LETTERS TO THE EDITOR

Chronic lung disease and subsequent respiratory symptoms

EDITOR—We read with interest the recent paper by de Winter et al.,1 and to determine more clearly how often chronic lung disease is associated with other respiratory symptoms, we studied a group of infants with chronic lung disease, together with a matched control group.

Sixty-nine infants born at Liverpool Maternity Hospital between January 1980 and December 1985 were identified as having chronic lung disease (having received mechanical ventilation during the first two weeks of life for more than 24 hours, and at 28 days of age requiring supplementary oxygen, associated with abnormalities of the lung parenchyma on the chest radiograph).

A respiratory questionnaire was sent by post to those 55 children who were still alive. This questionnaire was also sent to parents of a control group of children (n=42) who received mechanical ventilation for at least 24 hours within the first two weeks of postnatal life but did not develop chronic lung disease, and who were matched for sex, year of birth, and gestation to the nearest week, and birth-weight to the nearest 100 g. Following initial mailing and one repeat mailing to all non-responders (72%) of parents with infants with chronic lung disease and 24 (52%) of parents with control infants completed the questionnaire. The median age of the chronic lung disease group was 8 years (range 6-11) and that of the control group was 7 years (range 6-11).

There was no significant difference between the two groups with respect to: birth-weight (chronic lung disease 1040 g (650-2150) vs control 1080 g (858-21000)), gestation (chronic lung disease 28 (25-34) vs control 29 (25-34)), sex and parental smoking. Parental atopy, defined as asthma, eczema, or hayfever, diagnosed by a physician in the parental family, was more common in infants with chronic lung disease (52%) compared with the control group (21%) (P<0.05).

There was a higher incidence of phlegm with colds, wheeze, and effort intolerance in the chronic lung disease group compared with the control group. The incidence of respiratory symptoms in those infants who were oxygen dependent at 36 weeks postmenstrual age were even higher (table 1).

When regression analysis using each respiratory symptom as the dependent variable revealed that the increased incidence of phlegm with colds and wheeze among the chronic lung disease group was related entirely to the increase in parental atopy in this group, although effort intolerance was still independently associated with chronic lung disease (odds ratio 2.33, 95% confidence interval 1.01-9.37). We also performed logistic regression using chronic lung disease as the dependent variable and parental smoking, parental atopy, gestation, birthweight and male sex as the independent variables and found that the only significant independent predictor of chronic lung disease was parental atopy (odds ratio 2.12, 95% confidence interval 1.07-4.21). Our findings do not agree with those of de Winter et al., but support the work of Nickerson et al. who suggested that there is an increased incidence of a family history of asthma in infants with chronic lung disease.2 We used a different definition of chronic lung disease (oxygen dependency at 28 days) which may have resulted in the inclusion of a higher number of very low birthweight infants.

We have shown that the incidence of respiratory symptoms in survivors of chronic lung disease some years later is high and although chronic lung disease is a multifactorial disease, our work suggests that a family history of atopy may increase an individual's predisposition to the disease.

Table 1 Incidence of respiratory symptoms in CLD, control group, and those infants dependent on oxygen at 36 weeks postmenstrual age

<table>
<thead>
<tr>
<th>Symptom</th>
<th>CLD (n=42)</th>
<th>Controls (n=24)</th>
<th>Oxygen dependent at 36 weeks post menisural age (n=19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cough</td>
<td>16 (38%)</td>
<td>6 (25%)</td>
<td>9 (42%)</td>
</tr>
<tr>
<td>Phlegm with cold</td>
<td>30 (71%)</td>
<td>9 (38%)</td>
<td>17 (89%)</td>
</tr>
<tr>
<td>Ever wheezy</td>
<td>28 (67%)</td>
<td>9 (38%)</td>
<td>16 (84%)</td>
</tr>
<tr>
<td>Wheezy in past 12 months</td>
<td>24 (57%)</td>
<td>6 (25%)</td>
<td>15 (79%)</td>
</tr>
<tr>
<td>Asthma was used</td>
<td>19 (45%)</td>
<td>4 (17%)</td>
<td>8 (42%)</td>
</tr>
<tr>
<td>Current treatment</td>
<td>10 (25%)</td>
<td>2 (8%)</td>
<td>6 (32%)</td>
</tr>
<tr>
<td>Effort intolerance</td>
<td>15 (35%)</td>
<td>2 (8%)</td>
<td>10 (53%)</td>
</tr>
</tbody>
</table>

*P<0.05, **P<0.01 when compared with controls.

Changes in pulmonary artery pressure in infants with respiratory distress syndrome after treatment with Exosurf

EDITOR—Hamdan and Shaw1 with their confirmation of the finding of Kaasa et al.2 that surfactant therapy is accompanied by a fall in pulmonary artery pressure (PAP), have made an important contribution to the understanding not only of the mechanism of surfactant therapy, but also perhaps of one of its principal complications—pulmonary haemorrhage. If the fall in PAP is accompanied by an increase in pulmonary artery flow, and if both these changes occur, as they imply, sooner than would otherwise be expected in preterm infants with respiratory distress syndrome, this might explain the increased incidence of pulmonary haemorrhage seen in certain controlled trials of surfactant therapy.3 This might also explain two paradoxes, that the increase in pulmonary haemorrhage occurs despite a reduction in the incidence of persistent patent ductus arteriosus, and that pulmonary haemorrhage is reported to occur in babies whose condition is otherwise improving.

Their findings may also partly explain the reduction in the incidence of persistent pulmonary hypertension seen in the babies treated with surfactant, in the largest double blind placebo controlled trial.4 Discussing the possible mechanism of the fall in PAP, they speculate that Exosurf may act primarily as a pulmonary vasodilator, on the basis of the lack of change in static compliance seen with this surfactant in the study of Stensson et al.5 However, if, as many suggest, the initial effects of surfactant therapy are related to changes in functional residual capacity rather than in compliance,6 the increase in number of ventilated alveoli might itself be responsible for the fall in PAP, as well as for an increase in effective pulmonary flow (blood coming into contact with ventilated alveoli), explaining the early improvements in oxygenation. In this respect the effects of surfactant therapy have been likened to those of continuous positive airways pressure (CPAP), and it would be interesting to know if a similar fall in PAP is seen with the use of CPAP, and also what happens with the use of surfactants other than Exosurf.

BOOK REVIEWS


In parallel with the rapid changes taking place in molecular biology, technology of neonatal mechanical ventilation have leapt forward over the past few years. ECMO, high frequency conventional ventilation, and high frequency jet and oscillatory ventilation are all available to us; but technology is on the horizon. But there are few comprehensive sources of information about these
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