

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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Dr Shaw et al comment:

We agree with Käähä 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 Käähä 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.

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

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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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- 2 The International Neonatal Network. The CRIB (clinical risk index for babies) score: a tool for assessing initial neonatal risk of death and comparing the performance of neonatal intensive care units. *Lancet* 1993; 342: 193-8.
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Neonatal pulmonary arteriovenous malformation

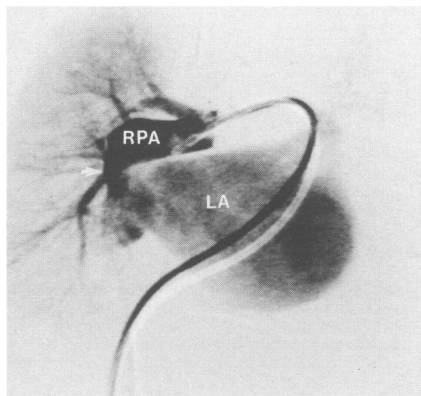
EDITOR.—Persistent central cyanosis in a neonate with structurally normal heart on cross-sectional echocardiography presents a difficult diagnostic problem.

A term neonate with central cyanosis at 30 minutes of age had an otherwise normal examination. Echocardiography in the neonatal unit revealed a normal heart, with a moderate sized arterial duct. Failure to improve oxygenation led to mechanical ventilation and in view of the possibility of persistent pulmonary hypertension treatment was started with prostacyclin infusion. At 72 hours of age the child was randomly allocated to the conventional treatment limb of the Multicentre Randomised Controlled Trial of Neonatal ECMO. He was then transferred to our unit for inhaled nitric oxide therapy, but did not improve.

At 8 days of age he had a continuous murmur over the right lower chest. Colour Doppler echocardiography showed enlargement of left atrium, and a greatly increased venous return from the right lung. There was no detectable tricuspid regurgitation. Pulmonary arterial angiography showed a large pulmonary arteriovenous malformation (PAVM) involving the right middle and lower pulmonary lobes (figure).

At surgery, the right middle and lower lobes were extensively infarcted, and were resected. The child remains well at follow up nine months later. The case underlines the importance of obtaining detailed and repeated colour Doppler echocardiographic assessments before considering ECMO for such neonates.

PAVMs are congenital, but they rarely present in a neonate. Typically multiple, there have been reports of single lesions.¹⁻³ A well, term neonate who fails the hyperoxia test and has a structurally normal heart without evidence of persistent pulmonary hypertension



Right pulmonary arteriogram (RPA) showing the AV fistula (arrow); LA=left atrium.

should be investigated further for PAVM. Doppler echocardiography may occasionally help but angiography is required to confirm and demonstrate the lesion.

Transcatheter coil embolisation has been used as primary treatment,³ but surgical resection may be necessary in some infants.

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BOOK REVIEW

Ethics and perinatology. Edited by Amnon Goldworth, William Silverman, David K Stevenson, *et al.* (Pp 484, £50 hardback.) Oxford University Press, 1994. ISBN 0-19-262379-6.

During this past decade medical ethics has become a major growth industry. Departments of medical ethics have blossomed in many of the major universities which have medical and/or legal departments and in some universities which have neither. Many universities involve religious leaders from one or more faiths to provide a viewpoint which is guided by the ethics of the founders of these various religions.

Although neonatology must be judged one of the outstanding success stories of medicine during this past decade, it has proved to be one of the more ethically troublesome areas of

medicine. The value judgments of perinatologists have been subject to challenge not least by the perinatologists themselves. As John Lantos says in the foreword to this challenging and thought-provoking treatise: 'physicians, parents, judges, insurance company executives, politicians, journalists, philosophers, and theologians have had to re-examine fundamental questions about the meaning of human community. In many contexts, and in many ways, they question whether neonatology is a dramatic success or a misguided effort'.

In addition to this foreword, there is also a preface by George Cattermole and John Goheen, and an introduction by David K Stevenson and Emlé W D Young, which together give a thematic overview. I would urge readers of the book not to bypass these three introductory contributions as they set the scene and whet the appetite for the remainder of the book. The 'meat' of the book is a series of 14 topics each of which is debated by two people. Topics include maternal-fetal conflicts, futile treatment, quality of life, epidemiological data and decision-making, withholding and withdrawing treatment and actively hastening death, organ transplantation, therapeutic and non-therapeutic research, informed consent, government regulations in the United States and United Kingdom, together with the economics of perinatal care in both countries, paediatric nursing ethics, and religious influences on decision-making. Contributors to each of these well argued and described topics include doctors, nurses, economists, legal theorists, ethicists, and philosophers.

The importance of prenatal, perinatal, and postnatal care from a cost-health-benefit perspective is discussed in detail. In most of the debates between the two contributors to each topic there is no complete agreement or consensus, which serves to highlight the need to continue debate. For example, Lewitt questions Harvey's economic justifications for expanded prenatal care, claiming that it is uncertain whether real cost savings would result. His reservations about the efficacy of prenatal care and his claims about the cost-effectiveness of such care must be further debated. The inevitable fact that economics can and does 'intrude' on the provision of perinatal care, and on the need to justify expenditure in relation to quality of life and quality of death issues, seem imponderables as great as the comprehension of infinity. However, the minds of those of us involved in perinatal care must try to grapple with these questions.

In your management strategies do you know how you move from probabilities to preferences? Are you sure that you understand how much your own upbringing and education influences your decision making? Are your decision-making values any better or any worse than someone brought up and educated within a different society and religious system? I would recommend this text as a well conceived, well constructed, and well argued synopsis of current debate, and we as paediatricians should enter into this debate and not allow it to be 'hijacked' by 'ethicists'.

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