LETTERS TO THE EDITOR

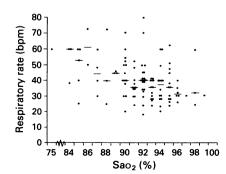
Pulse oximetry in acute asthma

SIR,—I read the article by Bishop and Nolan on pulse oximetry in asthma with interest. 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, 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 proponderance 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 vdecompensation) 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



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

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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- Bishop J, Nolan T. Pulse oximetry in acute asthma. Arch Dis Child 1991;66:724-5.
 Geelhoed GC, Landau LI, LeSoeuf PN. Predic-
- 2 Geelhoed GC, Landau LI, LeSoeuf PN. Predictive value of oxygen saturation in emergency evaluation of asthmatic children. BMJ 1988; 297:395-6.
- 3 McFadden ER Jr, Lyons HA. Arterial blood gas tensions in asthma. N Engl J Med 1968;278: 1027-32.
- 4 Mihatsch W, Geelhoed GC, Landau LI, LeSoeuf PN. Time course of change in oxygen saturation and peak expiratory flow in children admitted to hospital with acute asthma. *Thorax* 1990;45:438-41.

Drs 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%), 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.

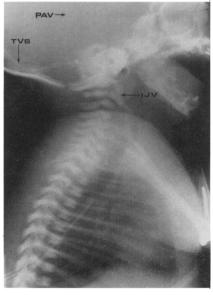
There is an area of common agreement in the study of Dr O'Keeffe, and studies by Geelhoed et al, Kerem et al, and our own, namely that oximetry is not accurate enough on its own but is useful as an adjunct to clinical observation. In our paper we attempted to quantify the utility of oximetry when used in combination with clinical judgment, while making use of the best feature of Sao2, its specificity. This works by using low posttreatment Sao₂ as a criterion for admission, not trusting a high result for Sao2 as sufficient criterion for discharge, and using highly sensitive clinical critera as the basis for the decision to admit among those with high posttreatment Sao₂.

- 1 Geelhoed GC, Landau LI, LeSoeuf PN. Predictive value of oxygen saturation in emergency evaluation of asthmatic children. BMJ 1988; 297:395-6.
- 2 Kerem E, Tibshirani R, Canny G, et al. Predicting the need for hospitalization in children with acute asthma. Chest 1990;98:1355-61.

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.