Occupational asthma due to oil mists

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ABSTRACT Twenty five patients who were exposed to oil mists at their place of work were investigated for possible work related asthma. Serial peak expiratory flow recordings showed 13 to have definite work related asthma, seven equivocal work related asthma, and three asthma unrelated to work; two had normal recordings. Subjects with work related asthma often produced different patterns of peak flow response during the working week; patterns also varied between patients. Six of these patients had bronchial tests with oil from their place of work. Three had asthma induced by exposure to unused (clean) soluble oil and one reacted to used but not to clean oil. The challenge tests in the remaining two gave inconclusive results. It is concluded that occupational asthma due to oil mists is common, the peak flow response is heterogeneous, and the provoking agent within the oil may vary from worker to worker.

Many workers, particularly in machine shops, are occupationally exposed to oil mists. These may occur in high concentrations and often more than one oil is in use. Industrial oils are in common use as coolants, cutting fluids, and lubricants. Three major classes of oils are used: (a) mineral oil, which may simply contain an extreme pressure additive; (b) emulsified mineral oil, which besides mineral oil and water contains emulsifiers, corrosion inhibitors, germicides, colourants, anti-foamers, extreme pressure additives, and perfumes (reorderants); (c) synthetic oils, which are also mixed with water and which contain the same additives as the emulsified oils but with the mineral oil replaced by a “synthetic oil,” such as a polyglycol. Oil diluted with water is often referred to as suds oils. The ultimate composition of the oil mist produced in the industrial setting depends on the type of oil used and the contamination that occurs with use, both from the metal that is being cut and from microbiological contamination, the latter being a particular problem with suds oil.

Exposure to oil mists at work can give rise to several different health problems. Reports of respiratory problems related to the inhalation of oil were initially limited to the description of lipoid pneumonia, although the occurrence of this condition is probably rare. Some reports have suggested an association with pulmonary fibrosis, but the evidence for this is inconclusive. Oil mists have also been proposed as a possible cause of lung cancer, but this is unlikely. Despite the fact that respiratory symptoms are more common in workers exposed to oil mists and are related to the ambient concentration of oil aerosol, only one survey so far has shown a reduction in spirometric values in exposed workers. This was apparent only when results were compared with predicted values, there being no significant difference between oil mist exposed and non-exposed workers. Other studies showed no deterioration in single spirometric measurements. We have previously reported a case of occupational asthma induced by oil mists. We now report our findings in 25 workers exposed to oil mists with symptoms suggesting work related asthma. Six of these workers had a bronchial provocation test with the oil they used at work.

Methods

We studied 25 patients exposed to oil mists at work and referred to an occupational respiratory clinic with asthmatic symptoms that improved on days away from work. None was identified by epidemiological survey. Details were obtained of the type of jobs associated with exposure to oil mists and the nature of the oils used. Length of exposure to oil mists and the time to the onset of the first symptoms were also recorded. Forced expiratory volume in one second (FEV1) and forced vital capacity (FVC) were measured with a dry wedge spirometer (Vitalograph) and expressed as a percentage of the predicted values. Skin prick tests were carried out with three common
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Table 1  Details of workers exposed to oil mist, grouped according to the relationship of asthma to work

<table>
<thead>
<tr>
<th>Subject No</th>
<th>Sex</th>
<th>Age (y)</th>
<th>Job</th>
<th>Oil type</th>
<th>Length of exposure (y)</th>
<th>Time to first symptoms (y)</th>
<th>Initial FEV₁ (% predicted)</th>
<th>FVC (%)</th>
<th>Peak flow pattern</th>
<th>IgE (IU/ml)</th>
<th>Smoking history</th>
<th>Skin test result</th>
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<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>67</td>
<td>Setter</td>
<td>Suds</td>
<td>43</td>
<td>33</td>
<td>60</td>
<td>105</td>
<td>Progr 112</td>
<td>315</td>
<td>Non</td>
<td>+</td>
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<td>2</td>
<td>M</td>
<td>37</td>
<td>Turner</td>
<td>Suds/min</td>
<td>18</td>
<td>17</td>
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<td>121</td>
<td>1st day</td>
<td>40</td>
<td>Curr</td>
<td>-</td>
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<tr>
<td>3</td>
<td>F</td>
<td>44</td>
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<td>0.25</td>
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<td>Ex</td>
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<td>64</td>
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<td>Suds</td>
<td>45</td>
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<td>45</td>
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<td>1500</td>
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<td>+</td>
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<td>Suds</td>
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<td>4</td>
<td>50</td>
<td>74</td>
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<td>36</td>
<td>Setter</td>
<td>Suds</td>
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<td>8</td>
<td>102</td>
<td>112</td>
<td>Progr 45</td>
<td>45</td>
<td>Ex</td>
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<tr>
<td>7</td>
<td>M</td>
<td>55</td>
<td>Manager</td>
<td>Suds</td>
<td>3</td>
<td>1.5</td>
<td>80</td>
<td>102</td>
<td>Progr 115</td>
<td>115</td>
<td>Ex</td>
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<tr>
<td>8</td>
<td>M</td>
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<td>15</td>
<td>12</td>
<td>85</td>
<td>85</td>
<td>1st day NA</td>
<td>NA</td>
<td>Curr</td>
<td>NA</td>
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<tr>
<td>9</td>
<td>M</td>
<td>49</td>
<td>Cutter</td>
<td>Suds</td>
<td>16</td>
<td>14</td>
<td>68</td>
<td>82</td>
<td>Progr 45</td>
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<td>Suds</td>
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<td>20</td>
<td>30</td>
<td>64</td>
<td>Progr 55</td>
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<td>12</td>
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<td>Various</td>
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<td>0</td>
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<td>102</td>
<td>1st day 625</td>
<td>625</td>
<td>Ex</td>
<td>NA</td>
</tr>
</tbody>
</table>

Table 2  Results of bronchial provocation tests on six subjects

<table>
<thead>
<tr>
<th>Subject No</th>
<th>Challenge material</th>
<th>Concentration (%)</th>
<th>Exposure (min)</th>
<th>Time (min)</th>
<th>% fall</th>
<th>Immediate</th>
<th>Late</th>
<th>Time (h)</th>
<th>% fall</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Superedge 4 suds</td>
<td>100</td>
<td>30 (stirred)</td>
<td>60</td>
<td>23</td>
<td>No reaction</td>
<td>6</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Duracut (mineral)</td>
<td>100</td>
<td>30 (stirred)</td>
<td>25</td>
<td>42</td>
<td>No reaction</td>
<td>7</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Saline</td>
<td>0.9</td>
<td>5 (neb)</td>
<td>0</td>
<td>2</td>
<td>No reaction</td>
<td>3</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Saline</td>
<td>0.9</td>
<td>5 (neb)</td>
<td>0</td>
<td>21</td>
<td>No reaction</td>
<td>8</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Unused cleargde</td>
<td>100</td>
<td>40 (neb)</td>
<td>15</td>
<td>4</td>
<td>No reaction</td>
<td>8</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Unused cleargde</td>
<td>100</td>
<td>30 (hot stirred)</td>
<td>0</td>
<td>11</td>
<td>11</td>
<td>9</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Unused cleargde</td>
<td>1</td>
<td>5 (neb)</td>
<td>15</td>
<td>1</td>
<td>11</td>
<td>21</td>
<td>13</td>
<td>11</td>
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</tbody>
</table>

Allergens, house dust mite, grass pollen, and cat dander, and total IgE was also determined.

All patients were asked to record their own peak expiratory flow measurements two hourly from wakening to sleeping, using a Wright’s mini peak expiratory flow meter. Three readings were made on each occasion and the best two had to be within 20% of each. Jobs carried out and treatment taken were also recorded daily. Records were plotted and initially assessed for the presence of asthma (diurnal variation in peak expiratory flow rate $\geq 20\%$). If asthma was present its relationship to work was defined as being either definite (work related changes in over 75% of working weeks), equivocal (work related changes in 25–75% working weeks), or absent (work related changes in less than 25% of working weeks). In those

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<td>No reaction</td>
<td>8</td>
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<td></td>
</tr>
<tr>
<td>6</td>
<td>Unused cleargde</td>
<td>100</td>
<td>30 (hot stirred)</td>
<td>0</td>
<td>11</td>
<td>11</td>
<td>9</td>
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<td></td>
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Neb—nebulised.
with definite or equivocal work related asthma the pattern of fall in mean peak flow throughout the working week was classified into one of four previously defined groups: first day worse (fig 1), midweek worse (fig 1), progressive weekday deterioration (fig 2) or an equivalent weekday deterioration.

**Bronchial Provocation Tests**

Six patients with definite work related asthma underwent bronchial provocation testing. The results in two cases are inconclusive.

**Case histories**

**Subject 1**

This man was described in the first report of a case of asthma due to oil mists.\(^8\) He was a toolsetter exposed to suds oil (table 1). Peak flow recordings showed a progressive deterioration throughout the working week with gradual improvement during a prolonged period away from work. Bronchial provocation tests were carried out with hard metals, emulsified oil (both used and clean) used at work, and several separate oil constituents. All oil challenges were carried out with stirred oil only. Appreciable reactions occurred in response to the emulsified oil (table 2). Subsequent challenges with the constituents showed a positive reaction to the reoderant containing pine oil.

**Subject 2**

A 37 year old machine tool operator had asthma that improved during holidays and was worse on work days, particularly on the first and second days back at work after a holiday. In his work he did metal turning, using concentrated mineral oil that produced little mist. He associated symptoms with the operation of a nearby bar grinder, which used a suds oil and produced a discernible mist and smell, particularly after a period of disuse. An assessment of his usual work exposure to oil mists was made by carrying out personal sampling with the patient in his usual place of work. The total oil concentration was 0.66 mg/m\(^3\)—three quarters of which was the soluble oil from the bar grinder. Peak flow recordings confirmed work related asthma (fig 1). The asthma resolved during the working weeks when he was no longer exposed to suds oil from the bar grinder but was still exposed to the mineral oil aerosol.

Bronchial provocation tests were performed with the mineral oil from his turning machine, and with clean and used suds oil from the nearby bar grinder. Culture of this oil produced a heavy growth of several organisms, including a *Klebsiella* species, a *Bacillus*
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Fig 3  Percentage change from baseline for FEV₁ in subject 2 before and after inhalation challenges (shaded area). The dotted line shows FEV₁ after five minutes nebulised challenge to 2% unused suds oil. The continuous line is the FEV₁ after a 15 minute nebulised challenge to 2% used suds oil.

species, and a Proteus species. The suds oil was diluted from its working concentration to concentrations of 1% and 2% by volume. A steady baseline FEV₁ was achieved before each provocation test. Initial tests were carried out with the undiluted mineral oil stirred for 30 minutes. He was subsequently challenged with the suds oil nebulised via an air driven Wright's nebuliser (see table 2). After an initial exposure for one minute spirometry was performed for 10 minutes, and if no significant fall in FEV₁ had occurred the exposure was repeated for two minutes with spirometric measurements for a further 10 minutes. The exposure was repeated again if there was no significant fall in FEV₁. An exposure of 3 mg/m² of suds oil was achieved when a concentration of 1% was being used.

After provocation testing with the 2% used suds oil solution he developed a late asthmatic reaction, with the FEV₁ falling 17% six hours after challenge (fig 3).

SUBJECT 3

A 44 year old woman machine tool operator was exposed to both a suds oil and a mineral oil at work. Asthmatic symptoms were worse when she started work in the morning and again in the evening when she went home. Although symptoms improved temporarily during weekends and holidays away from work they had gradually deteriorated over four years. Peak flow recordings showed considerable asthma, with progressive deterioration over the working week and recovery during periods away from work (fig 2).

Bronchial provocation tests were performed in a similar fashion to those of subject 2 (table 2). She developed a dual response to clean suds oil with an immediate fall in FEV₁ of 21% and a delayed fall at 8 hours of 15% (fig 4) with no change after a control challenge with saline. Further challenges with some of the constituents of the clean oil failed to show which agent was responsible.

SUBJECT 4

A 64 year old man had work that included both wet and dry grinding and cutting steel. The metals were cooled by a suds oil. Initially his breathlessness was better at weekends; as his symptoms progressed, however, this became less noticeable, although he still improved during longer periods away from work. Spirometry showed him to have substantial airways obstruction (FEV₁ 29% predicted, FVC 45% predicted). The peak flow recordings showed small “equivalent” and “progressive” falls during the working week and a second day improvement after a day or days away from work. Provocation testing with clean suds oil at 1% produced an immediate fall in FEV₁ of 11% at 10 minutes and a later fall of 19% at 2 hours (fig 5). There was no significant change after inhalational challenge with saline (table 2).
Of the 25 patients, 13 had definite work related asthma and seven had equivocal work related asthma (table 1). Three had asthma unrelated to work, and the records were normal in the remaining two. Most of the patients with work related asthma showed various patterns of peak flow response during the working weeks. A progressive deterioration throughout the working week was the most common pattern, being present in 14 out of 20, and three patients in the "definite" group had a "first day worse" pattern.

Most of those affected were men, with a mean age of 52 (SD 12) years. All but one were engaged in or exposed to some form of machine tool operating (grinding, milling, turning). The one patient not exposed to machine tool operations, a gear box assembler, was exposed to a lubricating oil. The most common form of exposure was to suds oil alone (13/20), while 5 had mixed exposure to suds and mineral oils; only one was exposed to mineral oil alone. The mean length of exposure to oil mists was 21 (SD 16) years, with a mean latent period before the first symptoms of 16 (SD 16) years. The mean initial FEV1 and FVC as percentages of predicted were 68 (SD 27) and 87 (23) respectively. A total IgE > 70 was found in nine out of 18 tested and eight out of 16 were skin prick positive.

Discussion

Previous studies have shown a considerable number of workers exposed to oil mists to have respiratory symptoms but failed to show a significant change in the mean lung function of exposed workers (both with and without symptoms). The sensitivity of single spirometric measurements in assessing occupational asthma is known to be poor. In this study 20 out of 25 workers with work related respiratory symptoms were found to have probable or possible work related asthma. Within this group of patients we have found patterns of peak flow reaction to be heterogeneous even within the same worker. A predominant pattern of progressive deterioration in peak flow was associated with specific reactions to clean oils on bronchial provocation testing, both in subjects 3 and 4 described here and in the previously described subject 1. First day worse deterioration in peak flow was associated with a reaction only to used oil (subject 2).

Exposure to oil mists had mostly been of long duration and often there had been a considerable latent period before the onset of symptoms. Suds oil was the most common type of oil to give problems; but whether more problems are associated with this type of oil or whether it is simply the most common type of oil in use is unknown.

Reproduction by bronchial provocation tests of an oil mist exposure similar to that occurring at work is difficult. The mist of oil that occurs during work is generated when the coolant or lubricating oil falls on a hot spinning metal part. As we were reluctant to nebulise the oil directly we carried out initial challenges with stirred oils, which were warmed only if no reaction occurred. This method of challenge releases only the volatile components contained within the oil. This was successful in subject 1, who was reacting to a pine reoderant; similar challenges given to further patients, however, showed that they were reacting to another constituent, which required a set of different challenge conditions. Discernible concentrations of oil mist were found at work and these were reproduced by the use of a diluted nebulised challenge. Not all challenges were successful and further work on recreating a challenge similar to conditions at work needs to be developed. We have not been able to produce inhalational challenge tests for both hot and nebulised oils.

The present study shows that the problem of oil
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exposure at work is complex, partly because the constituents of any commercial product may be varied without change in the product name and partly because of the considerable contamination that occurs with use.

References

Occupational asthma due to oil mists.

A S Robertson, D C Weir and P S Burge

Thorax 1988 43: 200-205
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