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 later 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 having required 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 birthweight to the nearest 100 g. Following initial mailing and one repeat mailing to all non-respondents, 42 (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: birthweight (chronic lung disease 1040 g (650–2150) *vs* control 1080 g (858–2100)), 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 either parent, 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).

Logistic 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 1.01–5.37). 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.

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1 de Winter JP, van Sonderen L, van den Anker JN, Merth IT, Brand R, van Bel F, *et al.* Respiratory illness in families of preterm infants with chronic lung disease. *Arch Dis Child* 1995; 73: F147–F52.

2 Nickerson BG, Taussig LM. Family history of asthma in infants with bronchopulmonary dysplasia. *Pediatrics* 1980; 65: 1140–4.

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

EDITOR—Hamdan and Shaw,¹ with their confirmation of the finding of Kaapa *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 Stenson *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.

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1 Hamdan AH, Shaw NJ. Changes in pulmonary artery pressure in infants with respiratory distress syndrome following treatment with Exosurf. *Arch Dis Child* 1995; 72: F176–9.

2 Kaapa P, Seppanen M, Kero P, Saraste M. Pulmonary hemodynamics after synthetic surfactant replacement in neonatal respiratory distress syndrome. *J Pediatr* 1993; 123: 115–9.

3 Rogers D. Pulmonary haemorrhage, surfactant, and low-birthweight babies. *Lancet* 1993; 341: 698.

4 Long W, Corbet A, Cotton R, Courtney S. The American Exosurf Neonatal Study Group I, The Canadian Exosurf Neonatal Study Group, *et al.* A controlled trial of synthetic surfactant in infants weighing 1250 g or more with respiratory distress syndrome. *N Engl J Med* 1991; 325: 1696–703.

5 Stenson BJ, Glover RM, Parry GJ, Wilkie RA, Laing IA, Tarnow-Mordi WO. Static respiratory compliance in the newborn. III: Early changes after exogenous surfactant treatment. *Arch Dis Child* 1994; 70: F19–24.

6 Milner AD. How does exogenous surfactant work? *Arch Dis Child* 1993; 68: 2534.

BOOK REVIEWS

New therapies for neonatal respiratory failure. A physiological approach. Edited by Bruce Boynton *et al.* (Pp 357, £65 hardback.) CUP, 1994. ISBN 0-521-43161-1.

In parallel with the rapid changes taking place in molecular biology, techniques 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: liquid ventilation is on the horizon. But there are few comprehensive sources of information about these

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

	CLD (n=42)	Controls (n=24)	Oxygen dependent at 36 weeks post menstrual age (n=19)
Cough	16 (38%)	6 (25%)	8 (42%)
Phlegm with cold	30 (71%)**	9 (38%)	17 (89%)
Ever wheezy	28 (67%)*	9 (38%)	16 (84%)
Wheezy in past 12 months	24 (57%)	6 (25%)	15 (79%)
Asthma diagnosed	19 (45%)	4 (17%)	13 (68%)
Current treatment	10 (25%)	2 (8%)	6 (32%)
Effort intolerance	15 (35%)**	2 (8%)	10 (53%)

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