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Sir,—Drs Kitching and Rice describe an interesting case of puerperal extradural abscess after normal delivery without extradural analgesia. A similar case was described by Male and Martin [1], who reported an extradural abscess that developed 6 days after normal delivery in a 22-yr-old woman who also had not received extradural analgesia. This patient had been admitted 10 days before delivery suffering from infective hepatitis with hepatocellular dysfunction and jaundice, whereas the patient described by Drs Kitching and Rice was presumably healthy.

I agree that it is possible for an extradural abscess to occur after extradural catheterization without a causal relationship existing. However, I would emphasize several points. The incidence of spontaneous extradural abscess in the general hospital population is estimated at 0.2–1.2 per 10000 which, indeed, is greater than the reported incidence of extradural abscess after lumbar extradural analgesia in obstetric patients, as pointed out by Drs Kitching and Rice. However, these populations are not equivalent. Closer inspection of reports within the general population demonstrates a high incidence of co-existing chronic disease, which is a risk factor for compromised immunity, and a predilection for the older age group, the majority of cases being patients older than 50 yr [2, 3]. In contrast, the average parturient is young and relatively healthy compared with the general population. Therefore a direct comparison of relative incidence between the obstetric population and the general population is inappropriate and statistically unsound.

I believe there is strong evidence of a causal relationship between the use of extradural anaesthesia and the development of an extradural abscess in our patient. This is supported by a close temporal relationship, correspondence of the location of the abscess and the site of extradural catheter insertion, and growth of the likely infective organism from a skin lesion at the puncture site.

Whilst Drs Kitching and Rice have demonstrated that extradural abscess can develop in the puerperium without obvious cause, I believe that extradural catheterization remains a risk factor for development of this serious condition in obstetrics and in other specialties, and would reiterate the importance of meticulous aseptic technique when performing these procedures.

Drs Vasdev and Leicht make pertinent comments regarding aseptic technique for extradural catheter insertion. However, I would draw to attention possible neonatal complications that may arise from maternal transdermal absorption of iodine from iodine-containing skin disinfectants. Neonatologists in our institution have shown recently that mothers exposed to topical iodine-containing antiseptics immediately before delivery had concentrations of iodine in their breast milk twice those of non-exposed mothers, and that this appeared to be linked to an increased incidence of transient neonatal hypothyroidism [4]. Iodine absorption would probably be minimized by the practice of rinsing off the iodine with 70% alcohol as recommended by Vasdev and Leicht, but in our delivery suite it is now policy to avoid completely the use of iodine-containing skin disinfectants.

We have favoured polyurethane dressings such as Tegaderm (3M) as dressings for extradural catheter sites because of their excellent fixation properties. Vasdev and Leicht cite evidence that these dressings may increase bacterial colonization, which indeed is of concern; we are currently reappraising usage in our practice.

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THE LARYNGEAL MASK AND INTRAOCULAR SURGERY

Sir,—We were interested in the article of Lamb, James and Janicki on the effect of the laryngeal mask airway (LMA) [1] on intraocular pressure [2]. In their conclusion, the authors mentioned the potential risk of loss of airway control with the LMA during general anaesthesia for intraocular surgery. This risk is not theoretical, but a practical disadvantage of the method, as the anaesthetist does not have access to the patient's airway during surgery.

We describe two patients who recently underwent intraocular surgery under general anaesthesia (thiopentone, fentanyl, enflurane in an oxygen–nitrous oxide mixture and vecuronium). In both patients, insertion of the LMA was easy and resulted in a tight seal, which allowed mechanical ventilation (with peak inspiratory pressure less than 15 cm H_2O) [3]. The LMA was secured with adhesive strips. The surgical procedure for the first patient was initially uneventful, but the LMA was displaced slightly toward the end of the procedure. This resulted in a massive gas leak that led to a dramatic decrease in the expiratory tidal volume (less than 100 ml). Airway control was resumed temporarily by pushing down the LMA, but the leak recurred almost immediately. The LMA was then held in place by the anaesthetist's hand under the drapes and this allowed completion of the procedure. Hypoxia was not detected by pulse oximetry. Recovery was uneventful. In the second patient, a gas leak became evident before the beginning of the procedure, but after drapes, instrumentation and microscope were in place. Because expiratory flow was not detected in the breathing system, the drapes were removed hurriedly. The LMA was removed, the patient's lungs were ventilated using a face mask, additional thiopentone (150 mg) given i.v. and the trachea intubated. Hypoxia was not detected. In both patients there were no harmful consequences.

We had chosen to control the airway during general anaesthesia for intraocular surgery with the LMA because, as Lamb, James and Janicki have shown, this could blunt any increase in intraocular pressure. However, a minor incident (displacement of the LMA) could be transformed into a potentially disastrous accident. In our opinion, the inability of the anaesthetist to gain convenient control of the patient's airway during the procedure is a major hazard. Because of our experience, we have discontinued the use of an LMA for airway control in intraocular surgery.

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BRAIN PARTIAL PRESSURES OF ISOFLURANE AND NITROUS OXIDE

Sir,—We read with interest the article by Dr Thornton and colleagues [1], but wish to comment on the methods used. Thornton's group used isoflurane or nitrous oxide, changed randomly in three consecutive 10-min periods. They do not state which anaesthesia system or what fresh gas flows they used.

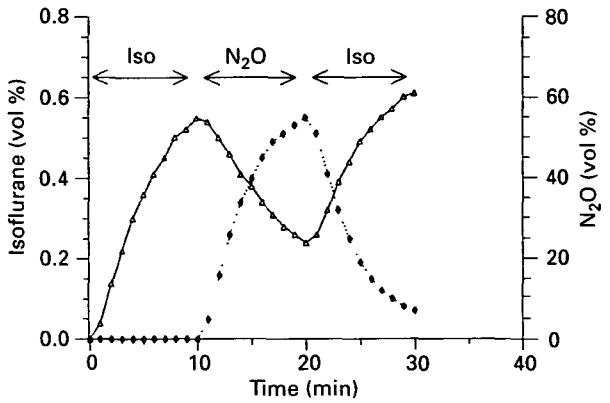

FIG. 1. Computer simulation of the brain partial pressures of isoflurane (△) and nitrous oxide (◆) during three 10-min periods in which either isoflurane (0.65–0.70 % end-tidal) or nitrous oxide (60–65 % end-tidal) was administered. Time = Time after beginning of anaesthesia. Iso = Isoflurane.

We have simulated an isoflurane–nitrous oxide–isoflurane anaesthesia period as described by Thornton and colleagues, with the GUS computer simulation program (Gas Uptake Simulation, iMedEd Inc, Phoenix, Arizona, U.S.A.) using a circle system and fresh gas flows of 12 litre min^{-1}. We agree that, when changing from isoflurane to nitrous oxide after the first 10-min period, stable end-tidal nitrous oxide values can be obtained within 5 min, after which the end-tidal partial pressure of isoflurane would not be less than 5 % of the initial value as stated by Dr Thornton and colleagues, but 0.23 vol % decreasing to 0.15 vol % at the end of the nitrous oxide period. Even with fresh gas flows of 20 litre min^{-1}, the residual end-tidal isoflurane washout from the brain would be even slower (fig. 1). Five minutes after the change from isoflurane to nitrous oxide, the brain partial pressure would be 0.38 vol %, decreasing to 0.24 vol % at the end of the nitrous oxide period. When, as in the first 10-min period, the end-tidal concentration of isoflurane is changed from 0 to approximately 0.70 vol %, a 10-min equilibration period does not produce stable brain partial pressures of isoflurane.

We would recommend longer equilibration periods in order to obtain stable conditions before measurements are performed.

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Sir,—Thank you for inviting us to reply to the interesting letter from Drs Petersen-Felix and Zbinden. We have used a similar program, Narkup [1], which was produced at Northwick Park, to reproduce exactly the brain concentrations shown in their figure, when the alveolar concentrations of isoflurane and nitrous oxide were maintained at 0.6 % and 60 %, respectively. Clearly, both these programs use, for modelling of the "brain compartment", blood flow, mass and partition coefficient based on whole brain values. This is a good example of the need to know exactly what assumptions are made in pharmacokinetic models. When Narkup is re-run using, for example, assumptions that the effect compartment is in the cerebral grey matter (40 % of the brain), with a high proportion of the blood flow (say 70 %), then brain concentrations are stable for the whole of the test periods. Of course, we have no means of knowing if the above assumptions are true, but the writers of each program also do not know.

The end-expiratory concentrations of both gases were measured directly either by the Capnomac, or by a mass spectrometer in both our 1992 paper, and a previous study which used the same methodology [2]; in both studies, end-expiratory concentration had decreased to less than 5 % of the original concentration before the test period for the next gas. The anaesthetic was delivered from a Manley MP3 (non-rebreathing) as a circle system would have been inappropriate to produce the rapid changes in concentration that we needed. Again, any computer modelling would be dependent on assumptions about minute volume and cardiac output.

The particular point about the three-period statistical construction that we chose [3] is that it effectively eliminates errors which are caused by carry-over from one period to another.

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HYPERTONIC SOLUTIONS IN MASSIVE HAEMORRHAGE

Sir,—We read with interest the case report by Dr Ngan Kee and transfusion by Donaldson, Seaman and Park [1] and would commend the authors on providing the reader with an excellent practical approach to the initial management of the acutely bleeding, hypovolaemic patient. However, we feel that a discussion on the crystalloid/colloid controversy should also now include some comment on the use of hypertonic solutions.

As the authors state, the success of initial resuscitation in the acutely hypovolaemic patient depends upon speed and adequacy of repletion. There has been increasing interest in the use of hypertonic solutions in the prehospital management of patients with traumatic hypovolaemic shock [2, 3]. The advantage of hypertonic over isotonic solutions in early resuscitation is that adequate resuscitation can be accomplished rapidly with small volumes through peripheral i.v. lines until conventional fluid therapy can be initiated. The smaller volume of fluid may also reduce complications related to dilutional coagulopathy and thermal stress. Hypertonic saline–Dextran 70 has been shown to improve mean arterial pressure, cardiac output, oxygen delivery and urine output and to increase survival compared with Ringer's lactate solution in severely traumatized patients [3].

Hypertonic solutions are still at an early stage of clinical investigation and further work is required before their role in fluid resuscitation is evaluated fully [4, 5]. The anticipated problems of acute hypernatraemia, hypervolaemia and venous injury have not materialized in the studies published so far [4].

We would suggest that in acute massive haemorrhage where venous access may be limited and rapid restoration of arterial pressure is required, there may be a role for hypertonic solutions. We would be interested in the authors' views on this subject.

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