

Respiratory symptoms among glass bottle workers — cough and airways irritancy syndrome?

S. B. Gordon,^{*,†} A. D. Curran,[†] D. Fishwick,^{*,†} A. H. Morice[†] and P. Howard^{*}

**Department of Medicine and Pharmacology, University of Sheffield, Royal Hallamshire Hospital, Sheffield S10 2JF, UK; †Health and Safety Laboratory, Broad Lane, Sheffield, UK; ‡Pulmonary Medicine, University of Sheffield, Northern General Hospital, Herries Road, Sheffield, UK.*

Glass bottle workers have been shown to experience an excess of respiratory symptoms. This work describes in detail the symptoms reported by a cohort of 69 symptomatic glass bottle workers. Symptoms, employment history and clinical investigations including radiology, spirometry and serial peak expiratory flow rate records were retrospectively analyzed from clinical records. The results showed a consistent syndrome of work-related eye, nose and throat irritation followed after a variable period by shortness of breath. The latent interval between starting work and first developing symptoms was typically 4 years (median = 4 yrs; range = 0–28). The interval preceding the development of dyspnoea was longer and much more variable (median = 16 yrs; range = 3–40). Spirometry was not markedly abnormal in the group but 57% of workers had abnormal serial peak expiratory flow rate charts. Workers in this industry experience upper and lower respiratory tract symptoms consistent with irritant exposure. The long-term functional significance of these symptoms should be formally investigated.

Key words: Bottle workers; cough; irritant; respiratory symptoms.

Occup. Med. Vol. 48, 455–459, 1998

Received 15 September 1997; accepted in final form 26 May 1998

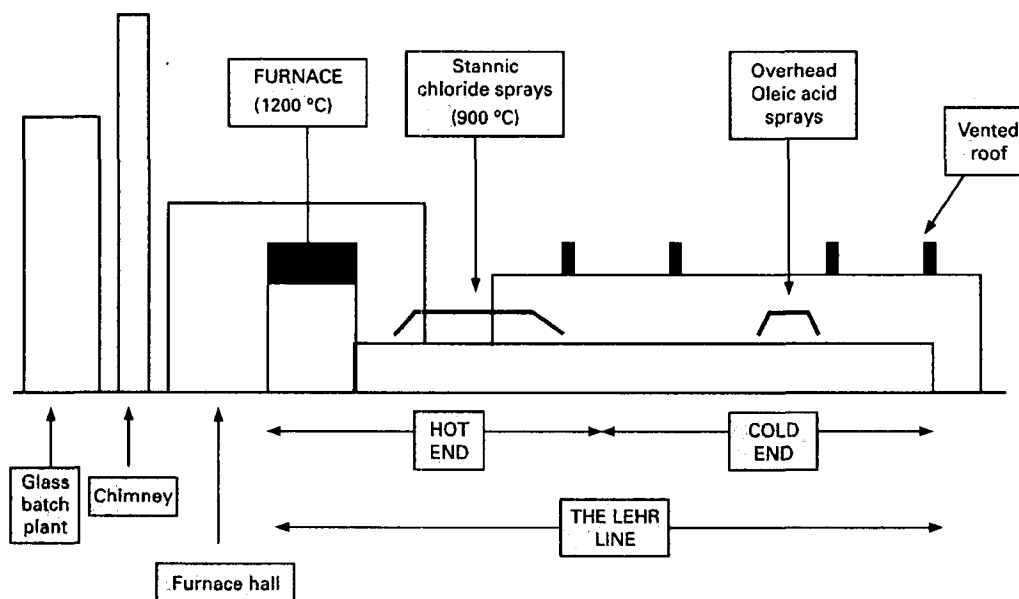
INTRODUCTION

Glass bottles are manufactured on a 'lehr line' system which can be divided into 'hot' and 'cold' processing areas (Figure 1). Molten glass is first produced from sand, dolomite, arsenic, soda ash and cullet dust (a form of recycled glass) in a furnace. It is shaped on moulds swabbed with sulphur, and then hardened with stannic chloride which is sprayed on to the hot glass. To prevent sticking in the packing process, the hot bottles are sprayed with an organic lubricant. Glass bottle workers work in close proximity to the process, often without respiratory protection. They are therefore exposed to a number of low molecular weight irritant substances, including oxides of sulphur, hydrochloric acid, long-chain fatty acids and the products of fatty acid breakdown induced by heat.

In the United States, respiratory symptoms were first reported in 1985 in workers exposed to stannic chloride in the manufacture of glass bottles.¹ The symptoms of chest pain, exertional dyspnoea, cough and wheezing were thought to be due to exposure to hydrochloric acid fumes produced in the hydrolysis of stannic chloride with steam. Glass bottles are manufactured in the UK using identical machinery and a very similar process to that used in the USA. We have recently published the results of a case-control study from the UK which showed a significant excess of upper and lower respiratory tract symptoms in a randomly selected group of glass bottle workers compared to matched controls.² The glass bottle workers had a lower cough threshold on cough challenge testing. Wheeze, however, was not shown to be significantly different between cases and controls in our study. Furthermore, methacholine challenge testing by a validated method³ showed no difference between the two study groups, or the results of a large community survey⁴ which used the same methacholine challenge technique. These symptomatic workers do not therefore have classical occupational asthma.⁵ This work describes

Correspondence and reprint requests to: Dr A. D. Curran, Health and Safety Laboratory, Broad Lane, Sheffield S3 7HQ, UK.
Tel: (+44) 114 289 2689; Fax: (+44) 0114 289 2768;
e-mail: andrew.curran@hsl.ga.uk

Figure 1. The layout of a typical glass bottle making factory.



in detail the clinical syndrome presented by a new cohort of 69 symptomatic glass bottle workers from the same factory.

METHODS

Selection of records

Glass bottle workers reporting respiratory symptoms to union officials over a 6-year period were referred to a respiratory physician (PH) for assessment, and for eventual consideration of compensation. None of the workers in this study had previously been studied by our group.² All of these records were made available for review.

Review of records

Full employment and medical histories were recorded separately by the same physician in each case, and general practitioner records were reviewed shortly following the initial consultation. These employment and medical histories were reviewed and information coded on a standard form which summarized the occupational history and the presence or absence of symptoms, their date of onset and exacerbating or relieving conditions including any relationship to work. Data regarding risk factors for bronchial hyperresponsiveness was also codified. Workers were defined as smokers if they had smoked more than five cigarettes per day at any time during the previous year.

Investigations

At initial assessment, a chest X-ray and spirometry were performed with assessment of reversibility to salbutamol. Workers were asked to complete 4 weeks of serial peak

expiratory flow rate (PEFR) charts including a work shift record. These radiology and spirometry reports were later reviewed and PEFR data were prepared for visual inspection using a prototype of the OASYS system⁶ by kind permission of the North Staffordshire Medical Institute. Following the retrospective PEFR chart analysis, 10 symptomatic workers with abnormal PEFR charts were assessed during this study for bronchial hyperresponsiveness by methacholine challenge testing. This was carried out using a Mefar (Brescia, Italy) dosimeter and Spirotrac III (Vitalograph, UK) software using a validated modification of the dosimeter protocol of Hendrick *et al.*^{3,7}

Data analysis

Codified data were entered into a database (Microsoft Access 1.0). This allowed the most common symptoms to be determined, and the frequency of these symptoms amongst workers, along with their date of onset and duration. During the analysis, hot and cold end workers were compared in terms of frequency and pattern of reported symptoms. Work-related symptoms were identified from the database and expressed as a percentage of the total reports of that symptom. The relation of symptoms to work was also described as a latent interval after starting work for each symptom. Finally, serial PEFR charts were screened after OASYS data entry to exclude charts with no clear documentation of shift, less than three blows per day, or evidence of confabulation. Within-day or between-day variation of PEFR in excess of 20% of the mean value was regarded as abnormal and indicative of asthma. An occupational relationship was implied by a worsening of asthma at or immediately following work. PEFR records showing a low mean value (mean more than 20% below predicted value for age, sex and height⁸) or a deterioration in mean value across the working week were also recorded as abnormal.

Ethics

Permission to review worker records was sought *via* trade union officials and ethical approval for methacholine challenge testing in bottle workers was granted by the South Sheffield Research Ethics Committee. Informed written consent was obtained from these workers.

RESULTS

Study population

Sixty-nine workers (68 males, one female) with mean age of 47.3 years (range = 24–67 yrs) were assessed. Approximately 500 were known currently to be members of the relevant union. Forty workers (58%) were smokers and of these, nine had a past history of asthma, eczema or hayfever. None of the non-smokers (19) had a history of asthma, eczema or hayfever.

Employment history

The group had a mean career duration of 20 years (SD = 7 yrs). Although most workers had carried out several tasks during their career, 29 workers described themselves as having worked predominantly at the cold end and 25 at the hot end. The remaining 15 workers had worked in both areas. Seventeen workers had retired since their symptoms started and of these 10 were cold end workers, six were hot end workers and one was a fitter who worked at both ends. The retired group had a mean career duration of 25.1 years (range = 15–41 yrs).

Symptoms

The most common symptoms were grouped into categories and then divided into upper respiratory symptoms (eye, nose or throat symptom) and lower respiratory symptoms (cough, wheeze and shortness of breath). Seventy-four per cent of workers had symptoms suggestive of irritation of the eye, nose or throat. None of the workers had these symptoms before starting work in the bottle factory. Cough (66% of workers), shortness of breath (64%) and wheeze (42%) were the next most common symptoms. None of the workers experienced recurrent cough before working in the factory, but four with previously diagnosed asthma had experienced wheezing episodes and two had been short of breath prior to starting work. Only four workers complaining of cough satisfied clinical criteria for chronic bronchitis. There was no difference seen in the pattern or frequency of symptoms between hot and cold end workers.

Symptom exacerbation at work

In the group of 63 workers who described upper respiratory symptoms, 44 (70%) stated that they were worse at work, whilst 20 (32%) described an improvement at weekends and 47 (75%) during holidays. Of the

62 workers describing a lower respiratory tract symptom, 37 (60%) reported this as being worse at work, with 20 (32%) reporting improvement at the weekends and 33 (53%) while on holiday.

Latency and duration of symptoms

Upper respiratory symptoms were seen to occur for the first time together and with a latency of less than 5 years between starting work and developing symptoms in the majority of cases (median = 4 yrs, inter quartile range = 1.75–10.88 yrs). Lower respiratory symptoms began with cough (median latency 11.5 yrs, inter quartile range = 3.83–15.5 yrs) and shortness of breath occurred much later (median latency 16 yrs, inter quartile range = 12.5–20.5 yrs). In all of these analyses, there was no difference seen between hot and cold end workers.

Incidence of symptoms over study period

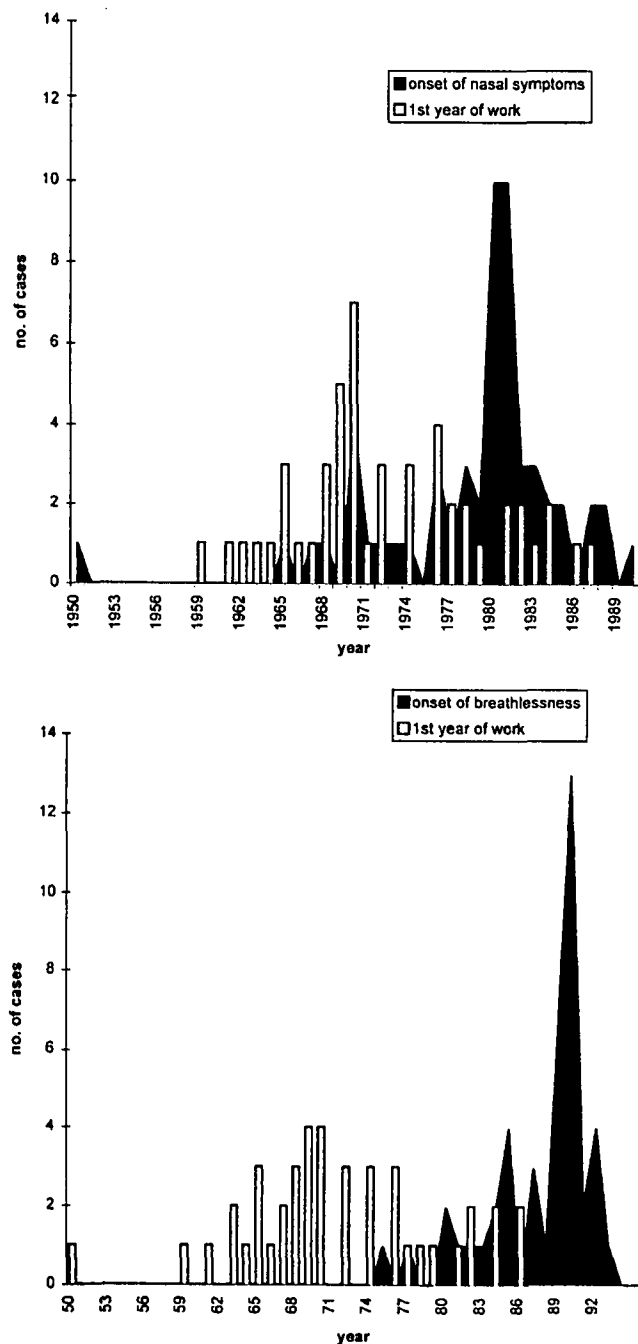
New cases of both upper and lower respiratory symptoms occurred between 1950 and 1994 (Figure 2). There was a peak of new cases of all upper respiratory symptoms in the period 1976–83 (median = 1980). New cases of breathlessness increased to a maximum in the period 1986–90 (median = 1989). There was no particular peak of cough or wheeze over the period spanned despite the pattern of recruitment shown in the figure. Recruitment in the sample studied spanned the period 1950–87 with a peak in the early 1970s (median = 1970; inter quartile range = 1968–77).

Investigations

Sixty-eight chest X-ray reports were reviewed of which 61 were normal. Pleural plaques (four), emphysema (one), scoliosis (one) and reticulonodular shadowing (one) were the abnormalities reported in the abnormal films. Spirometry was normal in 56 of the 63 workers tested. Abnormalities in the remainder were obstructive pattern (four), mild obstructive pattern (one), restrictive pattern (one) and hyper inflated (one). Response to inhaled β -agonist was tested in all 63 workers. A 15% improvement in FEV₁ was seen in one worker but five others showed a 10% improvement. Forty-two serial PEF_R records were judged to be of an adequate quality. Using the OASYS system, these were visually categorized as normal (18), asthma with an occupational relationship (seven), asthma without an occupational relationship (11), low mean peak flow of more than 20% below predicted (one) and mean PEF_R decreasing through the working week (five). The percentage of workers completing charts was therefore 62%, and the percentage of those that were abnormal was 57%.

Methacholine challenge testing was carried out on 10 workers with abnormal serial PEF_R records. Nine of these experienced a 20% bronchoconstriction at a dose of 6.4 mg of methacholine or less and five at a PD₂₀ of less than 1.5 mg methacholine. Eight of the 10 workers selected for methacholine challenge were present or

Figure 2. The presentation of new symptoms in this cohort showed a peak in 1980 for nasal symptoms and in 1989 for shortness of breath.



ex-smokers. Repeat spirometry prior to methacholine testing showed that those with bronchial hyperresponsiveness at methacholine challenge also had obstructive spirometry (reduced FEV1 and FEV1/FVC) prior to the challenge test.

DISCUSSION

This group of glass bottle workers has now been studied in detail by one physician. Although these workers underwent assessment in some cases with a view to compensation issues, and thus are not representative of a random

population, the medical information gathered furthers our knowledge of this group of workers, and particularly with reference to the time course and latency related to the development of these symptoms.

Glass bottle workers have previously been reported to have work-related symptoms^{1,2} and an excess of symptoms compared to matched control workers.² Indeed, glass bottle workers reported an excess of all respiratory symptoms enquired about except wheeze, when compared to a control population.² Interestingly, they did not exhibit an excess of bronchial hyperresponsiveness compared to matched controls, but did have a lowered cough threshold. The case-control study on which this data was based, therefore, suggested that the symptoms seen in this group of workers did not relate to a diagnosis of asthma, or indeed occupational asthma. Furthermore, it suggested that chronic, low dose exposure to 'irritant' agents may induce a syndrome characterized by heightened cough sensitivity but not bronchial hyperresponsiveness. Indeed, atopy did not appear to influence this response.

The current study, retrospective analysis of medical records, was designed to allow maximum further clinical information to be gleaned from this interesting group of workers. It studied a group of workers exposed to irritant fumes in a glass bottle factory for a mean duration of 20 years. The described syndrome consisted again of symptoms from the upper and lower respiratory tract.

What was striking, and adds further to the data from the case-control study, was that the latent period for the development of symptoms, after first exposure at work, was variable for each symptom. For example, the median latency for upper respiratory tract symptoms was relatively short at 4 years, suggesting that these symptoms may relate directly to a specific exposure at work. Although we have no evidence to support this in glass bottle workers, this duration is similar to those seen in workers who become truly sensitized to agents in the workplace.⁹

Conversely, the latency for the development of shortness of breath was long, with a median of 16 years. This suggests that either a proportion of this symptom relates to a low dose irritant exposure, and the effect of this takes many years to become clinically apparent or more likely that this simply reflects the process of ageing in a group of workers with high current smoking levels. The pattern of symptom latency was also interesting with distinct 'peaks' (as shown in Figure 2) for years of onset. There are many possible explanations for this finding, and these include possible changes in industrial processes and the chemicals used, or perhaps a single incident causing a very high exposure (akin to a reactive airways dysfunction syndrome). There is no evidence from the careful histories supplied from workers that the latter is the case. No further inference can be made on the possible rise in symptom levels recently, as this will be influenced by many uncontrollable factors such as recall bias and the population number from which these workers was drawn.

The individuals within this case series again did not exhibit an excess of atopy, as assessed by a positive history of asthma, eczema or hayfever, compared to population data.¹⁰

In summary, this group of workers did not appear to have either classical occupational asthma or the reactive airways dysfunction syndrome.¹¹

An excess of respiratory symptoms in workers exposed to low molecular weight irritant fumes has been reported in several other industries. In particular, exposure to irritants in the silicon carbide,¹² synthetic fibre¹³⁻¹⁵ and dye^{16,17} industries has been associated with an increase in cough, phlegm, wheeze and exertional dyspnoea among workers. Exposure to chlorine or hydrogen chloride gas in pulpmills and synthetic fibre plants has been associated with an increase in acute^{18,19} and chronic^{20,21} symptoms of chest-tightness, phlegm, wheeze, cough and breathlessness as well as throat and eye irritation and 'flu-like' symptoms.

There was apparent conflict in this dataset between the results of initial spirometry and serial PEF and methacholine testing, in that the majority of spirometry was normal, although 57% of PEF data (that was interpretable) were thought to be abnormal, and although repeat spirometry mitigated in favour of a respiratory abnormality being truly present in this predominantly smoking cohort.

Spirometric analyses of workers complaining of irritant-induced respiratory symptoms have been conflicting in other published reports. Initial findings²² in a group of sulphur dioxide-exposed copper smelter workers showed a progressive decline in FEV1 but follow-up studies failed to confirm this finding.²³ Data from the pulpmill industry²¹ also indicate that repeated exposure to chlorine gas may produce permanent obstructive lung damage.

In conclusion, irritant-exposed glass bottle workers show a consistent pattern of symptoms as well as the previously reported excess of symptoms when compared to other workers. In addition, the latency for different types of respiratory symptoms appeared to be highly variable.

It is possible that this syndrome of cough and airways irritancy may be followed after some time by respiratory impairment, although any interpretation of longitudinal loss in lung function will need careful assessment in view of the potential major role of cigarette smoking. These issues can only be assessed in a longitudinal study which should now include assessment of irritant fumes in the glass factories, cough threshold, BHR and objective assessments of exercise tolerance in these workers.

REFERENCES

1. Levy BS, Davis F, Johnson B. Respiratory symptoms among glass bottle workers exposed to stannic chloride solution and other potentially hazardous substances. *J Occup Med* 1985; 27: 277-282.
2. Gordon SB, Curran AD, Turley AJ, *et al.* Glass bottle workers exposed to low-dose irritant fumes cough but do not wheeze. *Am J Resp Crit Care Med* 1997; 156: 206-210.
3. Turley AJ, Gordon SB, Morice AH. Accurate computerised methacholine challenge testing using the Mefar dosimeter and Spirotrac software [Abstract]. *Am J Resp Crit Care Med* 1995; 151: A397.
4. Devereux G, Ayatollahi T, Ward R, *et al.* Asthma, airways responsiveness, and air pollution in two contrasting districts of northern England. *Thorax* 1996; 51: 169-174.
5. Bernstein IL, Bernstein DI, Chan-Yeung M, Malo J. Definition and classification of asthma. In: Bernstein IL, Chan-Yeung M, Malo J, Bernstein DI, eds. *Asthma in the Workplace*. New York, NY (USA): Marcel Dekker, 1993: 1-4.
6. Gannon PFG, Newton DT, Belcher J, Pantin CFA, Burge PS. Development of OASYS-2: a system for the analysis of serial measurements of peak expiratory flow in workers with suspected occupational asthma. *Thorax* 1996; 51: 484-489.
7. Hendrick DJ, Fabbri LM, Hughes J, *et al.* Modification of the methacholine inhalation test and its epidemiologic use in polyurethane workers. *Am Rev Respir Dis* 1986; 188: 600-605.
8. Cotes JE. *Lung Function*. Oxford, UK: Blackwell, 1979.
9. Chan-Yeung M. Assessment of asthma in the workplace. *Chest* 1995; 108: 1084-1117.
10. Fleming DM, Crombie DL. Prevalence of asthma and hay fever in England and Wales. *Br Med J* 1987; 294: 279-283.
11. Brooks SM, Bernstein IL. Reactive airways dysfunction syndrome or irritant-induced asthma. In: Bernstein IL, Chan-Yeung M, Malo J, Bernstein DI, eds. *Asthma in the Workplace*. New York, NY (USA): Marcel Dekker, 1993: 533-576.
12. Osterman JW, Greaves IA, Smith TJ, Hammond SK, Robins JM, Theriault G. Respiratory symptoms associated with low level sulphur dioxide exposure in silicon carbide production workers. *Br J Ind Med* 1989; 46: 629-635.
13. Kremer AM, Teake MP, Boleij JSM, Schouten JP, Rijcken B. Airway hyperresponsiveness and the prevalence of work-related symptoms in workers exposed to irritants. *Am J Ind Med* 1994; 26: 655-669.
14. Kremer AM, Pal TM, Boleij JSM, Schouten JP, Rijcken B. Airway hyperresponsiveness, prevalence of chronic respiratory symptoms, and lung function in workers exposed to irritants. *Occup Environ Med* 1994; 51: 3-13.
15. Kremer AM, Pal TM, Schouten J, Rijcken B. Airway hyperresponsiveness in workers exposed to low levels of irritants. *Eur Respir J* 1995; 8: 53-61.
16. Nilsson R, Norlinder R, Wass U, Meding B, Belin L. Asthma, rhinitis and dermatitis in workers exposed to reactive dyes. *Brit J Ind Med* 1993; 50: 65-70.
17. Docker A, Wattie JM, Topping M, *et al.* Clinical and immunological investigations of respiratory disease in workers using reactive dyes. *Brit J Ind Med* 1987; 44: 534-541.
18. Salisbury DA, Enarson DA, Chan-Yeung M, Kennedy SM. First-aid reports of acute chlorine gassing among pulpmill workers as predictors of lung health consequences. *Am J Ind Med* 1991; 20: 71-81.
19. Kennedy SM, Enarson DA, Janssen R, Chan-Yeung M. Lung health consequences of reported accidental chlorine gas exposures among pulpmill workers. *Am Rev Respir Dis* 1991; 143: 74-79.
20. Bherer L, Cushman R, Courteau J, *et al.* Survey of construction workers repeatedly exposed to chlorine over a three to six month period in a pulpmill; II. Follow-up of affected workers by questionnaire, spirometry, and assessment of bronchial responsiveness 18 to 24 months after exposure ended. *Occup Environ Med* 1994; 51: 225-228.
21. Chan-Yeung M, Lam S, Kennedy SM, Frew A. Persistent asthma after repeated exposure to high concentrations of gases in pulpmills. *Am J Respir Crit Care Med* 1994; 149: 1676-1680.
22. Smith TJ, Peters JM, Reading JC, Castle CH. Pulmonary impairment from chronic exposure to sulphur dioxide in a smelter. *Am Rev Respir Dis* 1977; 116: 31-39.
23. Smith TJ, Peters JM. Longitudinal evaluation of pulmonary function in copper smelter workers exposure to sulphur dioxide. *Am Rev Respir Dis* 1986; 133: 1332.