Sedation and pain score—record hourly		
Level	Sedation	Pain
1	Awake	Pain free
2	Drowsy	Comfortable except on moving
3	Asleep but moves spontaneously	Uncomfortable
4	Asleep responds to stimulation	Distressed but can be comforted
5	Hard to rouse	Distressed
Action:		Action:
Level 4: Stop infusion until return to level 3		Level 2: NCA—give bolus PCA—encourage bolus (10 minutes before activity) Epidural—increase rate
Level 5: Stop infusion. Call pain service		Level 3: Contact pain service NCA—give bolus PCA—encourage bolus Epidural—increase rate
Naloxone 4 µg/kg to be available at all times		Level 4: Contact pain service NCA—give bolus or increase background infusion PCA—encourage bolus or increase background infusion Epidural—give bolus or increase rate
		Level 5: Contact pain service

NCA=Nurse controlled analgesia. PCA=Patient controlled analgesia.

acceptable respiratory rate below which the infusion should be stopped and a respiratory rate at which naloxone 4 µg/kg will be given intravenously. The pain service can ensure that this is prescribed, and a nurse able to give it should be readily available on the ward.

Children find intramuscular injections unpleasant, but Gourlay and Boas provide good advice when they suggest that it is inappropriate to substitute them with a route for which absorption is entirely unpredictable, such as the rectal route. In our experience, it is quite possible to provide safe and satisfactory analgesia with intravenous or subcutaneous infusions of morphine or extradural blocks.

These techniques are effective and safe providing they are practised within the context of well defined protocols which have the approval of anaesthetic staff and that the patients are closely supervised by an acute pain service available for advice 24 hours a day.

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2 Commission on the Provision of Surgical Services. *Pain after surgery*. London: Royal College of Surgeons of England, College of Anaesthetists, September 1990.

SIR,—In their report of the fatal outcome of an overzealous approach to postoperative analgesia G K Gourlay and R A Boas fail to provide details of perioperative fluid management of the patient.¹ Blood loss of only 10 ml in a 9 kg child undergoing nephrectomy may be an underestimate, and the clinical findings described at 22 hours may well be due to hypovolaemia.

In addition to their suggestion that analgesic techniques require protocols for monitoring, I would add that they must be accompanied by good basic standards of care and education of all nursing and medical staff. Otherwise further unnecessary deaths will occur in the pursuit of postoperative analgesia.

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SIR,—G K Gourlay and R A Boas report that giving rectal morphine every four hours to a 9 kg boy resulted in fatal respiratory depression.¹ The child had suffered neonatal renal vein thrombosis

and had had a nephrectomy. The authors do not allude to the influence of renal dysfunction on clearance of morphine.

The dose of morphine normally recommended for intravenous infusion in children over 3 months old is 20 µg/kg/h.² The dose given to this child was about 110 µg/kg/h, which is high even allowing for reduced bioavailability from the rectal route.

The lesson from this tragic case was that, to use opioids safely, regular assessment of their analgesic efficacy and adverse effects is important.³ The report gives no practical advice on how assessment should be performed. There is an implication that monitoring for respiratory depression by counting the respiratory rate is adequate. It is well recognised that this is not the case and that results of continuous pulse oximetry while the patient breathes ambient air are a sensitive indicator of important ventilatory dysfunction.^{4,5} Changes in arterial oxygen tension (which are in turn affected, through the alveolar gas equation, by changes in arterial and alveolar carbon dioxide tension) are rapidly reflected in changes in arterial oxygen saturation. This is because when the patient is breathing ambient air the changes occur along the steep linear portion of the sigmoid shaped curve of arterial oxygen saturation against arterial oxygen tension.

Pulse oximetry has been used widely in acute pain relief in adults and, more recently, in paediatric practice.^{6,7} We have developed a monitoring protocol for children receiving opioids. A simple four point behavioural pain score is used to assess analgesic efficacy. Adverse effects are assessed by pulse oximetry, the respiratory rate, and the sedation and nausea scores and by logging the residual syringe volume. Recordings are charted hourly and interpreted regularly by a trained anaesthetist, and the opioid delivery regimen is adjusted regularly. The monitoring protocol has developed from a research tool into a practical ward nursing procedure with excellent compliance among nurses and acceptability to patients.

Many analgesic techniques used in adults are being tried in paediatric practice. Appropriate training and monitoring are essential, especially outside intensive care areas. We are not prepared to use these techniques, or to recommend their use, without this level of monitoring.

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Deaths from haemolytic disease of the newborn

SIR,—In 1985 we expressed our concern that the figures published for deaths from haemolytic disease due to red cell alloimmunisation were underestimating the true incidence.¹ We have the same anxieties about the report by Ruth Hussey and Cyril Clarke.² We presume that the figures they report are derived from analyses of death certificates completed for stillbirths, neonatal deaths, and infant deaths. If this is so, the great majority of pregnancy losses due to alloimmunisation will have been missed.

During the three years in question we managed 61 pregnancies complicated by very high maternal red cell antibody concentrations; intrauterine procedures have been used, and six pregnancies have been lost: two as neonatal deaths and three from spontaneous abortion, and one pregnancy was therapeutically aborted. Over the 10 years 1982-91, 142 pregnancies have been similarly managed, of which 26 did not result in a surviving child, four (15%) because of neonatal death and 10 (77%) because of spontaneous abortion; two (8%) were terminated. Thus 85% of these losses were not registered and are therefore excluded from perinatal mortality statistics. We believe our results are equivalent to those in other centres in the United Kingdom dealing with this pregnancy complication since we know that of 10 pregnancies of women resident in the Oxford region similarly managed by other units six did not survive, five ending as spontaneous abortions. Our thesis is further supported by legal abortion statistics for 1988, 1989, and 1990,^{3,5} when there were 23 therapeutic abortions performed because of rhesus disease; we anticipate these cases did not feature in the 52 deaths due to haemolytic disease recorded for those years by Hussey and Clarke.² If our experience is translated to the national figures, the losses from haemolytic disease due to red cell alloimmunisation would be 160, 85, and 100 for 1988, 1989, and 1990 respectively.

We concur with the view of Hussey and Clarke that alloimmunisation due to other antigens, such as Kell, C, and E antigens, continues to be important and requires vigilance on the part of clinicians and laboratory staff. Losses due to these antibodies, however, rarely occur before 28 weeks' gestation.^{6,7}

Finally, data from the Oxford region, illustrated in the figure, are different from those of Tovey (given as Dovey in Hussey and Clarke's letter).

Incidence of rhesus D sensitisation 1971-91 in the Oxford region since introduction of immunoprophylaxis

The incidence of rhesus D immunisation has been relatively static since 1973; as yet, there has been no demonstrable effect from the introduction in part of the region in 1986 of routine antenatal prophylaxis. We thus fear that the trend of the past 18 years is likely to continue.

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AUTHORS' REPLY.—MacKenzie and colleagues' presumption about our figures is correct. We thought our method of ascertainment was made clear at the start of the "methods and results" section of our paper¹; a similar procedure has been used for all our reports on deaths from rhesus haemolytic disease since 1977.²

We have always appreciated that deaths before 28 weeks were underreported because the Office of Population Censuses and Surveys registers deaths and stillbirths from 28 weeks only. However, the underreporting of these deaths will have gone back many years, long before the introduction of anti-D prophylaxis, and is a constant feature; our figures relate to neonatal deaths and stillbirths from haemolytic disease of the newborn after postnatal anti-D was introduced about 1970.

We were aware of the problem of immunisation during pregnancy rather than at delivery (not necessarily resulting in a dead baby), but Bowman, Bowman and Pollock, and Tovey *et al* suppressed most of these cases.^{3,5} However, Dovey, whom we quote in our letter⁶ (not Tovey as MacKenzie *et al* seem to suppose) found a static immunisation rate in Yorkshire, as have the workers in Oxford. Admittedly Bowman and Pollock⁴ give a much bigger dose than that used in Yorkshire, but we cannot understand why the Tovey regimen, which we believe is being followed in Oxford, is not as successful there as it was in Yorkshire. MacKenzie *et al* show a histogram giving the incidence of sensitisation for the whole of the Oxford region. Could we know the findings in those districts of the Oxford region where the antenatal trial is actually taking place?

A further point in MacKenzie *et al*'s letter needs clarification. Since D antibodies do not cause intrauterine death before about 18 weeks at the earliest, at what stage of pregnancy did the 23 therapeutic abortions performed in Oxford in 1988-90 "because of rhesus disease" occur? Do MacKenzie *et al* mean that the patient or her obstetrician did not want to run the risk of having another seriously affected baby?

We are grateful to Professor P L Mollison and Dr D Lee for advice on some aspects of this reply.

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- 1 Hussey RM, Clarke CA. Deaths from Rh haemolytic disease in England and Wales in 1988 and 1989. *BMJ* 1991;303:445-6.

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What counts as cot death?

SIR.—Recent comment on the incidence of cot death makes it essential to agree what counts as a cot death.¹ The term, first used in 1954 by Barratt—"an apparently healthy infant is unexpectedly found dead in its sleeping quarters"—originally included deaths later explained at postmortem examination.²

In 1965 Carpenter and Shaddick narrowed the definition to "those cases in which the information available does not reveal the cause or causes of death."³ This corresponded closely with the definition of the term sudden infant death syndrome proposed by Beckwith in 1969: "The sudden death of any infant or young child, which is unexpected by history, and in which a thorough post mortem examination fails to demonstrate an adequate cause of death."⁴ This diagnosis is reached by exclusion of explained deaths.

The unsatisfactory situation of the 1950s and '60s, when many unexplained infant deaths were attributed to a respiratory cause,⁵ was recognised by the inclusion in the eighth revision of the ICD (in 1968) of a category for sudden death (cause unknown)—code 795.

Since 1971 the registrar general and the Coroners' Society of England and Wales have accepted sudden or unexpected death in infancy syndrome as a natural, registrable cause of death, and the Office of Population Censuses and Surveys (OPCS) has identified sudden infant death when there is any mention of sudden or unexpected death in infancy, cot death, or such a term in the death certificate. These figures are published every two years in *OPCS Monitor DH3*, usually incorrectly headed sudden infant death syndrome.

In 1979 the ninth revision of the ICD included sudden infant death syndrome (code 798.0). In some districts, however, coroners or pathologists rarely use this as a cause of death but follow Emery and Weatherall's recommendation that a specific cause should be given,⁶ mentioning also "unexpected" when this is clinically appropriate. Such deaths are counted by OPCS as sudden infant deaths. The figures for the sudden infant death syndrome (code 798.0), published annually in *OPCS Monitor DH2*, however, comprise deaths in which the syndrome or sudden infant death or cot death is the sole cause given in the death certificate and are therefore an underestimate. VS3 mortality statistics for regions and districts, which R R Gordon used in his table,⁶ give figures for category XVI—symptoms, signs, and ill defined conditions (ICD numbers 780-799).

For monitoring purposes in England and Wales two statistics may be used. Firstly, any mention of sudden infant death may be used: the numbers (and rates/1000 live births) from birth to 1 year are 1593(2.3), 1326(1.9), and 1193(1.7) for 1988, 1989, and 1990 respectively. Secondly, the sudden infant death syndrome (ICD 798.0) given as the sole cause of death may be used; since 1986 only postneonatal figures have been published, whereas about 5% of cases occur in the first month of life. The numbers (and rate/1000 live births) for the syndrome are 1419(2.1), 1190(1.7), and 1079(1.5) for 1988, 1989, and 1990 respectively.

We recommend that doctors, pathologists, and coroners should mention in the death certificate if

an infant death was clinically unexpected, whatever the cause, and that the figures for any mention of sudden infant death should be used for monitoring cot deaths. The OPCS should publish these figures annually by region and district for England and Wales.

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Treatment of back and neck complaints

SIR.—Manipulation usually takes only a moment or two and yet the median duration of therapy in the "manipulative" treatment group in the study by Bart W Koes and colleagues was 40 minutes.¹ This would seem to imply that much of the time was in fact taken up with the mobilisation procedures.

That the therapists were permitted to modify the regimens makes it even more difficult to be sure what can be extrapolated from such group comparisons.

It is interesting, however, that after the experience of conducting this large study the authors should in effect conclude that the most pressing research requirement is to find ways of reliably distinguishing "specific" sorts of low back pain from among the numerous patients who present with "non-specific low back pain."

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Ozone depletion and skin cancer

SIR.—The report on the alarming levels of chlorofluorocarbons (CFCs) over North America and Europe in recent months¹ included reference to the prediction from the United Nations environment programme² that depletion of stratospheric ozone by chemical reactions involving the degradation products of CFCs will lead to a rise in the incidence of skin cancers as a consequence of increased levels of solar ultraviolet radiation at the earth's surface. Implicit in these estimates is that behaviour and time spent outdoors remain unchanged in populations at risk.

Unlike agricultural and marine ecosystems, which are also at risk from the potential effects of increased ultraviolet radiation,³ humans have the opportunity to modify their behaviour and so their exposure. By combining a behavioural model of human exposure to solar ultraviolet radiation with total ozone trends for United Kingdom latitudes obtained from satellite data⁴ and the expected increase in terrestrial ultraviolet radiation conse-