Table 1 Mean haemoglobin concentrations in paired venous and skin puncture blood samples

Haemoglobin range in venous blood (g/l)	Mean (SD) skin puncture haemoglobin (g/l)	p	Mean (SD) venous haemoglobin (g/l)	Difference	
				Mean (g/l)	Mean (2SD) CI (g/l)
≤ 90 (n=42)	84.1 (13.2)	p < 0.0001	79.5 (10.4)	4.7	- 5.7 to 15.1
91-110 (n=94)	104.8 (7.5)	p < 0.0001	101.2 (5.0)	3.6	- 7.7 to 14.9
≥ 111 (n=52)	121.4 (8.5)	p < 0.005	119.1 (6.3)	2.3	- 8.7 to 13.3
All (n=188)	104.8 (16.1)	p < 0.0001	101.3 (15.6)	3.5	- 7.6 to 14.6

It seems unwise to assume the haemoglobin concentrations reported by Emond *et al* are lower than those that would have been obtained from venous blood from the same children. Their method of sampling appears to be similar to our own, and given the bias to slightly higher values obtained with the Hemocue (assuming the values given by the laboratory analyser represent truth), it is possible that venous haemoglobin values in their population could be on average some 5 g/l lower than those reported for skin puncture samples.

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Challenges in the management of childhood brain tumour

EDITOR,—A number of major challenges need to be faced if the outcome for children with brain tumours is to improve. Primary and secondary care physicians need to have a greater awareness of the symptoms and clinical signs that justify the urgent referral of children with tumours of the central nervous system and special arrangements for handling such referrals need to be negotiated. Families need improved access to information at the time of diagnosis so that they can learn about the full range of available therapeutic options.

It is mandatory that a national network of specialist neuro-oncology teams should be developed to which children would be selectively referred. Clearly such referrals should take place before any surgical intervention is undertaken. This may mean that individual neurosurgeons have to accept that they cannot operate on children with brain tumours if they are not able or prepared to manage the child within an appropriate multidisciplinary team. Such teams should be patient, not specialist, centred and would develop strong links with local community paediatric services. Such a change in attitude

may need the combined intervention of health professionals and parents, the latter using the rights for special needs education prescribed by the Children Act as a basis for their lobbying.

The United Kingdom Children's Cancer Study Group (UKCCSG) has made considerable progress in developing audited, collaborative research protocols that will allow assessment of the relative merits of different treatments. There is a need for ever closer neurosurgical input into clinical trial development.

Such a reorganisation of facilities for childhood brain would be greatly assisted by the development of specialist purchasing guidelines that define core standards of care. This process has been discussed by representatives of the paediatric neurosurgical and oncological interest groups of the UKCCSG. Approval of all the relevant royal colleges is being sought.

We hope that we can ensure more consistent service provision for UK children with brain tumours. Current inequalities in health service availability become only too obvious when high profile cases seeking international referral hit the national headlines.

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Paediatricians' knowledge of cardiac arrest guidelines

EDITOR,—I would like to draw attention to an important inconsistency in the article by Buss *et al* in your journal.¹ Two references are cited that draw attention to the facts needed in a study of paediatricians' knowledge of paediatric cardiac arrest guidelines.^{2,3} However on examination of the protocols from the two sets of guidelines published in 1993 and 1994, there are a number of differences between them due to updating. In the 1994 ventricular fibrillation protocol a preliminary precordial thump has been added.² In the 1994 asystole protocol atropine has been removed completely, while it was an integral part of the protocol in 1993.³ Also in the asystole protocol the giving of bicarbonate has changed from a necessity in 1993 to being just a consideration in the 1994 protocol.^{2,3}

Because of rapid updating of the guidelines there are at present two sets published and widely available, which have a number of differences. The study does not clearly specify which guidelines were used and the conclu-

sions drawn are based on the fact that the two published sets are equivalent.

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Dr Buss comments:

There was a typographical omission from the reference for the APLS guidelines—hence the problem that Dr Ward encountered. The third reference should have ended: London: BMJ Publishing Group, 1993 (reprinted with revisions 1994).

The study itself used the current guidelines at the time (1994), and we stressed in our second paragraph that the 'Guidelines for paediatric resuscitation published by the European Resuscitation Council (1994) are incorporated within the advanced paediatric life support protocols'. This directly infers that we were using the 1994 APLS protocols but the failure to indicate this accurately in the references was not been picked up by ourselves or the referees and Dr Ward is to be congratulated for noticing this incongruity.

The controversy over the use of bicarbonate was clearly mentioned in the second part of our paragraph on asystole, and although results were included they did not affect overall figures for sequence failure. With regard to the use of a praecordial thump—this has similar connotations to bicarbonate usage and in the scenario that we gave would be neither warranted or desirable.

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Sleeping position and cot death

EDITOR,—The trend of the incidence of the sudden infant death syndrome (SIDS) in Austria¹ strikingly resembles the one presented by Gilbert from England and Wales² (see figure 1). However, in our opinion there are several arguments against the widespread assumption of a causal relationship between the prone sleeping position and SIDS.

Firstly, it was at the 13th International Paediatric Congress in Vienna in 1971 that the assumed advantages of the prone sleeping position were first presented by the Austrian paediatricians Reisetbauer and Czermak.³ If the prone sleeping position were to be blamed for the growing occurrence of SIDS,

Figure 1 Mortality from SIDS and postneonatal mortality (PNM) in England and Wales (E/W) and Austria (A).

one would expect the SIDS incidence to increase in Austria shortly after its introduction in 1971. Yet in our country a marked increase cannot be discerned until after 1977, whereas in England and Wales the rise of SIDS occurred much earlier (1971).

Secondly, it can be observed in Austria, as in England and Wales, that the decline of SIDS started several years before the introduction of media campaigns against prone sleeping. The peak year for SIDS was 1988 in both countries, whereas the first nationwide media campaigns against prone sleeping were not launched until October 1991 in England and Wales,⁴ and in April 1993 in Austria.

Thirdly, the rise and fall of SIDS does not seem to have affected the continuity of the decline of postneonatal mortality in our country (see figure). Postneonatal mortality was neither increased by the occurrence and the increase of SIDS, nor was its further steady decline accelerated by the decreasing SIDS rate.

Our claim is that the dynamics of cot death need to be explained by diagnostic transfer between the SIDS and non-SIDS components of postneonatal mortality. Diagnostic transfer should be taken into account before interpreting trends of SIDS as a result of public health interventions.

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Uses and abuses of pulse oximetry

EDITOR,—The recent review article by Moyle correctly outlined sources of inaccuracy in the use of the pulse oximeter,¹ however an important omission was the behaviour of pulse oximetry in conditions of low arterial pressure. Significant inaccuracies² can occur at mean pressures less than 30 mm Hg and pulse pressures of only 20 mm Hg. The 'dropout' (the inability to produce a signal at low arterial pressures) at finger placement sites limits the usefulness of these devices under conditions of reduced cardiac output, hypotension, and peripheral vasoconstriction.

Moyle in his review also mentions that central placement of oximeter probes (tongue and cheek placement) would confer an advantage in terms of response times to changes in saturation of haemoglobin. It

could be inferred from this review that oximeter probes located around the head should be the preferred site of probe placement. This placement could lead to further erroneous readings.

It has been shown that the overall performance of ear, nose, and forehead probes for various devices may be worse than for finger probes when hypoxia is induced at low radial arterial pressures.³ The ability to detect a weak signal when the probes of some devices are located on the head can give erroneous values for saturated oxygen. Unless the clinician knows precisely the specific behaviour of his particular probe at low arterial pressures it can be recommended that head placement of oximeter probes should be used only if the finger site is unavailable. Signal dropout, as well as an indicator that there is a problem with the peripheral perfusion, is then also a cue for an urgent alternative measure of saturation by blood gas analysis.

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