LETTERS TO THE EDITOR

Pulse oximetry in sickle cell disease

EDITOR,—I was interested to read the paper by Pianosi *et al.*¹ The use of pulse oximetry is becoming more widespread, and it is sometimes easy to accept 'machine values' which may be unreliable in a given physiological setting.

However, I feel the methodology used in this paper was not able to examine the proposed hypothesis. The use of arterialised capillary oxygen tension has been shown to be an inaccurate estimation of true arterial oxygen tension. Further the use of calculated saturations has been shown to be unreliable, compared with that measured directly using a co-oximeter, particularly in the presence of an abnormal dissociation curve.

In short although this paper questions the reliability of pulse oximetry in sickle cell disease, it provides no evidence that it is unreliable. The only way to do this would be to use simultaneous pulse oximetry and arterial sampling followed by co-oximeter estimation of saturation. Anything less merely clouds the issue.

S W JONES
Department of Neonatal Medicine,
Hospital for Sick Children,
Elizabeth Street,
Toronto,
Canada M5G 1H4

- 1 Pianosi P, Charge TD, Esseltine DW, Coates AL. Pulse oximetry in sickle cell disease. *Arch Dis Child* 1993; **68**: 735–8.

Dr Pianosi comments:

The points raised in Dr Jones' letter are important and merit comment. The use of arterialised capillary blood oxygen tension (PO_2) has limitations, but we do not feel they affect our results. The mean PO_2 found was 10.3 kPa (77 mm Hg),¹ identical to arterial PO_2 values found by Wall *et al.*¹ in children with sickle cell disease, many of whom had a history of acute chest syndrome or abnormal pulmonary function tests. Hence, we believe that our PO_2 measurements were valid reflections of arterial blood. While co-oximeter measurement of oxygen saturation is certainly the gold standard against which pulse oximetry should be judged, enough blood to perform duplicate blood gas measurements and saturation measurements was seldom obtained. Thus we had to rely on calculated saturation. We were initially hesitant to do so (computed using the Kelman subroutine²). However, as a check on its accuracy, we also computed saturation, knowing the arterialised capillary PO_2 and the P_{50} , using the Hill equation.³ This computation of arterial saturation, arrived at by a radically different approach, yielded identical values for saturation in 18 patients, and was within 3% of the Kelman derived saturation in the remaining patients. For this reason, we feel confident in our reporting of arterial saturation. Nevertheless, we agree with Dr Jones' final comment and hope that our report prompts others to undertake the study proposed.

- 1 Wall MA, Platt OS, Streider DJ. Lung function in children with sickle cell anemia. *Am Rev Respir Dis* 1979; **120**: 210–4.
- 2 Kelman GR. Digital computer subroutine for the conversion of oxygen tension into saturation. *J Appl Physiol* 1966; **21**: 1375–6.
- 3 Bunn HF. Human hemoglobins: normal and abnormal. In: Nathan DG, Oski FA, eds. *Hematology of infancy and childhood*. Toronto: WB Saunders, 1973: 699.

Human parvovirus B19 encephalopathy

EDITOR,—Central nervous system involvement in erythema infectiosum has been rarely reported.^{1,2} Anderson *et al.* confirmed that human parvovirus B19 (HPV-B19) caused erythema infectiosum,³ and recently Okumura *et al.* reported a case of aseptic meningitis caused by HPV-B19.⁴

We describe here two cases of HPV-B19 encephalopathy.

Case reports

CASE 1

A 5 year old girl, without any significant past history, was admitted to our hospital because of convulsions and drowsiness. Four days later she developed infectious mononucleosis-like symptoms and erythema. Her acute phase sera had IgM and IgG antibodies against HPV-B19 confirmed by enzyme linked immunosorbent assay, and HPV-B19 DNA was found using polymerase chain reaction techniques. Cerebrospinal fluid (CSF) studies showed normal cell count, an increase in protein concentration, and positive HPV-B19 DNA results. She became completely well on discharge.

CASE 2

A 5 year old girl had erythema compatible with erythema infectiosum. Five days later she experienced convulsive status requiring high dose pentobarbitone treatment. Her acute phase sera had IgM and IgG antibodies against HPV-B19, and HPV-B19 DNA. CSF studies revealed lymphocyte dominant pleocytosis, an increase in protein concentration, and negative HPV-B19 DNA result. She was much improved on discharge.

To our knowledge, this is the first report of HPV-B19 encephalopathy that has been serologically confirmed. Although immunological mechanisms may have induced encephalopathy in our patients, it is not clear how they were related to HPV-B19.

TOHRU WATANABE
MASAHISA SATOH
YOSHIHIKO ODA
Department of Paediatrics,
Niigata City General Hospital,
2-6-1 Shichikuyama,
Niigata 950, Japan

- 1 Balfour HH, Schiff GM, Bloom JE. Encephalitis associated with erythema infectiosum. *J Pediatr* 1970; **77**: 133–6.
- 2 Hall CB, Horner FA. Encephalopathy with erythema infectiosum. *Am J Dis Child* 1977; **131**: 65–7.
- 3 Anderson MJ, Jones SE, Fisher-Hoch SP, *et al.* Human parvovirus, the cause of erythema infectiosum (fifth disease)? *Lancet* 1983; **i**: 1378.
- 4 Okumura A, Ichikawa T. Aseptic meningitis caused by human parvovirus B19. *Arch Dis Child* 1993; **68**: 784–5.

Ocular contamination with BCG vaccine

EDITOR,—The complications of BCG vaccination in both the immunocompetent, with local and lymph node ulceration, and in the immunocompromised, with disseminated infection, are familiar to most paediatricians. Moreover, the risks to the doctor from needlestick injury are well known. There are probably few other risks for the vaccinator but we describe a case of ocular contamination with BCG vaccine.

During attempted intradermal injection of BCG vaccine into a struggling neonate's upper arm, the syringe slipped out of the infant's skin discharging its contents into the attending doctor's eye. The doctor had received BCG vaccine in childhood. Despite lavage of the eye with water, a painful follicular conjunctivitis developed 24 hours later. There was a rapid response to topical steroids, and the inflammatory response settled completely over the subsequent week. Although it was assumed that this was a delayed-type hypersensitivity response, anti-BCG cover was given with a one month course of oral isoniazid.

A J POLLARD
Department of Paediatrics

R H GEORGE
Department of Microbiology,
Children's Hospital,
Ladywood Middlesway,
Ladywood,
Birmingham B16 8ET

Becker muscular dystrophy: an unusual presentation

EDITOR,—Thakker and Sharma are incorrect in believing that theirs is the first report of Becker muscular dystrophy presenting with myoglobinuria.¹ We reported on a boy with an identical presentation, who had a deletion of exons 3–7 in the dystrophin gene.² This child died of acute rhabdomyolysis and cardiac arrest during a general anaesthetic for a dental procedure. It is important to think of unusual presentations of Becker dystrophy in an otherwise barely symptomatic child, such as myoglobinuria and muscle cramps,³ because these children are at risk of cardiac arrest under anaesthesia.^{2,4} Guidelines for prevention have been published.⁵

ANDREW BUSH
Royal Brompton National Heart and Lung Hospital,
Sydney Street,
London SW3 6NP

VICTOR DUBOWITZ
Department of Paediatrics and Neonatal Medicine,
Hammersmith Hospital, Du Cane Road,
London W12 0HS

- 1 Thakker PB, Sharma A. Becker muscular dystrophy: an unusual presentation. *Arch Dis Child* 1993; **69**: 158–9.
- 2 Bush A, Dubowitz V. Fatal rhabdomyolysis complicating general anaesthesia in a child with Becker muscular dystrophy. *Neuromuscul Disord* 1991; **1**: 201–4.
- 3 Dubowitz V. *Muscle disorders in childhood*. Philadelphia: WB Saunders, 1978.
- 4 Chalkiadis GA, Branch KG. Cardiac arrest after isoflurane anaesthesia in a patient with Duchenne's muscular dystrophy. *Anaesthesia* 1990; **45**: 22–5.
- 5 Britt BA. Malignant hyperthermia. *Canadian Anaesthetists Society Journal* 1985; **32**: 666–7.