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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1 Gourlay GK, Boas RA. Fatal outcome with use of rectal morphine for postoperative pain control in an infant. *BMJ* 1992;304:766-7. (21 March.)
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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1 Gourlay GK, Boas RA. Fatal outcome with use of rectal morphine for postoperative pain control in an infant. *BMJ* 1992;304:766-7. (21 March.)

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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1 Gourlay GK, Boas RA. Fatal outcome with use of rectal morphine for postoperative pain control in an infant. *BMJ* 1992;304:766-7. (21 March.)
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3 Royal College of Surgeons of England and College of Anaesthetists. *Report of the working party on pain after surgery*. London: RCS and College of Anaesthetists, 1990.
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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