packed cell volumes with a lower limit in black American women may be due to the high frequency of α thalassaemia in people of African descent and to total compensation for lifelong slightly lower haemoglobin concentrations.

Steer and colleagues consider that it "remains possible that genuine anaemia . . . has adverse consequences on fetal growth and long term outcome." Obstetricians and others working in developing countries know well the severe consequences of maternal anaemia on placental size, fetal growth, length of gestation, fetal nutrition, infant morbidity and mortality, and maternal morbidity and ultimately mortality.⁴ It remains the duty of obstetricians to try to bring maternal haemoglobin concentration into the optimal range for pregnancy. Evidence suggests that the optimal range is 110-140 g/l rather than the lower values proposed by Steer and colleagues.

ALAN F FLEMING
Director of laboratory services

University Teaching Hospital,
P/Bag RW 1X,
Ridgeway 15102,
Lusaka,
Zambia

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Author's reply

EDITOR,—To answer the questions of Sarah Vause and colleagues in reverse order, I can reassure them that the data given in our paper referred only to singleton pregnancies. This information was included in our original submission but was inadvertently omitted when we revised the paper to answer points made by referees. I am grateful for the chance to put the record straight.

The St Mary's maternity information system records the lowest haemoglobin concentration but not the gestation at which the lowest value occurred. The references we quoted in our paper make it clear that most of the expansion in plasma volume (which is reflected in the fall in haemoglobin concentration) has occurred by 20-24 weeks' gestation and there is comparatively little change thereafter. Thus, estimation of haemoglobin concentration at any gestation after 24 weeks is likely to show a similar relation with birth weight.

Finally, although we referred mainly to the relation of the lowest haemoglobin concentration to birth weight, I confess that we assumed several times that the lowest concentrations showed the largest falls from prepregnancy values. This assumption is consistent with the longitudinal studies of plasma volume in pregnancy that we cited in our paper. I accept that without measuring prepregnancy haemoglobin concentrations it cannot be proved. If the assumption were not true, however, the average fall in haemoglobin concentration would be the same (or greater) with a high pregnancy haemoglobin concentration as with a low (95-105 g/l) concentration. This would lead to the intriguing conclusion that women with higher than average haemoglobin concentrations when not pregnant would have smaller babies than their peers with lower values. I am not aware of any obvious physiological basis for such a hypothesis. To test it would require a large number of women to have their haemoglobin concentrations

measured before they became pregnant; such a study would probably present insuperable logistical problems.

PHILIP STEER
Professor

Academic Department of Obstetrics and Gynaecology,
Charing Cross and Westminster Medical School,
Chelsea and Westminster Hospital,
London SW10 9NH

Outcomes of neonatal intensive care

EDITOR,—Christine Sanderson and D M B Hall support the concept of improving the availability of data on the outcome of neonatal intensive care.¹ Their suggestion that this can best be achieved by each unit employing "someone trained in counselling, child development and psychological assessment to follow up its graduates" seems, however, to be against the current trend.

The Audit Commission initiated this debate in the report *Children First* after being unable to obtain even the most straightforward population based data on outcomes.² It suggested that simple information (Is the child blind? Is the child deaf?) should be collected routinely for all babies who had received intensive care. The report mentioned by Sanderson and Hall was an attempt by the relevant professional groups to reach consensus on both what should be included in such a minimum dataset and the optimum timing of the collection of data.³ To fulfil the challenge issued by the Audit Commission it was considered essential that all infants who had received intensive care, not simply those in whom problems might be expected, should be included in any review of outcome. Although clinical data may sometimes give a strong indication of subsequent disability, prediction continues to be unreliable.

Given the mobility of the population and the current role of community paediatric services, it is appropriate that health staff based in the community should monitor the later health status of those who receive neonatal intensive care. In Sanderson and Hall's region (Trent) each health district still tries to examine all children, whether or not they have required intensive care, between 1½ and 2½ years of age. These reviews already aim to collect all the items of the minimum dataset except the date on which the child's mother left full time education.¹ The information is recorded and stored centrally but rarely used. Surely it is sensible to try to refine and focus this activity instead of inventing a new system at further cost.

DAVID FIELD
Senior lecturer in child health

Department of Child Health,
University of Leicester School of Medicine,
Leicester Royal Infirmary,
PO Box 65,
Leicester LE2 7LX

- 1 Sanderson C, Hall DMB. The outcomes of neonatal intensive care. *BMJ* 1995;310:681-2. (18 March.)
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Water births

Could saline in the pool reduce the potential hazards?

EDITOR,—I have recently attended four water births; three went smoothly, but in one the baby had some difficulty breathing and was admitted to the special care baby unit, where he had a convulsion and was found to have a low sodium concentration. Inhalation of water was thought to be a likely cause for this, and the diagnosis on

discharge was freshwater drowning. Apparently the inhalation of even a small quantity of fresh water can cause appreciable haemodilution and the kind of problem experienced by this baby (who made a full recovery).

The proponents of water birth argue that a baby cannot inhale water even after the head is delivered because of chest compression in the birth canal. I do not believe this: on several occasions I and midwives have heard a baby whose head has been delivered make an attempt to cry. If the same happened under water a small amount of fresh water would be inhaled.

If the contents of the birthing pool were not fresh water but isotonic saline then presumably this haemodilution effect could be avoided. In the most recent water birth I attended, the patient was a qualified pharmacist, who told me that the birthing pool held 909 litres of water, to which the addition of 9 kg of salt would produce a solution of isotonic saline. I have discussed the idea locally with colleagues, who can see no obvious flaw in it, and I would appreciate others' comments.

C N BARRY
General practitioner

Wroughton Health Centre,
Wroughton,
Swindon SN4 9LW

*We sent Dr Barry's letter to an expert, asking for his comments.

EDITOR,—The suggestion that salt should be added to make the water isotonic in a water birth would give rise to few theoretical objections by those who insist on having a water birth and would probably have an appeal, following the experience of C N Barry. A convulsion due to hyponatraemia in a baby who has been immersed in fresh water is important. The phenomenon is well recognised in the so called "drownproofing" of infants, in which well meaning but misguided people attempt to teach children under the age of 12 months to swim. Under these circumstances the infants can be trained to struggle across up to 10 m. They are submerged for an appreciable part of this distance and swallow a considerable amount of water. Convulsions have been reported after lessons and are known to be associated with similar hyponatraemia to that seen after the swallowing of large quantities of fresh water with subsequent haemodilution.

Nevertheless, it would be wrong to add salt in this way, or indeed in any way to encourage the practice, until experimental studies with animals were undertaken. Any experimental trials by well meaning adults that use infants or children as research subjects are unacceptable until double blind experimental studies have been reported and the procedure is found to be safe.

JOHN PEARN
Professor of child health

Department of Child Health,
Royal Children's Hospital,
Brisbane,
Queensland 4029,
Australia

Trends in sudden infant death

The few remaining sudden infant deaths are likely to be explicable and avoidable

EDITOR,—E A Gilman and colleagues report a dramatic fall in the rate of the sudden infant death syndrome in England and Wales after a peak in 1988.¹ This particularly occurred after the Department of Health issued advice at the end of 1991 that babies should not be placed to sleep prone; this instituted the "back to sleep" campaign. I have reported a campaign in the Scarborough district to avoid overdressing, overwrapping,

and overheating babies; the campaign started in 1985-6, before advice about avoiding the prone sleeping position was available.² Since 1991 the emphasis of this campaign has been maintained, while we have tried to ensure that as few babies as possible sleep prone. We have advised that the normal maximum indoor clothing needs at 18°C are a nappy, vest, sleepsuit, and cardigan, with coverings of a sheet and three single blankets. Before the Department of Health's publicity, virtually all babies in this district who died had been placed and were found dead in the prone position.

The table shows the cumulative effect of these two campaigns in reducing postneonatal infant mortality. By 1991 the sudden infant death syndrome due to avoidable overheating had become rare, but detailed investigations at the scene of death showed that many babies were still hot or sweating when found dead; this has been postulated as likely to happen if feverish babies sleep prone.^{3,4} Since 1992 no cot deaths have occurred in this district.

Postneonatal infant mortality in Scarborough district, 1983-94

Years	No of births	All deaths	All cases of SIDS	Cases of SIDS due to avoidable overheating
1983-6	6061	39	30	19
1987-91	8124	20	16	5
1992-4	4519	5	0	0

SIDS=Sudden infant death syndrome.

Significance of reduction in all cases of the sudden infant death syndrome: 1983-6 v 1987-91, $P=0.003$; 1987-91 v 1992-4, $P=0.007$ (two tailed χ^2 test for independence with Yates's continuity correction).

All districts will continue to have sporadic cases of the sudden infant death syndrome, as unexplained deaths occur at every age. The Department of Health's advice is empirical, so that success in following the advice cannot prevent all cases of the syndrome and failure to follow the advice cannot be used as the explanation. The fall in unexpected deaths suggests, however, that the few that now occur are likely to be explicable and avoidable, even before the challenge of preventing parental smoking is addressed. The target rate for the syndrome in future years should be zero, not just a downward trend.

A N STANTON
Consultant paediatrician

Scarborough Hospital,
Scarborough,
North Yorkshire YO12 6QL

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Infant respiratory death rates mirror sudden infant deaths

EDITOR,—E A Gilman and colleagues speculate about the reasons for the dramatic falls in sudden infant deaths that occurred during the first and second quarters of 1992 as compared with the decreasing trends in the rates for these two quarters over the previous four years.¹ The dramatic falls occurred after the launch of the campaign to encourage parents to put infants to sleep on their backs and to avoid smoking near them or overheating them.

The authors put forward as one possible explanation of these falls the interaction between the prone sleeping position and the underlying patho-

genic mechanisms that it affects. In this regard, we would draw attention to the fact that the secular and seasonal trends in the monthly numbers of sudden infant deaths over recent years have been mirrored by those for infant deaths attributable to respiratory disease. The figure shows the two series for the years 1986-92. The concordance between

Numbers of sudden infant deaths and infant deaths attributable to respiratory disease, England and Wales, 1986-92 (source: Office of Population Censuses and Surveys)

these two series is evident in the summer troughs as well as the winter peaks. This phenomenon suggests that whatever has led to the seasonal variation and decreasing trend in sudden infant deaths, including the pronounced decrease during 1992, has produced a similar pattern for infant deaths attributable to respiratory disease. Furthermore, we have shown that during 1986-90 there was a close association between the monthly incidence of respiratory infectious diseases in the age group 0-4 years.²

D L CROMBIE
Research consultant
K W CROSS
Statistical consultant
D M FLEMING
Director

Birmingham Research Unit,
Royal College of General Practitioners,
Birmingham B17 9DB

- 1 Gilman EA, Cheng KK, Winter HR, Scragg R. Trends in rates and seasonal distribution of sudden infant deaths in England and Wales, 1988-92. *BMJ* 1995;310:631-2. (11 March.)
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Dutch doctor convicted of murdering disabled infant

EDITOR,—Tony Sheldon reports the death at the hands of a Dutch gynaecologist of a baby with serious congenital malformations who was in great pain and had a limited life expectancy; the doctor was convicted of murder but not sentenced.¹ Bringing about the death of a severely disabled infant is an attempt to wipe out a distressing problem for the attendant adults by attributing their anguish to the child and then bringing that to a quick end. This overlooks three important facts.

Firstly, the natural adult reaction to any crisis of expectation is to try to deny it. Doctors can deny reality by continuing aggressive treatment for an incurable illness as well as by agreeing to bring it to a quick end. In either case this can arrest or delay the anger, guilt, bargaining, and depression of grief, which may all arise during its natural resolution but which, when thwarted, can leave bereaved people with serious emotional upsets. Removing the problem may not stop the pain.

Secondly, babies are born ready to relate to parents along all five sensory pathways. Even when serious disability hinders such normal mutual interaction (and this is not always so) parents who attempt it are more likely to feel satisfied that the child's life, though brief, was enhanced by their

personal care. Their grief is also likely to resolve better.

Thirdly, babies who are born disabled do not know that this is their condition and may never do so. They may cry because of discomfort, but symptomatic relief is available for them, just as it is for adults. Those who advocate "mercy" killing for babies (and others) tend to polarise their options into futile attempts to cure or final advice to kill. There is, however, a third way. The specialty of palliative medicine is inadequately represented in the Netherlands but is well established in Britain, both for adults and for older children. Dying babies, too, can be helped to live until they die.

All these points were illustrated in a paper that a colleague and I wrote in 1988.² To ignore the normal reaction to grief and to destroy the opportunity for parents and the child to have even a brief time to enjoy each other is to sow a legacy of chronic emotional pain. For most of us, disabled babies included, life's chief value lies not in the number of days or in physical and mental assets but in secure and creative relationships. Any doctor-parent-baby relationship should have this as its goal.

JANET GOODALL
Retired consultant paediatrician

Trentham,
Stoke on Trent,
Staffordshire ST4 8SP

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Misleading meta-analysis

Public policy is based on results of epidemiological meta-analyses that contradict common sense

EDITOR,—Matthias Egger and George Davey Smith's scepticism about the validity of meta-analyses whose conclusions are contradicted by those of single megatrials¹ can be extended to meta-analyses of epidemiological studies whose findings contradict biological common sense. The alleged causative contribution of passive smoking to lung cancer is based, among other things, on a meta-analysis of 10 case-control studies and three prospective studies that showed a highly significant 35% increased risk among non-smokers living with smokers compared with non-smokers living with non-smokers.² This risk is especially pronounced for adenocarcinoma in the periphery of the lung (a histological category not associated with smoking in Doll and Hill's studies of doctors) rather than for the central squamous cell cancers seen most commonly in active smokers.³ If the conclusion of this meta-analysis is to be believed it is necessary to suppose that carcinogenic cigarette smoke inhaled in massive doses over 30 years causes one type of lung cancer whereas the same cigarette smoke at much lower doses causes a completely different type of cancer, not usually associated with smoking, in a different part of the lung.

Similarly, on the basis of a meta-analysis of 78 trials of a reduction in salt intake in hypertension it has been claimed that a reduction of 3 g a day in Western populations could reduce the incidence of stroke by 26% and of ischaemic heart disease by 15%.⁴ This seems improbable for two reasons. Firstly, the immediate physiological response to salt reduction is that salt is conserved in the kidneys. Secondly, the maintenance of a steady blood pressure is under the control of at least six independent physiological homeostatic mechanisms, so the contribution of salt intake, if any, is likely to be very small. The results of salt restriction have been more plausibly attributed to a placebo effect.⁵