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 the ductus arteriosus.2,3 The fall is not instantaneous, the reason for our 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 experimental neonatal pulmonary Doppler 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.5 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.

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, if 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 impossible to estimate the 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. Furthermore, if there is the presence of measurable tricuspid regurgitation then it would be impossible to measure the pulmonary artery pressure in babies with measurable tricuspid regurgitation. Furthermore, if there is the presence of measurable tricuspid regurgitation 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. Furthermore, if the presence of measurable tricuspid regurgitation depended merely on pulmonary hypertension then it would be impossible to estimate the 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 estimate the 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 estimate the 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 estimate the 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 estimate the 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 estimate the 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 estimate the 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 estimate the 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 estimate the 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 estimate the 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 estimate the pulmonary artery pressure in babies with measurable tricuspid regurgitation.

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 in a state that is different from our surfactant treated group 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.


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

EDITOR,—We congratulate the SE Thames group on the first risk adjusted, population based study using CRIB (clinical risk index for babies). The study of 643 infants supports previous findings that CRIB is more accurate than other scoring systems for 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 the mortality rate in 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 perform better.

Regional studies are essential, but may not be sufficiently large or representative to provide results which are clear or applicable beyond their boundaries.2 A new performance indicator2 for NICUs is the 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 sepsis is an important outcome of neonatal care which may lead to increased mortality, costs, and length of stay, and it is an important measure of 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.6 Although CRIB was developed to predict death, it stratifies risk into groups of <3 weeks of gestation or <1501 g in birthweight more accurately than birthweight or gestation.7 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 caveat7 that much larger samples are needed for comparisons between institutions.

WILLIAM TARNOW-MORDI
GARETH ALFORD
CRAIG GOULD
PETER FOWLIE
Department of Child Health,
Ninewells Hospital and Medical School,
Dundee DD1 9SY, Scotland
Indirect estimates of pulmonary artery pressure.

P. Kääpä, M. Seppänen and P. Kero

*Arch Dis Child Fetal Neonatal Ed* 1996 74: F79
doi: 10.1136/fn.74.1.F79

Updated information and services can be found at:
http://fn.bmj.com/content/74/1/F79.1.citation

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/