LETTERS TO THE EDITOR

Indirect estimates of pulmonary artery pressure

EDITOR,—We read with interest the paper by Hamdan and Shaw showing an increase in the Doppler derived ratio of pulmonary artery acceleration time to the right ventricular ejection time (AT:RVET), an indirect estimate of pulmonary artery pressure (PAP), after the first and second dose of synthetic surfactant (Exosurf) in infants with respiratory distress syndrome (RDS).¹ This finding is not very new, as we have already shown that repeated synthetic surfactant replacements in RDS result in a fall of PAP, assessed directly with Doppler technique from tricuspid regurgitation, the reported reference method.^{2,3} Our early data indicated that the fall in PAP induced by surfactant was transient, subsiding in 12 hours.² Hamdan and Shaw suggested a persistent fall after Exosurf and they postulated that this difference compared with our data could have been due to the presence of severe pulmonary hypertension, associated with tricuspid regurgitation in our infants, leading only to a transient depressor response to surfactant.¹ It is true that tricuspid regurgitation may be more common in sick newborn infants than healthy ones,⁴ but no clearcut connection with the level of the pulmonary artery pressure has been found. On the other hand, changes in pulmonary vascular resistance are not always connected with simultaneous changes in PAP. Indeed, our new experience with Doppler derived PAP measurements, from ductal shunt flow velocity, suggest that surfactant treatment in infants with RDS may acutely decrease the pulmonary vascular resistance, but due to a subsequent increase in ductal left-to-right shunting and hence pulmonary blood flow, may result in no or only a transient reduction in PAP.³ Furthermore, our recent clinical experience suggests that in some forms of neonatal pulmonary hypertension, such as in association with septic infection, the pulmonary vasoactive disturbance may not permit an acute surfactant induced fall in PAP or pulmonary vascular resistance.

We have shown before that the systolic PAP of distressed infants declines steadily, although more slowly than in the controls, during the first days of life, and that the rate of this fall is not influenced by surfactant treatment.⁴ Although suggested, the uncontrolled data of Hamdan and Shaw do not bring any conclusive evidence of a surfactant induced, sustained fall in PAP in infants with RDS. Furthermore, estimation of PAP using their indirect method is easily influenced by other circulatory disturbances. Our experience strengthens the widely accepted view that the assessment of PAP should be preferably done from the tricuspid regurgitant or ductal shunt flow velocity. When performed repeatedly during the acute course of RDS, these assessments may contribute significantly to the medical management of prematurely born infants.

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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 Kääpä P, Kero P, Saraste M. Synthetic surfactant replacement therapy decreases estimated pulmonary artery pressure in respiratory distress syndrome. *Am J Dis Child* 1992; 146: 961–4.
- 3 Kääpä P, Seppänen M, Kero P, Saraste M. Pulmonary hemodynamics after synthetic surfactant replacement in neonatal respiratory distress syndrome. *J Pediatr* 1993; 123: 115–9.
- 4 Seppänen MP, Kääpä PO, Kero PO, Saraste M. Doppler-derived systolic pulmonary artery pressure in acute neonatal respiratory distress syndrome. *Pediatrics* 1994; 93: 769–73.

Dr Shaw et al comment:

We agree with Kääpä and colleagues that using tricuspid regurgitation and ductal velocity patterns in measuring PAP may be advantageous, in that it is possible to obtain a quantitative estimate of pulmonary artery pressure. However, these measurements are technically quite difficult and not possible in all ventilated preterm infants. A major disadvantage is that it is only possible to estimate pulmonary artery pressure in babies with measurable tricuspid regurgitation. Furthermore, if the presence of measurable tricuspid regurgitation depended merely on pulmonary hypertension then it would be impossible to make sequential measurements in babies if the PAP fell. In Kääpä and colleagues' work, only 71% of infants had measurable tricuspid regurgitation at 72 hours, dropping from 92% at 2 hours¹; in Skinner and colleagues' work only 31% had this at 37 hours, dropping from 53% at 12 hours.² By contrast, measurement of the ratio of acceleration time to right ventricular ejection time is possible in all infants and is relatively straightforward to measure using a standard ultrasound machine. Both methods have shown good correlation with invasive measurements of pulmonary artery pressure.³

The assertion that surfactant treatment in infants with RDS may acutely decrease the pulmonary vascular resistance, but due to a subsequent increase in ductal left-to-right shunting and hence pulmonary blood flow, may result in no or only a transient reduction in PAP is interesting. However, this is difficult to substantiate from the study cited.⁴ In this study PAP was determined only up to 60 minutes after surfactant treatment, and in fact had fallen during that time.

In our study we did not assess the PAP on a group of infants with RDS who did not receive surfactant treatment, who could have been used as a control group. We feel it would have been unethical to withhold surfactant treatment from infants who had developed RDS. However, our data can be compared with measurements of PAP obtained from infants studied in the era before surfactant was available.⁵ Despite the gradual and persistent 'background' fall of PAP in the infants with RDS in our study, our analyses suggest an accelerated reduction in PAP associated with administration of surfactant.

- 1 Seppänen MP, Kääpä PO, Kero PO, Saraste M. Doppler-derived systolic pulmonary artery pressure in acute neonatal respiratory distress syndrome. *Pediatrics* 1994; 93: 769–73.
- 2 Skinner JR, Boys RJ, Hunter S, Hey E. Non invasive assessment of pulmonary arterial

pressure in healthy neonates. *Arch Dis Child* 1991; 66: 386–90.

- 3 Kosturakis D, Goldberg SJ, Allen HD, Loeber C. Doppler echocardiographic prediction of pulmonary arterial hypertension in congenital heart disease. *Am J Cardiol* 1984; 53: 1110–5.
- 4 Kääpä P, Seppänen M, Kero P, Saraste M. Pulmonary hemodynamics after synthetic surfactant replacement in neonatal respiratory distress syndrome. *J Pediatr* 1993; 123: 115–9.
- 5 Evans NJ, Archer LNJ. Doppler assessment of pulmonary artery pressure and extra pulmonary shunting in the acute phase of hyaline membrane disease. *Arch Dis Child* 1991; 66: 6–11.

CRIB and performance indicators for neonatal intensive care units (NICUs)

EDITOR,—We congratulate the SE Thames group on the first risk adjusted, population based study¹ using CRIB (clinical risk index for babies). The study of 643 infants supports previous findings that CRIB is more accurate than birthweight or gestation in predicting hospital death.² This has major implications for those who adjust for birthweight or gestation when comparing neonatal intensive care unit (NICU) mortality.

The authors concluded that, as risk adjusted mortality in larger NICUs was higher than in smaller ones, either CRIB was not a sensitive indicator of performance or the larger NICUs performed badly. However, the 95% confidence intervals for relative odds of mortality between large and small hospitals were wide and overlapping. The sample may have been too small to achieve adequate power, making it inappropriate to infer that larger NICUs had excess mortality.

Regional studies are essential, but may not be sufficiently large or representative to provide results which are clear or applicable beyond their boundaries. Using the PRISM score to adjust for risk in a national, prospective, stratified, random sample of 5415 children in the USA,³ mortality was greater in teaching hospitals (relative odds of dying 1.79; 95% confidence interval (CI) 1.23–2.61), lower in hospitals with a paediatric intensivist (relative odds 0.65; 95% CI 0.44–0.95), and was not related to size. Similar research would be valuable in a national sample of United Kingdom NICUs.

We agree that other indicators of performance are needed. Nosocomial septicaemia is an important outcome of neonatal care which may lead to increased mortality, costs, and length of stay, and is independently related to initial illness severity as measured by SNAP (score for neonatal acute physiology).⁴ Cerebral impairments and subsequent disabilities are not always outcomes of neonatal care, as some originate before birth.⁵ Comparisons of NICU performance will therefore require adjustment for the risks, estimated shortly after birth, of subsequent impairment or disability.⁵ Although CRIB was developed to predict death, it stratified risk of disability in infants <31 weeks of gestation or <1501 g in birthweight more accurately than birthweight or gestation.⁵ Gestation may have an important role in predictive models for disability, especially if survivors of <32 weeks' gestation are included, as in the SE Thames study.¹ However, we would re-emphasise our original caveat² that much larger samples are needed for comparisons between institutions.

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