no improvement during the follow-up period. These results provide additional evidence for the presence of abnormal pulmonary function in fire fighters. They suggest that the decrement in function is related to frequent, repeated insults over time rather than sudden decreases associated with specific exposures.

We wish to acknowledge the help of Mr James Hudgens, Safety Officer, Mr Leonard Miles, safety co-ordinator, and Mr Wayne Dees, previous safety co-ordinator, all of the Houston Fire Department, who assisted us in obtaining the follow-up studies.

References

1 Fire Administrations. Annual death and injury survey. *International Firefighter* 1976; 9–16.
2 Dyer RF, Esch VH. Polyvinyl chloride toxicity in fires: hydrogen chloride toxicity in fire fighters. *JAMA* 1976; **235**:393–7.
3 Genovesi MG, Tashkin DP, Chopra S, Morgan M, McElroy C. Transient hypoxemia in firemen following inhalation of smoke. *Chest* 1977; **71**: 441–4.
4 Musk AW, Smith TJ, Peters JM, McLaughlin E. Pulmonary function in fire fighters: acute changes in ventilatory capacity and their correlates. *Br J Ind Med* 1979; **36**:29–34.
5 Musk AW, Peters JM, Wegman DH. Lung function in fire fighters. I: a three-year follow-up of active subjects. *Am J Public Health* 1977; **67**: 626–9.
6 Morris JF, Koski A, Johnson LC. Spirometric standards for healthy non-smoking adults. *Am Rev Respir Dis* 1971; **103**:57–67.
7 Stinson JM, McPherson GL, Hicks K, Scott V, Sykes R, Cobbs W. Spirometric standards for healthy black adults. *Am Rev Respir Dis* 1979; **119**:237.
8 Nie NH, Bert DH, Hull CH. *SPSS: statistical package for the social sciences.* New York, McGraw-Hill Book Company, 1970.
9 Sidor R, Peters JM. Prevalence rates of chronic non-specific respiratory disease in fire fighters. *Am Rev Respir Dis* 1974; **109**:255–61.
10 Peters JM, Theriault GP, Fine LJ, Wegman DH. Chronic effect of fire fighting on pulmonary function. *N Engl J Med* 1974: **291**:1320–2.
11 Musk AW, Peters JM, Wegman DH. Lung function in fire fighters. II: a five-year follow-up for retirees. *Am J Public Health* 1977; **67**:630–3.
12 Tashkin DP, Genovesi MG, Chopra S, Coulson A, Simmons M. Respiratory status of Los Angeles firemen: one-month follow-up after inhalation of dense smoke. *Chest* 1977; **71**:445–9.
13 Kibelstis JA, Morgan EJ, Reger R, Lapp NL, Seaton A, Morgan WKC. Prevalence of bronchitis and airway obstruction in American bituminous coal miners. *Am Rev Respir Dis* 1973; **108**:886–93.
14 Walker DD, Archibald RM, Attfield MD. Bronchitis in men employed in the coke industry. *Br J Ind Med* 1971; **28**:358–63.
15 Brinkman GL, Coates EO Jr. The prevalence of chronic bronchitis in an industrial population. *Am Rev Respir Dis* 1962; **86**:47–54.
16 Lapp NL. Industrial bronchitis. In: Morgan WKC, Seaton A. *Occupational lung diseases.* Philadelphia: WB Saunders, 1975: 265–73.
17 Lapp NL, Hankinson JL, Burgess DB, O'Brien R. Changes in ventilatory function in coal miners after a work shift. *Arch Environ Health* 1972; **24**:204–8.