Correspondence
Gastro-oesophageal reflux and childhood asthma: the acid test

Sir—We would like to comment on the paper by Dr N Wilson and colleagues (August 1985;40:592–7) concerning the relationship between gastro-oesophageal reflux and childhood asthma. The authors have demonstrated that some asthmatic children have increased bronchial responsiveness after swallowing acid solutions, detectable by a change in histamine PC_{20} (the concentration of histamine producing a 20% fall from baseline peak expiratory flow), but not by changes in peak expiratory flow measurements. The relationship between this phenomenon and oesophageal reflux, as measured by sleeping oesophageal pH monitoring, is less convincing.

It is well established that reflux is a normal physiological occurrence; pathological reflux occurs as a quantitative deviation from normal as measured by various parameters on extended pH monitoring. The authors have selected two of these: the occurrence of reflux (pH < 4) lasting more than two minutes and the percentage of time for which pH is < 4. Measurements of these and other parameters in normal children are few, but available data¹ ² suggest that, in some circumstances, up to 16% of the time pH < 4 may be normal. Other measurements, such as total number of refluxes with pH < 4, number of refluxes lasting more than five minutes (not two minutes as in this study), the longest period for which pH is < 4, and oesophageal clearance time, should be considered before deciding whether reflux is pathological or not. Normal values for each of these measurements are likely to change with age, relationship to food, sleep state, and posture, and have not yet been adequately established.

The authors have tried to relate reflux (as defined by their criteria) to changes in histamine PC_{20}. It is clear from figure 2 in the article that approximately equal numbers of children with and without reflux showed a significant change in PC_{20} after drinking acid. This does not support the hypothesis that pathological reflux contributes to nocturnal wheezing in the subgroup of asthmatic children who are in some way sensitive to swallowed acid.

<table>
<thead>
<tr>
<th>Change in histamine PC_{20}</th>
<th>Present</th>
<th>Absent</th>
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</thead>
<tbody>
<tr>
<td>Reflux</td>
<td></td>
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<tr>
<td>Present</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Absent</td>
<td>6</td>
<td>4</td>
</tr>
</tbody>
</table>

Finally, in order to evaluate the usefulness of the “acid test” it would be important to know whether non-asthmatic children have detectable changes in histamine PC_{20} after swallowing acid.

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* * * This letter was sent to the authors, and Drs Wilson and Silverman reply below.

Sir—We are disappointed that Dr Macfarlane and others failed to understand the point we were making about the relationship between reflux and asthma. We demonstrated that a drink of acid could, in some asthmatic children, increase bronchial responsiveness. We reasoned that if these children had a sufficient reflux to stimulate the oesophagus to a similar degree then reflux could exacerbate their asthma. We did not claim that the reflux was abnormal or pathological. Indeed, we implied that any child with asthma could be vulnerable, as “silent reflux may occur in anyone.” Neither did we claim to show any association between the degree of reflux and the bronchial response to ingested acid, as they suggest. We pointed out that, if the reflux coexisted, reflux could be relevant to symptoms of asthma. In common with other triggers of asthma, reflux occurs in normal people and asthmatics alike, but only in those whose airways respond does it become a stimulus for asthma. It would be interesting, but irrelevant to our conclusions, to assess the effect of the “acid test” in normal children.

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First Markfield Symposium on Interstitial Lung Disease

The Chicago Lung Association and Chicago Thoracic Society invite submission of papers for the First Markfield Symposium on Interstitial Lung Disease on 21 and 22 October 1986. The programme will consist of state of the art lectures on different aspects of IPF, each followed by a series of 10 minute paper presentations. Those wishing to present papers are invited to submit an abstract typed in double spacing and not exceeding one side of one page in length. The title should be in capitals. The author’s name and affiliations should be placed under the title. Abstracts should be submitted to Dr Dean Schraufnagel, Department of Medicine, University of Illinois at Chicago, PO Box 6998, Chicago, IL 60680, USA, to arrive before 1 May 1986.
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P Macfarlane, V Miller and J Couriel

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