Children’s perception of breathlessness in acute asthma

I Male, H Richter, P Seddon

Abstract

Aim—To determine whether asthmatic children who present to hospital with hypoxia perceive breathlessness less well than non-hypoxic presenters.

Methods—A total of 27 children aged 5–16 years (mean age 10) admitted with acute asthma had recordings of oxygen saturation (SO2), clinical score, forced expiratory volume in one second (FEV1), and breathlessness score (HMP) at admission and at 5, 10, 24, 48, and 72 hours after admission. Those defined as hypoxic (SO2 <92%) at admission were compared with a non-hypoxic group.

Results—Twelve children were hypoxic at admission. Compared with the non-hypoxic group they were younger (8.6 (SD 2.8) v 11.2 (2.8) y, p = 0.02), and had greater airway obstruction (FEV1, 32.5 (10)% v 54.3 (26)%, p = 0.0073, 95% confidence interval (CI) −36.9 to −6.6) yet had a trend towards less breathlessness (median HMP 4 (26)% v 3 (26)%, p = 0.062, CI −0.001 to 2.00) at admission. The hypoxic group had a smaller change in breathlessness from admission to discharge, despite a similar improvement in FEV1, reflected in a lower ratio of change in HMP to change in FEV1 (ΔHMP/ΔFEV1) (median ΔHMP/ΔFEV1 0.021% v 0.073%, p = 0.0081, CI −0.075 to −0.016). Linear regression analysis showed a strong relation between ΔHMP/ΔFEV1 and initial SO2 (p = 0.004, r = 0.54).

Conclusions—Asthmatic children who present to hospital hypoxic tend to perceive themselves as less breathless than non-hypoxic children. This may predispose to a future life threatening attack.

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Keywords: asthma; perception of breathlessness

Despite many advances in its management, childhood asthma mortality rates, in the United Kingdom, remain static at approximately 0.5 per 100 000 per year.1–5 Research into the causes of asthma deaths falls into two main categories: retrospective case reviews of actual deaths; and studies of subjects with a previous life threatening attack, who are known to have a high risk of subsequently dying from asthma.6–8

In children, deaths occur at two peak age groups: preschool children,4–8 and teenagers.12–34 In the former they occur mostly in hospital, reflecting the difficulties in treating young children. In teenagers, as with adults, many deaths occur at home or on the way to hospital.4–8

While this sometimes reflects the rapidity of onset,34 fatal attacks frequently develop over several hours.4–8 On review the latter have often been rated as avoidable,4–8 for in this age group, once effective treatment is started it is unusual for death to occur.13–15 Martin et al, in reviewing the circumstances of life threatening attacks in 30 children, found 80% had gradual onset, 17% sudden onset, and judged 83% as preventable.16 The commonest reason identified for preventable asthma deaths has been delay in seeking medical care.4–8 10–12

Recent research in adults, largely based on patients with a previous life threatening attack, has suggested that many such patients have a reduced ability to recognise the deterioration in their asthma, through a decreased ability to perceive the sensation of breathlessness.4–8 13 14 McFadden suggests: “Some patients are unable to sense the presence of even marked airway obstruction and have no symptoms until their respiratory reserve is virtually exhausted. Thus when they report breathlessness and wheezing, they may be close to death.5”

As yet there is little research into perception of breathlessness in children. The aim of this study was to determine whether poor perception of breathlessness could be a factor in children presenting to hospital with a severe attack. We used the presence of significant hypoxia (oxygen saturation (SO2) <92%) at admission as an indicator of a severe, and potentially life threatening, asthma attack. Geelhoed et al have shown that an initial SO2 <92% at hospital admission predicts a severe attack, likely, for example, to require intravenous therapy.17 Our hypothesis was that children with poor perception of breathlessness are more likely to present to hospital having already developed significant hypoxia.

Methods

Children (aged 5–16 years) admitted to the Royal Alexandra Children’s Hospital in Brighton from September to December 1995 were entered into the study and assessed on repeated occasions: soon after admission (0 hours), and then at 5, 10, 24, 48, and 72 hours or until discharge. An initial history recorded details of age, sex, length of acute illness, including any delays in presentation, previous need for intensive care or intravenous therapy, source of referral, severity of chronic symptoms, and acute and chronic treatment. A score was given for severity of chronic symptoms and level of chronic treatment, based respectively on the Global Initiative for Asthma (GINA)1 and the 1997 British Thoracic Society (BTS) asthma guidelines for adults and schoolchildren.16 The chronic symptom score allocated was 1 for GINA stage “intermittent” up to 4 for GINA stage “severe persistent”. The treatment score allocated was equivalent...
Changes in lung function parameters (% predicted) and SaO₂ (%) during admission, for the total study group.

### Table 1  Characteristics of the total, hypoxic, and non-hypoxic study groups

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Total</th>
<th>Hypoxic</th>
<th>Non-hypoxic</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>10.0 (SD 3.0)</td>
<td>8.6 (SD 2.8)</td>
<td>11.2 (SD 2.8)</td>
<td>p = 0.02</td>
</tr>
<tr>
<td>Median AHMP/AFEV₁, (%−1)</td>
<td>0.042</td>
<td>0.021</td>
<td>0.073</td>
<td>p = 0.0081</td>
</tr>
<tr>
<td>Male sex</td>
<td>16/27</td>
<td>8/12</td>
<td>8/15</td>
<td>p = 0.48</td>
</tr>
<tr>
<td>Length acute illness (h)</td>
<td>62 (101)</td>
<td>45 (44)</td>
<td>76 (130)</td>
<td>p = 0.45</td>
</tr>
<tr>
<td>Median chronic treatment score</td>
<td>2 (1–4)</td>
<td>2 (1–3)</td>
<td>2 (1–4)</td>
<td>p = 0.27</td>
</tr>
<tr>
<td>Self referral rate</td>
<td>21/27</td>
<td>9/12</td>
<td>12/15</td>
<td>p = 0.76</td>
</tr>
<tr>
<td>Median chronic severity score</td>
<td>2 (1–4)</td>
<td>2 (1–4)</td>
<td>3 (1–4)</td>
<td>p = 0.71</td>
</tr>
<tr>
<td>Previous ICU/IV therapy</td>
<td>2/26</td>
<td>2/11</td>
<td>0/15</td>
<td>NS</td>
</tr>
</tbody>
</table>

Clinical score as developed and validated by Wood and colleagues20 21 was then assessed. With this a maximum score of 10 is obtainable, representing clinical signs of severe asthma. SaO₂ was then recorded by pulse oximetry (Biox 3700e, pulse oximeter, Ohmeda, Louisville, USA). At the same time, spirometry, including peak expiratory flow (PEF), forced expiratory volume in one second (FEV₁), and maximal mid expiratory flow (MMF)—a measure of small airway function—was recorded using a pneumotachograph spirometer (Master Screen Pneumo 4.2, Erich Jaeger, Wurzburg, Germany), and expressed in all cases as percent predicted for height.

Mean values for each of the observations were calculated for the total study group at admission (0 hours), and at 5, 10, 24, 48, and 72 hours after admission, and plotted graphically. Initial HMP score, FEV₁, and the ratio of initial HMP to initial FEV₁ for the hypoxic and non-hypoxic groups were compared using the Mann–Whitney U test. The relation between the ratio of initial HMP to initial FEV₁ and initial SaO₂ was also assessed, using linear regression.

The ratio of change in HMP score to the change in FEV₁ between first and last assessments (ΔHMP/ΔFEV₁) was then calculated to assess the individual subject’s ability to perceive changes in airway obstruction. The change was that between the first assessment (0 hours) and the final assessment before discharge: mean interval (SD) 32 hours (21.5). Multiple regression analysis was used to examine the possible relation between ΔHMP/ΔFEV₁ and SaO₂ and additionally age, length of acute illness, chronic asthma score, and chronic treatment level. The Mann–Whitney U test was used to compare ΔHMP/ΔFEV₁ in the hypoxic and non-hypoxic groups. These were further compared with the results of 14 children (mean age 10.5 years) with mild asthma in whom the ΔFEV₁ was induced by a histamine challenge as part of the previous study validating the breathlessness scale (mild asthma group).

Ethical approval was obtained from the Brighton Area Ethics Committee. Signed, informed consent was obtained from parents and, when appropriate, the child.

### Results

A total of 27 children were recruited with mean age 10 years. Of these 12 had significant hypoxia (SaO₂ <92%) at admission. The hypoxic group were younger (8.6 (SD 2.8) years) v 11.2 (2.8) years, p = 0.02, but otherwise both groups were similar (see Table 1).

Mean values for larger airway obstruction, as indicated by PEF and FEV₁, improved rapidly in the first 10 hours after admission (see fig 1).
but more slowly thereafter. Clinical score showed a similar pattern of improvement (see fig 2). Small airway obstruction, reflected by MMF, was slow to improve, reached a plateau, and was still below 50% predicted at 72 hours. $\text{SaO}_2$, which in part also reflects small airway function, was similarly slow to improve. It is notable that improvement in perceived breathlessness (increasing HMP score in fig 2) mirrors the pattern of large airway parameters, continuing to improve between 20 and 72 hours, when MMF and $\text{SaO}_2$ were static.

Comparison of the hypoxic and non-hypoxic groups at admission showed, as one would expect, significantly worse lung function in the hypoxic group (mean $\text{FEV}_1$, as percent predicted, 32.5 (SD 10)% vs 54.3 (26)%; $p = 0.0073$, CI $-36.9$ to $-6.6$). Despite this, there was a trend for the initially hypoxic children to report less initial breathlessness ($\text{median HMP} = 4$ vs $3$, $p = 0.062$, CI $-0.001$ to $2.00$, see fig 3). The HMP/$\text{FEV}_1$ ratio was higher in the hypoxic group (median $0.021\%^{-1} \pm 0.073\%^{-1}$, $p = 0.0081$, CI $-0.075$ to $-0.016$, see fig 4), suggesting that they were less aware of changes in airflow obstruction. The hypoxic group also had a significantly lower $\Delta \text{HMP}/\Delta \text{FEV}_1$ than the mild asthma group (median $0.021\%^{-1} \pm 0.097\%^{-1}$; $p = 0.0001$, CI $-0.11$ to $-0.042$) while the non-hypoxic and mild asthma groups were similar ($p = 0.12$).

Linear regression analysis showed a significant, positive relation between $\Delta \text{HMP}/\Delta \text{FEV}_1$ and initial $\text{SaO}_2$ ($r = 0.54$, $p = 0.004$, see fig 5). This relation persisted ($p = 0.011$) on performing multiple linear regression analysis, although no relation could be shown with age ($p = 0.15$), chronicity score ($p = 0.65$), length

Figure 2 Changes in HMP score and clinical score during admission, for the total study group.

Figure 3 Initial HMP score plotted against initial $\text{SaO}_2$ (%).

Figure 4 $\Delta \text{HMP}/\Delta \text{FEV}_1$ (%$^{-1}$) ratios for the hypoxic, non-hypoxic, and mild asthmatic (validation) groups. Individual values and median are shown.
Figure 5  $\text{AHMP}/\Delta \text{FEV}_1$, (%$^\text{c}$) ratio plotted against initial $\text{SaO}_2$ for the total study group. The regression line is shown.

Discussion

This study showed that, overall, children were able to recognise and report decreasing breathlessness as measures of larger airway function improved. This suggests that the HMP scale enables children to report breathlessness appropriately. Changes in clinical score followed a similar pattern. Small airway obstruction, measured by MMF and reflected by $\text{SaO}_2$, persisted even as the child appeared to be, and felt, better, as has previously been recognised both following an acute attack and in asymptomatic asthmatics.

However, those children who were hypoxic at presentation to hospital, appeared to have reduced perception of breathlessness. They had a trend towards reporting lower basal levels of breathlessness despite significantly greater airway obstruction, and experienced a smaller decrease in breathlessness felt for a similar improvement in $\text{FEV}_1$, in comparison with children presenting without hypoxia and with mild asthmatics not admitted to hospital. This observation is supported by the direct relation between ability to perceive breathlessness and initial $\text{SaO}_2$, such that the greater the degree of initial hypoxia, the less able the child was to perceive breathlessness. This is also consistent with studies in adults showing that those presenting with life threatening asthma attacks perceive breathlessness poorly, and has been reported in two children who died from acute asthma.

Our findings provide evidence in support of the hypothesis that differences in perception of breathlessness occur among asthmatic children, and influence the severity of presentation to hospital. If this is true, where could these differences in perception arise from? They may simply be caused by a normal distribution of perception of breathlessness. There is evidence for a genetic influence, for example in family studies of asthmatics with a history of respiratory failure, or in natives of high altitude regions and endurance sportsmen where this may confer an evolutionary advantage. However, other studies suggest poor perception of breathlessness may be associated with hypoxic ventilatory response, such as chronic persistent asthma or congenital cyanotic heart disease. Medical intervention, such as carotid body resection, may also have an influence. If poor perception is acquired it could also be possible to reverse it, for example by aggressive management of chronic airway obstruction.

In summary, this study suggests that reduced perception of airway obstruction and breathlessness may be associated with hypoxic presentation to hospital during acute asthma. As research in adults suggests poor perceivers of breathlessness are at risk of dying from a future asthma attack, then presence of significant hypoxia at admission should be taken as a warning of future severe attacks, which the child may not be able to recognise. It is therefore important to measure $\text{SaO}_2$ at admission, and in those children found to be initially hypoxic, to consider regular objective measurement of airflow obstruction, for they may not otherwise be able to recognise when their asthma is deteriorating. While home peak flow measurement or spirometry may help, further research is required to determine how best to monitor such children and prevent future life threatening attacks.

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