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Occupational Asthma in a Fibre Glass Works

M. J. FINNEGAN, C. A. C. PICKERING
Department of Thoracic Medicine, Wythenshawe Hospital, Manchester and The Group for Occupational Lung Diseases (GOLD)

P. S. BURGE
Department of Thoracic Medicine, East Birmingham Hospital, Birmingham

T. R. P. GOFFE
Factory Medical Officer

P. K. C. AUSTWICK, P. S. DAVIES
Aerobiology Unit, Brompton Hospital, Frimley, Surrey

Summary

Seven cases of work related asthma occurring in a continuous filament glass fibre plant are described. All but one had no past history of asthma. Bronchial challenge testing with several agents has failed to identify the cause. The work related rhinitis complained of by 20 per cent of the workforce appears to be due to airborne glass fibre fly, since this symptom was reproduced by challenge with fly. Occupational asthma in association with continuous filament glass fibre production has not previously been reported.

Introduction

Fibre glass is not regarded as a harmful or toxic substance (Hill, 1977) and is widely used throughout many industries. Its effects on the eye have been recently described by Stockholm et al. (1982). Effects on the skin have been appreciated for many years and are thought to be due to mechanical penetration of the skin and subsequent release of histamine (Possick et al., 1970). This is believed to be of nuisance value only and to have no long term harmful effects on the skin.

Irritation of the upper respiratory tract caused by inhalation of fibre glass dust has also been described (Milby and Wolf, 1969) but adverse effects on the lower respiratory tract have not been detected in epidemiological studies (Working Party on Toxic Substances, 1979). To our knowledge no paper describing work related asthma (WRA) in association with the manufacture of continuous filament glass fibre has been published. Two case reports (Hill, 1977) have described asthma in association with the use of glass fibre in clothing and textile factories respectively. Other possible causes for the asthma were not, however, excluded.

Materials, Subjects and Methods

Following the identification of a person with humidifier fever (HF) in a glass fibre works, an environmental study was initiated in the factory. The study was essentially divided into two parts:

a. an inspection of the work process;
b. a cross sectional survey which was open to the whole workforce and consisted of a doctor administered respiratory questionnaire, simple spirometry using a bellows spirometer (Vitalograph) and a blood specimen.

The Production Process

The glass plant is 240 m long, 40 m wide and 12 m high. It is divided into three sections (*Fig.* 1): the filament forming section, the strand conversion area (SCA) and the warehouse (in which a fibre glass mat plant has subsequently been built). The process is continuous. Molten glass from the furnace flows from 52 'bushings' above the filament forming platform. Each is a metal box with 400–1200 uniform holes of diameter 45–83 thousandths of an inch. As they emerge from the bushing the glass fibres are red hot. They are cooled by water sprays and pass over a roller which applies a dressing to them. They are drawn together to form a

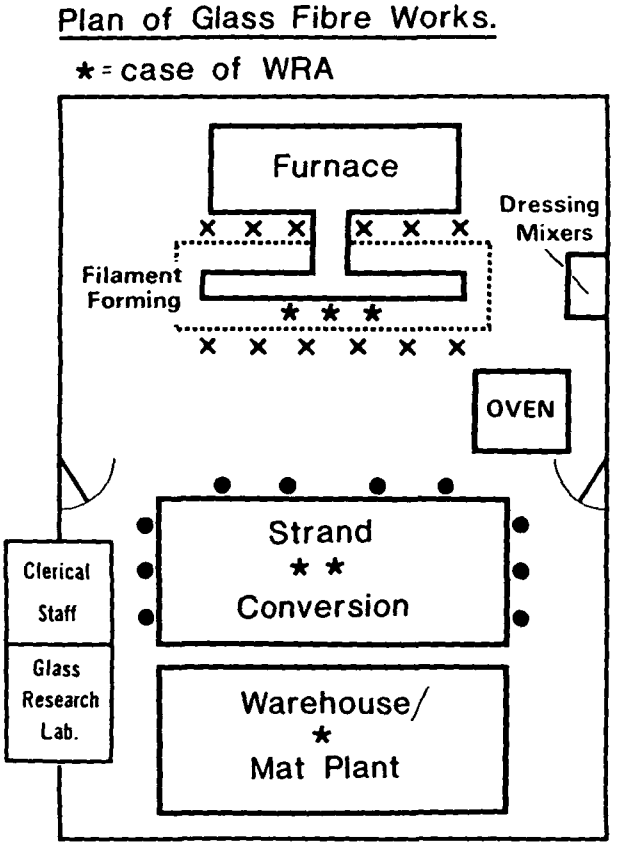

Fig. 1. Plan of glass fibre works. • represents a humidifier. Each × represents a bushing on the filament forming platform.

strand. The type of dressing used depends on the planned use of the glass fibre. The dressings are mixed in a room near the filament forming platform.

A winding head stretches the fibres to a diameter of 6–15 μ and winds them to produce a 'cake'. These are passed on a monorail into a large drying oven where they are dried for 13 hours at temperatures up to 135 °C. This also effects various chemical changes in the dressing. The ovens were known to leak fume which can easily be smelt in the factory.

The dressings are complex chemical mixtures in a water emulsion. Their exact content is commercially sensitive, but the broad groups and amounts of most of the chemicals are known. The major component is the film former: either polyvinyl acetate, a polyester, a polyurethane or an epoxy compound. A lubricant is also present as well as a resin acceptor (usually a silane or chrome compound). Neither phenol- nor urea-formaldehyde were used at this plant. Formaldehyde (0·01 per cent) used to be used as a fungicide, but this is no longer used.

The cakes then pass on the monorail into the SCA or to the mat plant (MP). In the SCA glass fibre may be chopped into 3–25 mm lengths, air textured or several strands may be wound cylindrically. In the MP, chopped strand matting is made. All these procedures in the SCA and MP lead to the production of fine particles of airborne glass fibre (fly).

The SCA was humidified by ten pairs of wall-mounted spinning disc humidifiers each of which had a small water reservoir which is refilled by a cistern. The purposes of humidification were:

i. to decrease the level of fly;
ii. to decrease the static electricity on the cakes;
iii. to achieve a relative humidity (RH) of approximately 50 per cent.

The humidifiers were cleaned every 6–8 weeks although some of the more inaccessible ones had not been cleaned for up to 4 months. All were contaminated with a green-grey slime.

The two main groups of workers in the factory were the 'filament end' and the 'strand conversion' workers. Other groups include monorail operators, packers (in the warehouse), mat plant workers, dressing mixers, batch mixers, furnace operators, glass research and clerical staff. The last four groups spend little time on the shop floor and are a 'low exposure group'. Dressing mixers and filament end workers are exposed to the wet dressing as well as fume from the heated dressing in the oven. Most other workers are exposed to fume from the oven and fly.

Clinical Study

The questionnaire used enquired into nasal, eye and throat symptoms, chest tightness, shortness of breath and wheezing and their relationship or otherwise to work. A symptom was considered to be work related if it:

a. had developed or become worse since working in the fibre glass factory;
b. improved over weekends and/or holidays.

There was also a question concerning symptoms of HF. Smoking habits, occupational history and the presence or absence of previous hay fever, asthma and chronic bronchitis were also noted. Simple spirometry using a bellows spirometer (Vitalograph) was performed, height was measured and a serum sample obtained.

Antigen Preparation

Samples of water, slime and dust were taken from the humidifiers and freeze dried extracts were prepared. The slime extract was found to be the most highly antigenic by standard double diffusion testing in agar gel by within serum comparison.

Peak Flow Records

Subjects who answered the questionnaire in a manner which suggested they had work related chest tightness, shortness of breath, or wheezing were asked to record their peak flow rate (PFR) 2 hourly for 4 weeks. Each recorded measurement was the best of three attempts provided that the second best attempt was no more than 20 l/min below the best. If it was, three more readings were performed. The maximum, minimum and mean peak flow recordings for each 24 hour period was

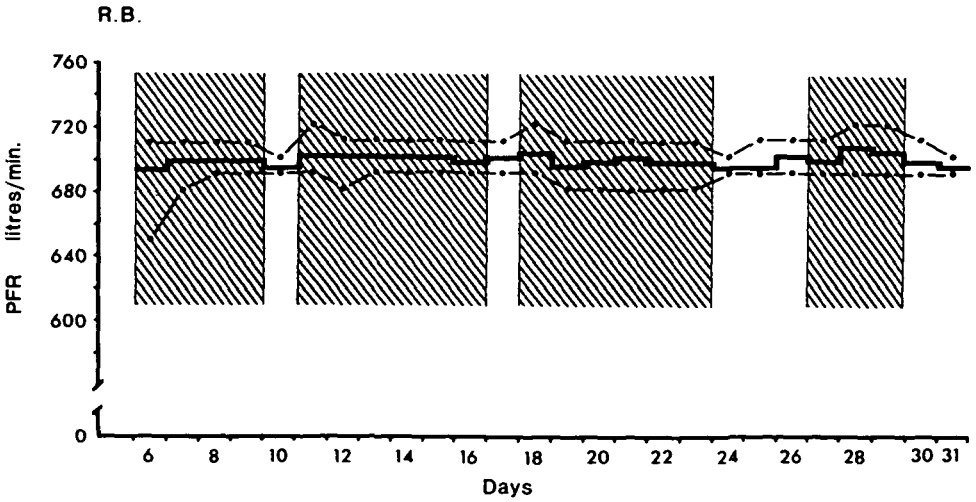

Fig. 2. Peak flow record in a worker without WRA. The upper line is the maximum, the lower line is the minimum and the central line the mean peak flow on each day. The shaded areas indicate periods at work. Usually the first 2 days are ignored to allow for the learning process. Note there is no significant difference between days at work and days away from work.

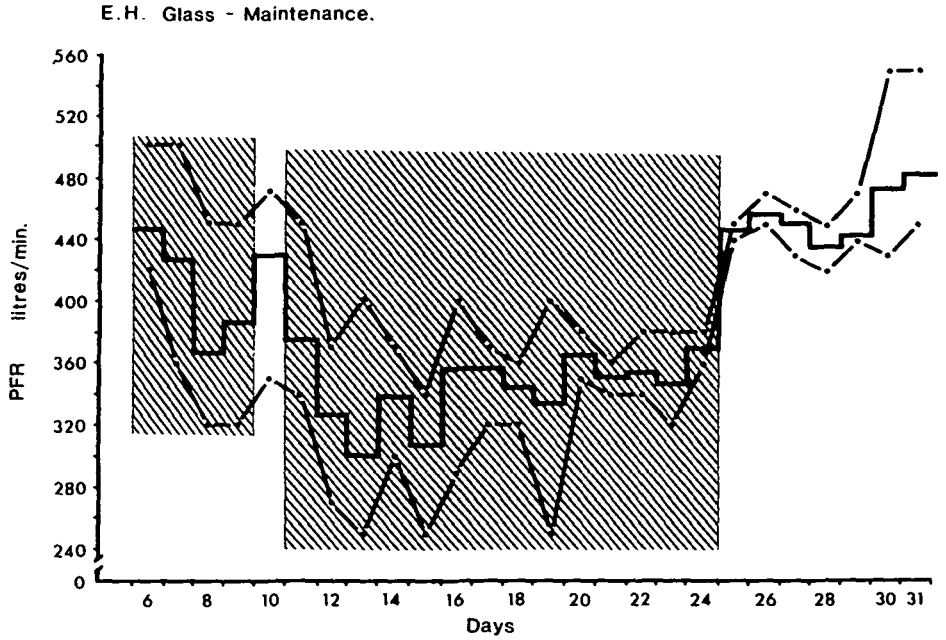

Fig. 3. Peak flow record in *Case 1*. Note the fall during working days which is reversed by a single day away from work and the considerable improvement during a week away from work.

plotted on a graph. The 24 hour period was advanced or withdrawn 12 hours if the subject changed from day to night shift or night to day shift respectively (*Figs.* 2–9).

The maximum diurnal variation in peak flow was calculated by expressing the difference between the highest and lowest peak flow each day as a percentage of the highest value. Asthma was diagnosed when the diurnal variation in peak flow reached 20 per cent or more on at least 1 day after the first 2 days of the record. Work related asthma was diagnosed if at least 75 per cent of the periods away from work were associated with a definite improvement compared to periods at work.

Skin Tests

Prick testing was also performed with several common environmental antigens. For the purposes of this study 'atopy' is defined as one or more positive skin prick tests to common environmental antigens. A positive prick test is one in which the resulting wheal measured 20 minutes after the test is at least 2 mm larger than the control.

Challenge Testing

Bronchial challenge testing was performed in hospital in four symptomatic subjects with WRA. Their peak flow records are shown in *Figs.* 3–6.

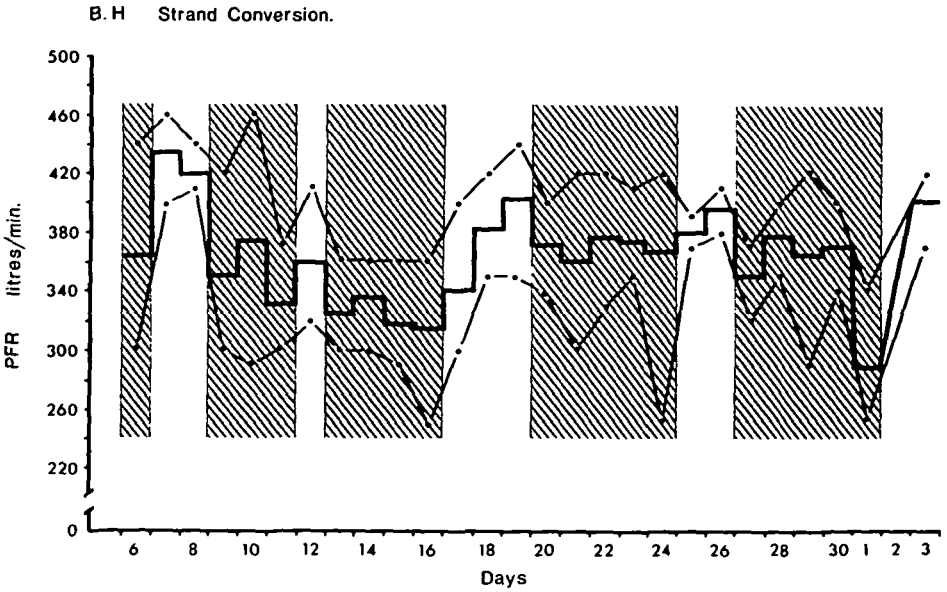

Fig. 4. Peak flow record in *Case 2*. Note the repeated pattern of deterioration at work with subsequent improvement away from work. These readings were performed after use of the humidifiers had been discontinued.

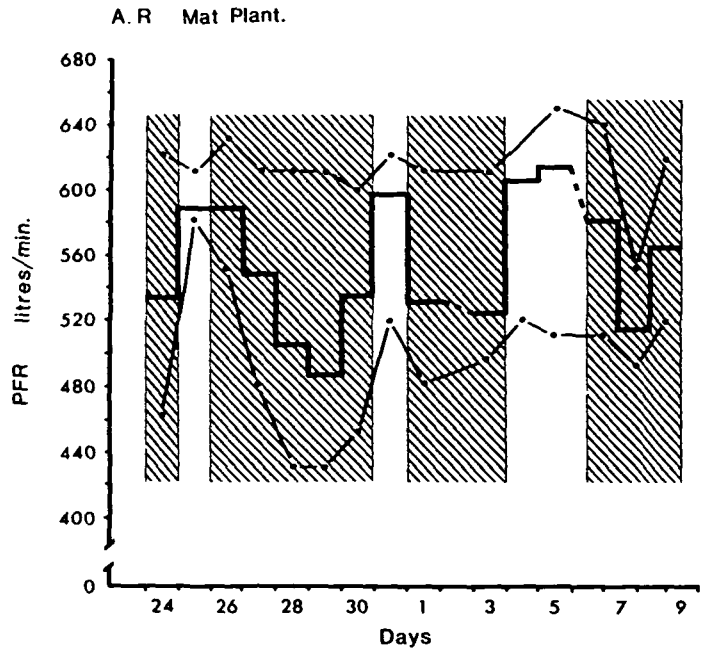

Fig. 5. Peak flow record in *Case 3*. These readings were performed while working on the mat plant.

1. CHALLENGE WITH FUME. Previously unheated glass fibre coated with various dressings was heated to 130 °C in a challenge chamber and the fume allowed to collect for 90 minutes before starting the challenge. Subjects were exposed for either 1, 5, 10 and 15 minutes (31 minute challenge) or 5, 10, 15 and 30 minutes (60 minute challenge) to the fume with 10 minute gaps between each exposure period.

2. CHALLENGE WITH FLY. Subjects were exposed to airborne glass fibre fly for two periods of 30 minutes separated by 10 minutes.

3. CHALLENGE WITH DRESSING. This was performed by either decanting the dressing for 10, 20 or 30 minute periods separated by 10 minutes or by being exposed to sprayed dressing for periods of 1, 1·5 and 5 minutes, each separated by 5 minutes.

4. CHALLENGE WITH HUMIDIFIER ANTIGEN. This was performed using 1 mg/ml or 10 mg/ml concentrations of the freeze dried humidifier slime in Coca's solution in a Wrights nebulizer. For each concentration, 1 ml of solution was nebulized.

Each challenge started at 8.30 a.m. Spirometry was monitored until 10 p.m. in each case. No patient was challenged more than once on any day. A 'blank challenge' using the same oven at the same temperature was performed in every case. In the blank challenge the oven was empty.

5. HISTAMINE CHALLENGE. This was performed using a standardized method (Cockcroft et al., 1977).

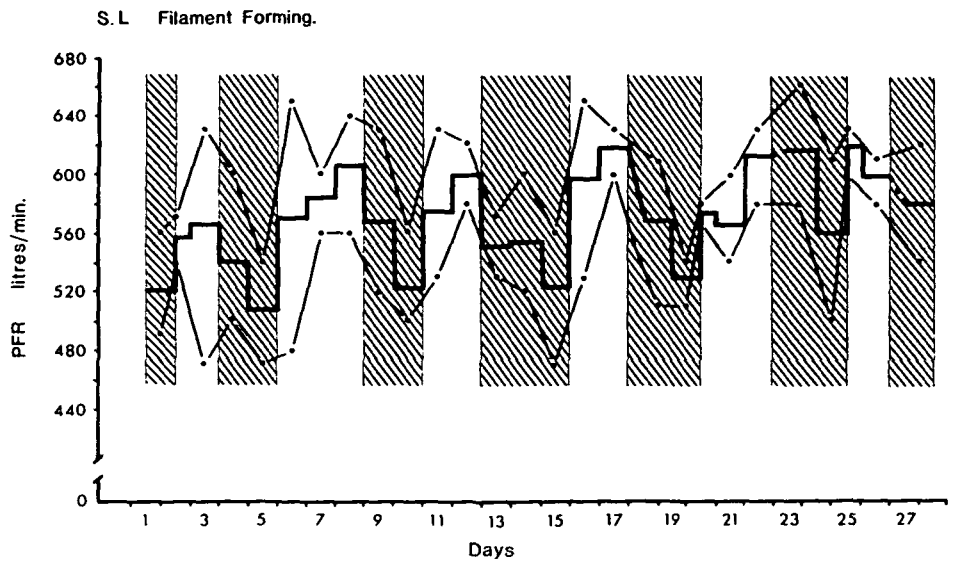

Fig. 6. Peak flow record in *Case 4*. A repeated work related pattern is shown.

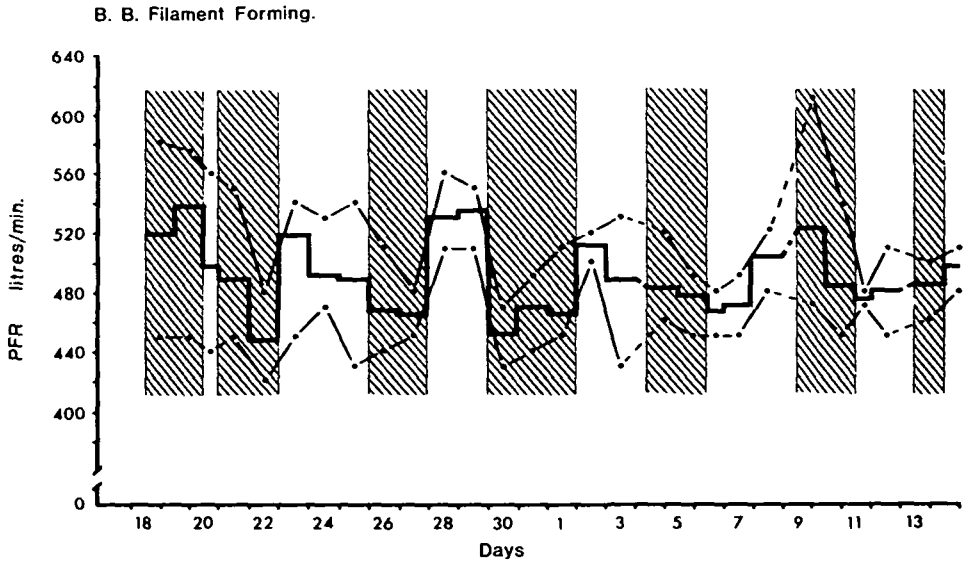

Fig. 7. Peak flow record in *Case 5.*

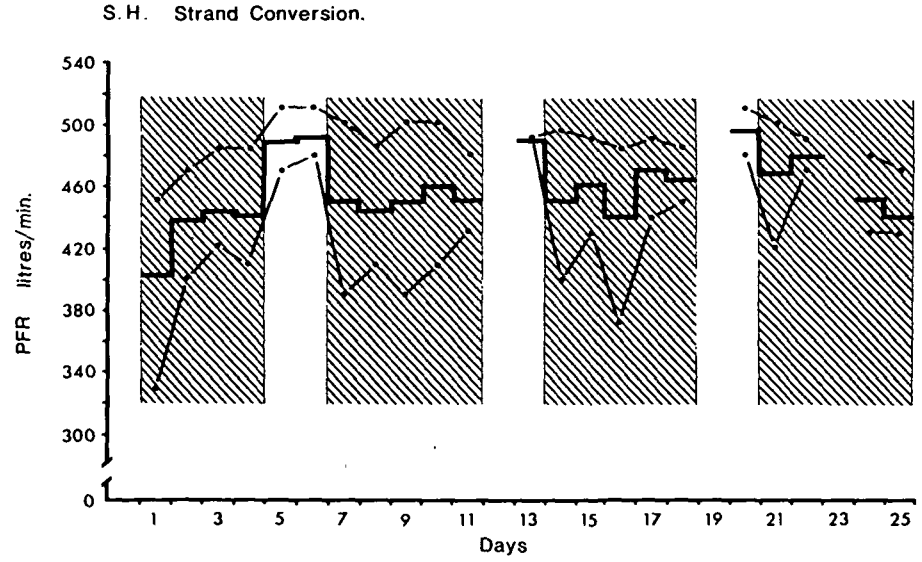

Fig. 8. Peak flow record in *Case 6.*

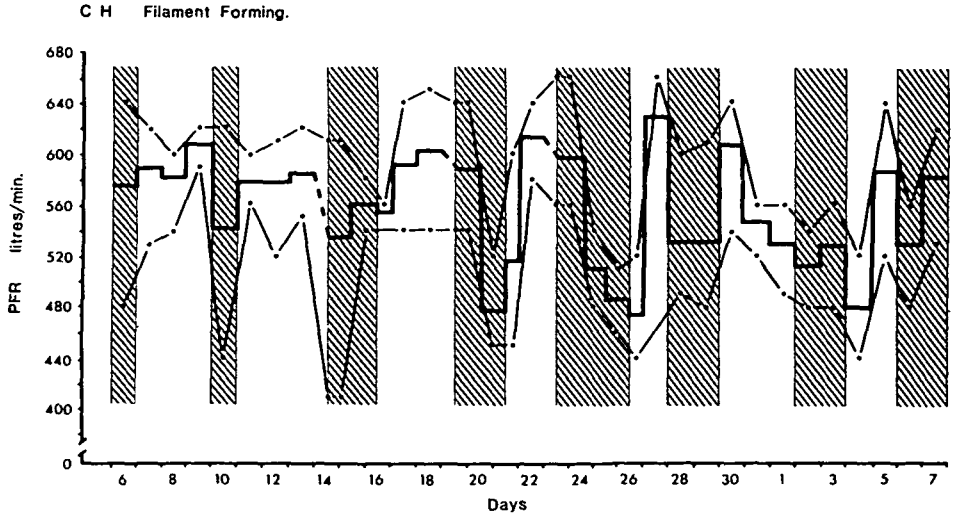

Fig. 9. Peak flow record in *Case 7.* He is a known asthmatic. Although he is shown on occasion to deteriorate at home, the predominant pattern is to deteriorate at work and improve away from work.

6. CONTROL CHALLENGE. This was performed using an atopic, non smoking asthmatic who does not work at the fibre glass factory. He has increased bronchial reactivity (PC 20=1·1 mg histamine/ml).

Remedial Action

Since the person whose symptoms originally led to the study had HF, management had ordered the humidifiers to be steam cleaned every 2 weeks but even this was not sufficient to prevent moderate contamination and they were therefore removed.

Results

The study was open to the entire workforce of the glass plant. We were able to see 216 of the 257 (83 per cent) permanent shop floor workers. Twenty-one others were seen who were never or only infrequently on the shop floor. These included clerical and glass research staff, batch mixers (who mix the raw materials) and furnace operators (the low exposure group).

Of the 235 workers interviewed, 40 gave questionnaire answers suggesting work related asthma (chest tightness, shortness of breath or wheeze at work, improving away from work), and each of these was asked to perform peak flow recordings. Three of these failed to produce any recordings and 5 produced inadequate recordings.

The remaining 32 records were assessed and 7 cases of WRA were identified. (For details, see *Figs*. 3–9 and *Table I*). Three worked at the filament forming end in close contact with the fibre glass dressings, 2 worked exclusively in the SCA and 1 worked in SCA when initially seen but then moved to the mat plant. One further case was a maintenance fitter who worked all over the glass plant. One of the cases from the filament forming end (see *Fig*. 9) had asthma as a child. This restarted before he worked in the glass plant, but he felt it was considerably worse at work and his peak flow recordings confirmed that observation.

Six of the 7 reported no change in their symptoms when the humidifiers were switched off. *Case 2* however reported some improvement but not resolution of her chest symptoms when humidification was ceased. Repeat peak flow recordings were performed which again showed WRA and these are shown in *Fig*. 4. Her symptoms were not suggestive of humidifier fever. The person who moved to the mat plant (*Case 3*) performed peak flow recordings for 1 month both in the SCA and while on the mat plant. His WRA continued while on the mat plant (see *Fig*. 5).

A typical case history is that of *Case 1* who is a 61-year-old fitter who has worked throughout the glass plant since 1975. He was well until 1980 when he developed a 'tight throat' and coughing at work. In 1982, he developed runny eyes and nose and sneezing at work. The runny nose and eyes and sneezing would improve over a weekend but the other symptoms only improved on holidays. He is an ex-smoker who smoked 30 cigarettes/day until 13 years ago. He takes no regular therapy. He is non atopic. The only abnormalities on lung function testing were a significantly raised residual volume (RV) and reduced V-max-60 and 75.

Table I. Details of cases of WRA

No.	Age (yrs)	Sex	Smoking habit	Atopy	History of OAD	Work site	Service before symptoms
1*	60	M	Ex	–	–	All	5 yrs
2*	34	F	15/d	+	–	SCA	<1 yr
3*	44	M	20/d	+	–	SCA	4 yrs
4*	32	M	2 oz/wk	+	–	Fil	<1 yr
5	42	M	2/d	?	–	Fil	2 yrs
6	27	F	20/d	?	+†	SCA	<1 yr
7	20	M	Non	?	+	Fil	Predate

M: Male, Fil: Filament forming, F: Female, ?: Not tested, *: Challenge test performed, OAD: Obstructive airways disease, †: This case had repeated winter bronchitis, not asthma.

Results of Challenge Testing

Cases 1 and 2 were challenged for 31 minutes with fume from glass fibre coated with a dressing containing polyvinyl acetate (PVA) as a film former. *Cases 3* and *4* were challenged for 60 minutes with fume released from a mixture of glass fibre samples coated with PVA, aliphatic and aromatic polyurethanes. All cases were challenged with glass fibre fly for 1 hour; in *Cases 1* and *2* this challenge was repeated. *Case 3* was challenged with 1 mg/ml and 10 mg/ml of humidifier extract and *Case 4* was also challenged with dressing containing an aliphatic polyurethane.

No case developed symptoms in response to the blank challenge. Only *Case 1* developed any symptoms in response to fume and these were headache and eye irritation. There was no spirometric change in any of the cases in response to fibre glass fume challenge. There was no symptomatic or spirometric response by *Case 3* to either concentration of inhaled humidifier antigen, or by *Case 4* to challenge with the aliphatic polyurethane based emulsion.

Cases 1 and *2* developed marked symptoms in response to challenge with fibre glass fly collected from the SCA. *Case 1* developed sneezing, runny nose, eye irritation and coughing, but not a 'tight throat'. His symptoms cleared rapidly after challenge. These symptoms occurred with the same challenge on the next day with no significant change in his spirometry.

Case 2 developed tight chest, coughing and sneezing during the challenge which recurred 4 hours later. Following the first 30 minutes of the challenge her FEV_1 and FVC fell by more than 20 per cent when compared with either her initial readings on that day or with readings at a comparable time on her baseline day. However after the second period of 30 minutes, her FEV_1 and FVC had returned to baseline levels. Her symptoms occurred again with the same challenge on the next day, however on this occasion they lasted for 2 hours, easing off around midday. On this second occasion her spirometry did not change significantly.

The control developed nasal discharge, sneezing and irritation of the throat which lead to coughing on exposure to fibre glass fly, but there was no significant change in his spirometry.

Discussion

Asthma has been described in textile workers working with glass fibre in two papers: Tara (Hill, 1977) describes a single case with asthma, eosinophilia and micronodular shadowing on her chest radiograph. The condition cleared on cessation of exposure, but no proof of the cause was obtained. Dadashian (Hill, 1977) described five textile workers who had asthma which was attributed to glass fibre, but other possibilities were not excluded. WRA has not previously been described in association with fibre glass manufacture. The peak flow records of the cases (see *Figs.* 3–9) confirm that the asthma is work related, with definite improvement occurring on days away from work with deterioration on return to work in a repetitive pattern. These records give no clues as to the cause of the asthma.

Work related asthma has been reported in association with a contaminated humidification system (Finnegan and Pickering, 1984) and this was a possibility we considered. However, six of the seven cases reported no improvement in their symptoms when the humidifiers were switched off and the case who reported some improvement was shown by repeat peak flow recordings to have an ongoing WRA (see *Fig.* 4). Further three of the seven cases work at the filament forming end which is not humidified, and they must be minimally exposed to humidified air. We feel therefore that the humidifiers were not the cause of the WRA.

Bronchial challenge testing in the four workers chosen has not conclusively determined the cause of the WRA, although it has excluded some of what seemed to be the more likely possibilities. The reaction in *Cases 1* and *2* to glass fibre challenge was to reproduce almost exactly their symptoms at work. *Case 1*, however, did not experience his 'tight throat' sensation. The control also developed rhinitic symptoms in response to the fly. Further, the low exposure workers did not suffer from work related rhinitis at all. It seems likely therefore that airborne fly is causing the work related rhinitis complained of by many of the workers in the SCA (see *Table II*) and that this is an irritant response. The rhinitis among the filament end workers was thought by them to be due to the dressings on the glass. We have not been able to confirm this but it is interesting to note that *Case 4* had no rhinitic reaction to challenge with fly. Further there is little airborne fly at the filament end compared to the SCA.

The significant fall in FEV_1 and FVC which occurred in *Case 2* was probably an irritant reaction since it reversed after a further 30 minute challenge with fly. Further, this spirometric change was not reproduced by an exactly similar challenge the next day. It seems unlikely therefore that glass fibre fly is the cause of the WRA.

Table II. Prevalence of work related rhinitis among SCA workers/packers filament end and low exposure workers

	Number with rhinitis	*Percentage with rhinitis*
SCA/packer $n=96$	19	20
Filament $n=83$	24	29
Low exposure $n=21$	0	0

Because of the many chemicals used in the factory and the innumerable ways they may be combined, we cannot regard our challenge investigation as exhaustive. There were numerous other challenges which one could have performed, but these were impractical. Consequently the cause of the WRA in this continuous filament glass fibre plant remains unknown.

Acknowledgements

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Requests for reprints should be addressed to: Dr M J Finnegan, Department of Clinical Pharmacology, Clinical Sciences Building, Leicester Royal Infirmary, Leicester.