LETTERS TO THE EDITOR

Pulse oximetry in acute asthma

SIR,—I read the article by Bishop and Nolan on pulse oximetry in asthma with interest.¹ Their conclusions about the value of oximetry alone are at odds with the original work of Geelhoed *et al*, who found pretreatment arterial oxygen saturation (SaO₂) highly predictive of outcome,² but a number of factors may underlie this discrepancy.

Arterial hypoxaemia, like airway obstruction, is a characteristic feature of acute asthma, reflecting regional abnormalities of ventilation and perfusion.³ Although oxygen tension is linearly related to forced expiratory volume in one second at all levels of airway obstruction, its relationship to SaO₂ is non-linear and is reflected in the shape of the oxyhaemoglobin dissociation curve. It therefore follows that hypoxaemia accompanying mild airway obstruction may not be detected by pulse oximetry and it is possible that the poor predictive value of oximetry noted by Bishop and Nolan may be attributable to a preponderance of mild/moderate asthmatics in their sample. Unfortunately they do not provide information about selection criteria or other parameters of severity.

Bishop and Nolan noted a correlation of -0.37 between respiratory rate and SaO₂; however, I feel that this may not emphasise adequately the variability in both parameters. For example, in a consecutive series of 132 asthmatic children studied by pulse oximetry at St George's Hospital, London, we noted a wide scatter of pretreatment SaO₂ values. As illustrated in the figure an asthmatic child with a SaO₂ of 90% could have a respiratory rate between 20 and 70 breaths per minute (bpm), and a child with a respiratory rate of 40 bpm could have a SaO₂ anywhere between 84 and 94%. This might suggest that the two variables are measuring different aspects of the acute attack (for example, compensation *v* decompensation) and that both should be recorded.

In our study, as with previous studies, initial SaO₂ neither predicted duration of inpatient stay nor the time spent on nebuliser treatment every two hours. The failure of oximetry to predict outcome may relate in part to the different time courses of recovery for SaO₂ and airway function.⁴ In addition clinical decisions after admission are usually made on

parameters that reflect airway obstruction such as peak flow measurements and wheezing.

In general SaO₂ can reflect asthma severity in acute plasma in children, though as with other parameters used in asthma severity scales there is considerable overlap between grades of severity. This should not mitigate against oximetry as a valuable tool in the assessment of asthma in children.

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- 1 Bishop J, Nolan T. Pulse oximetry in acute asthma. *Arch Dis Child* 1991;66:724-5.
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Dr's Bishop and Nolan comment:

Dr O'Keeffe has made an important point about the non-linearity of the relationships of SaO₂ to severity in acute asthma, and the difficulties ensuing when studying predictive accuracy in groups of differing severity. These difficulties apply not only to groups in different settings, but also to comparisons of pretreatment to post-treatment oximetry, and SaO₂ in the very young compared with other children.

Information about selection criteria and severity of patients in our study had been removed from the original paper at the editors request, in order to present a short report. The population of 102 that we studied at the Royal Children's Hospital (RCH) clinic who were included in the oximetry study included 30 referred by a general practitioner and four referred by a specialist, and the remainder referred themselves. Sixteen percent had persistent or frequent episodic asthma, 12% attended with their first recognised asthma ever, and the remainder had a history of infrequent episodic asthma. A total of 43% had a nebuliser at home. The admission rate was a little lower at RCH than that reported in the study of Geelhoed *et al* (42% compared with 54%),¹ though the relapse rate of 46% reported by Geelhoed *et al* means that the unfavourable outcome rate in that study was 75% compared with 52% in our study. Overall it does not seem likely that our study group had a preponderance of mild/moderate asthmatics in the sample, when compared with the group studied by Geelhoed *et al*.

It would not usually be thought necessary to report a scatter plot as illustration of a correlation coefficient, unless the point was to demonstrate outliers or non-linearity. In fact in our data the scatter plot of the relationship of SaO₂ to respiratory rate was similar in appearance to the one Dr O'Keeffe presents (without the corresponding correlation coefficient).

It would, however, be quite incorrect to use a moderate correlation as the basis for inferring that accurate predictions can be made. The evaluation of a diagnostic test is best done using sensitivity and specificity. We would be interested in the values for these parameters for admission from the study that Dr O'Keeffe has carried out.

Recently, Kerem *et al* reported data from which it can be calculated that pretreatment SaO₂ had a sensitivity of 38% for admission (37% for unfavourable outcome).² There is not sufficient detail in their report to allow calculation of specificity; however their fig 1 suggests that specificity was very high. Note that in all three studies the specificity of SaO₂ is higher than the sensitivity with one exception: the pretreatment measurement in the study of Geelhoed *et al*.

There is an area of common agreement in the study of Dr O'Keeffe, and studies by Geelhoed *et al*, Kerem *et al*, and our own, namely that oximetry is not accurate enough on its own but is useful as an adjunct to clinical observation. In our paper we attempted to quantify the utility of oximetry when used in combination with clinical judgment, while making use of the best feature of SaO₂, its specificity. This works by using low post-treatment SaO₂ as a criterion for admission, not trusting a high result for SaO₂ as sufficient criterion for discharge, and using highly sensitive clinical criteria as the basis for the decision to admit among those with high post-treatment SaO₂.

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Subdural fat effusion complicating parenteral nutrition

SIR,—I read with interest the report of Rushforth *et al* describing a subdural fat effusion complicating parenteral nutrition but would like to offer a more likely anatomical explanation for this complication.¹

The venous anatomy of the scalp is very variable and the posterior auricular vein may communicate directly with the intracranial transverse venous sinus via the mastoid emissary vein.² The practical significance of this is illustrated by a radiograph of a premature

Radiograph of a premature neonate with venous catheter outlined by contrast material.

Respiratory rate and SaO₂ in 132 asthmatic children. Bars indicate medians.

neonate who underwent percutaneous insertion of a Vygon central venous catheter after surgery for oesophageal atresia. Injected intravenous contrast material outlines the course of the catheter from posterior auricular vein (PAV) to transverse venous sinus (TVS) via the mastoid emissary vein; contrast also fills the sigmoid venous sinus and internal jugular vein (IJV). Retrograde flow of parenteral feeding solution around a catheter inserted via the posterior auricular vein could therefore reach the transverse venous sinus more directly than the route suggested in the report. Unless the catheter passes directly extracranially to a central vein from this site of entry, which is uncommon, it should be removed. I would endorse previous recommendations that the position of the catheter must always be checked radiographically,^{3 4} but would not accept a line inserted via the posterior auricular vein that was 'probably in the external jugular vein'. Optimum catheter tip position is in the mid right atrium but parenteral feeding solutions of restricted osmolality can be safely administered for periods of up to 10 days via catheters positioned directly in central great veins (the superior and inferior vena cavae and the internal jugular vein), provided blood can be freely aspirated through the line.⁵

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- 4 Shaw JCL. Technical problems in parenteral nutrition of the premature infant. *Acta Chir Scand* 1981(suppl):258-66.
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Complications of splintage in congenital dislocation of the hip

SIR,—May I comment on the recent report by Langkamer, Clarke, and Witherow?¹ For many years the great majority of infants in Bristol with congenital dislocation of the hip (CDH) have been managed by the neonatal paediatric staff. With a CDH incidence of approximately 1.5% and more than 10 000 births each year, it can be estimated that some 1500 infants will have been treated for a few weeks in a light abduction splint (von Rosen or Aberdeen) during the course of a decade. This form of treatment has proved remarkably effective and safe. In a cohort of 445 cases followed up for five years, primary success was achieved in 99% and there were no cases of avascular necrosis.² The most serious complications were parental anxiety and superficial skin problems. It was thus with concern that I read of the six infants with splintage complications reported by our orthopaedic colleagues, who further stated that 'these cases illustrate that treatment of neonatal hip instability does have a significant complication rate'.¹ On this basis they then discussed alternative options to early splintage. Unfortunately no information was given in their report on the population from which their cases were drawn. With their help and permission this crucial data can now be made available. The six infants were born between 1980 and 1987; four were referred into Bristol for treatment of their complications, having been born and splinted elsewhere. Of the two Bristol born infants, one was treated by the paediatric staff while the other was referred at once for orthopaedic care because of a second deformity. (It is of interest that four of the six infants had been treated in a Pavlik harness.) Although incomplete, this information appears to confirm the relative safety of early splintage, in sharp contrast to the high morbidity associated with delayed management.² All the same I wish to support strongly my orthopaedic colleagues' plea regarding the need for expert supervision and regular review of all abduction splintage.

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- 1 Langkamer VG, Clarke NMP, Witherow P. Complications of splintage in congenital dislocation of the hip. *Arch Dis Child* 1991;66: 1322-5.
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Children injured by other children

SIR,—At child protection conferences perpetrators of injuries to children cannot always be identified. If parents deny responsibility and have no explanations for the injuries the suspicion is that something is being concealed. There is a grave risk of their children entering the care system because the parents are either believed to be the perpetrators or they are deemed to have failed to protect the children.

All workers with children need to be aware of the full range of ways in which injuries occur to children: self injury, injury by parent, by sibling or other children, or by a third party. Some children injure themselves and conceal that they have done so in order to get adults into trouble.¹

The possibility that one child may injure another, or a sibling injure a sibling, is sometimes too easily dismissed. Research on burned children in the early 1960s showed that nine of 105 children were burned by their siblings or other children—that is, 8.6% of burns in children were child inflicted.²

What are the rates of child injured by child for other types of injury that are sometimes unexplained such as bruises, lacerations, fractures, etc?

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- 1 Benians R, Goldacre P. Non-accidental injury in children: two cases of concealment of self injury. *BMJ* 1984;289:1583.
- 2 Benians R. Psychological aspects of burns in children. Cambridge: Cambridge University, 1974. (MD thesis.)