REFERENCES

Pulse Oximetry during Repair of Congenital Diaphragmatic Hernia
Sir,—The pulse oximeter is a useful addition to the anaesthetist’s armamentarium. This Journal recently addressed this issue in an Editorial (Hanning, 1985) and some specific clinical applications have been reported in the literature (Friesen, 1985). I have had an opportunity to use this monitor in a different clinical situation—the reduction of a congenital diaphragmatic hernia.

In our study of 45 patients, 80% of patients on a similar regimen required no or only one “top-up” for a mean infusion time of 7 h (range 3–13.5 h). After a preload of Hartmann’s solution 1 litre an extradural catheter was sited at the L3/4 space. A 3-ml test dose of 0.25% bupivacaine was given, followed 5 min later by a further 12 ml. An infusion of 0.125% bupivacaine was commenced 30 min later using an IVAC syringe pump set to deliver 10 ml h⁻¹.

If analgesia was inadequate, a bolus of 10 ml of infusate was given and repeated, if necessary. The patient’s arterial pressure was recorded initially at 5-min and then at 30-min intervals (Datascope: Accutor). The patients were positioned to avoid aorto-caval compression.

The mothers were assessed hourly by the anaesthetist for analgesia, motor blockade and levels of sensory analgesia. Co-operation of the mother and her participation in the delivery were assessed by the midwife. Post-natally, mothers were interviewed for their evaluation of benefit during labour and delivery and to enquire as to the duration of blockade after delivery. Fifty-six percent of mothers had good pain relief with the infusion alone for a mean infusion time of 6 h 30 min until delivery (range 3–12.5 h). Twenty-four percent of mothers needed only one extra bolus of infusate during a mean infusion time to delivery of 8 h (range 3 h 12 min–13 h 30 min). Twenty percent of mothers needed more than one bolus. Mean infusion time to delivery was 10 h 42 min (range 3 h 5 min–16 h 15 min).

In general, sensory level regressed with time; hence, more bolus doses were required for the longer labours. No patient had motor blockade intense enough to prevent her turning herself. There was good cardiovascular stability and no complication relating to the extradural.

The infusion was continued until after delivery, providing good analgesia with effective maternal effort. Most mothers were fully mobile shortly after discontinuation of the infusion. This system provides safe and effective continuous analgesia and approaches the ideal for both 1st and 2nd stage labour. Perhaps the most significant aspect of this method was that the midwives considered it advantageous for both mothers and themselves. This is particularly important, since midwives are practitioners in their own right and their opinion is very much valued.

G. L. Van Hasselt
P. Rogers
Southampton

REFERENCES

LOW DOSE BUPIVACAINE INFUSION
Sir,—We read with interest the work by Drs Li, Rees, and Rosen (1985) on continuous extradural infusion of bupivacaine and we would like to confirm their conclusion that an infusion of 0.125% bupivacaine 10 ml h⁻¹ is optimal.

In our study of 45 patients, 80% of patients on a similar regimen required no or only one “top-up” for a mean infusion time of 7 h (range 3–13.5 h). After a preload of Hartmann’s solution 1 litre an extradural catheter was sited at the L3/4 space. A 3-ml test dose of 0.25% bupivacaine was given, followed 5 min later by a further 12 ml. An infusion of 0.125% bupivacaine was commenced 30 min later using an IVAC syringe pump set to deliver 10 ml h⁻¹.

If analgesia was inadequate, a bolus of 10 ml of infusate was given and repeated, if necessary. The patient’s arterial pressure was recorded initially at 5-min and then at 30-min intervals (Datascope: Accutor). The patients were positioned to avoid aorto-caval compression.

The mothers were assessed hourly by the anaesthetist for analgesia, motor blockade and levels of sensory analgesia. Co-operation of the mother and her participation in the delivery were assessed by the midwife. Post-natally, mothers were interviewed for their evaluation of benefit during labour and delivery and to enquire as to the duration of blockade after delivery. Fifty-six percent of mothers had good pain relief with the infusion alone for a mean infusion time of 6 h 30 min until delivery (range 3–12.5 h). Twenty-four percent of mothers needed only one extra bolus of infusate during a mean infusion time to delivery of 8 h (range 3 h 12 min–13 h 30 min). Twenty percent of mothers needed more than one bolus. Mean infusion time to delivery was 10 h 42 min (range 3 h 5 min–16 h 15 min).

In general, sensory level regressed with time; hence, more bolus doses were required for the longer labours. No patient had motor blockade intense enough to prevent her turning herself. There was good cardiovascular stability and no complication relating to the extradural.

The infusion was continued until after delivery, providing good analgesia with effective maternal effort. Most mothers were fully mobile shortly after discontinuation of the infusion. This system provides safe and effective continuous analgesia and approaches the ideal for both 1st and 2nd stage labour. Perhaps the most significant aspect of this method was that the midwives considered it advantageous for both mothers and themselves. This is particularly important, since midwives are practitioners in their own right and their opinion is very much valued.

G. L. Van Hasselt
P. Rogers
Southampton

REFERENCES
setting of 30 b.p.m. and positive end-expiratory pressure (PEEP) of 5 cm H₂O were maintained. Preoperative PaO₂ was 22.1 kPa. Dopamine hydrochloride 5–10 mg kg⁻¹ h⁻¹ was utilized to support the arterial pressure.

In addition to routine monitoring, a pulse oximeter (Nelcor, Hayward, CA) was applied to the right thumb during surgery. Management consisted of the continuation of dopamine, pancuronium and 100% oxygen. Ventilation was controlled by hand at a rate of 30 b.p.m. Peak inspiratory pressure (PIP) was kept below 20 cm H₂O. Initial oxygen saturation (SaO₂) was 75%. Several times during the procedure SaO₂ decreased to less than 60%. The ventilatory rate was increased and PEEP was added. After the reduction of the hernia, SaO₂ increased to 98–99%. At this time the heart rate increased from 160 to 200 beat min⁻¹ and 0.5% halothane was administered. After closure of the wound the patient was transferred to the Intensive Care Nursery in an isolet with the oxygen saturation of 98–99%. At this time the heart rate increased from 160 to 200 beat min⁻¹ and 0.5% halothane was administered. After closure of the wound the patient was transferred to the Intensive Care Nursery in an isolet with the oxygen saturation monitor in place.

After the operation the initial PaO₂ was 10.1 kPa on 100% oxygen and ventilation was increased progressively to 150 b.p.m. with only moderate improvement. Five hours into the postoperative period the PaO₂ increased to 52.5 kPa, probably as a result of expansion of the left lung. Inspired oxygen was decreased to 30% over the next few hours. The patient had a smooth recovery and the tracheal tube was removed on the 4th day after operation. The child was discharged on the 7th day after operation.

The pulse oximeter was a great help to assessing respiratory status. Our greatest challenge was maintaining SaO₂ greater than 60%, while keeping PIP less than 20 cm H₂O. The two occasions on which the SaO₂ decreased to less than 60% were directly related to a decrease in ventilatory rate produced by the anaesthetist. There are two concerns here. First, that hypoxia will cause return to a fetal circulation via re-opening of the ductus arteriosis. Second, increases in PIP can cause contralateral pneumothorax. Many sources recommend PIP be kept less than 20–30 cm H₂O (Bray, 1979; Dierdorf and Krishna, 1981; Stehling, 1982). In addition, the manometer pressure reading can be misleading if there is an obstruction in the endotracheal tube (Smith, 1980). The pulse oximeter aided us in maintaining oxygenation within this narrow range. If pneumothorax occurred, the SaO₂ would be expected to decrease along with changes in breath sounds, colour and blood-gas tensions (Smith, 1980).

When abdominal contents are replaced into the abdominal cavity, closure of the surgical wound causes increased intracavity pressure. This may lead to cephalad displacement of the diaphragm and reduced functional residual capacity (Dierdorf and Krishna, 1981). If this displacement is sufficient to produce changes in oxygenation, it should be reflected in a change in SaO₂. While this did not occur in our patient, the oximeter can aid the anaesthetist and surgeon in determining the need for creating a ventral hernia to relieve tension on the diaphragm.

Finally, we used the monitor during transportation of our patient from the operating theatre to the intensive care nursery. It gave an excellent guide to the effectiveness of ventilation and the adequacy of tissue oxygenation. There was no manometer on the Ambu bag used for transportation. We therefore maintained SaO₂ between 90 and 95% during transport, with the lowest possible inflation pressure.

E. A. Norman
New Jersey

REFERENCES


FRESH GAS REQUIREMENT OF THE T-PIECE SYSTEMS

Sir,—I feel I must point out that I have been misquoted in recent articles by Dr Hatch and others, concerning the fresh gas flow requirement of the T-piece systems (Hatch, 1985; Lindahl, Charlton and Hatch, 1985). In these articles I am said to have suggested that the fresh gas flow for the Bain and Jackson Rees systems need not exceed the minute volume during spontaneous ventilation—a claim which, to my knowledge, has never been made.

In our evaluation of the Bain system (Meakin and Coates, 1983), a mathematical model was constructed from the pneumotachograph traces of anaesthetized patients in order to evaluate the degree of rebreathing (percentage of tidal volume rebreathed) at selected fresh gas flow rates. Our results confirmed earlier reports (Willis, Pender and Mapleson, 1975) that a flow rate greater than twice minute volume was required to prevent rebreathing completely, but we were also able to show that a flow rate of 1.5 times minute volume resulted in rebreathing less than 10% of the tidal volume, which had no effect on respiration. As minute volume is usually depressed to two-thirds of its normal value during inhalation anaesthesia (Nunn, 1960), it is apparent that a flow rate equal to the normal minute volume, being 1.5 times the actual minute volume, should be sufficient to prevent significant rebreathing.

In adults, the normal minute volume may be taken as 100 ml kg⁻¹. In children, values of normal minute volume can be obtained from tables based on weight and height (Engström and Herzog, 1959). However, when selecting the fresh gas flow rate for use with a T-piece, it is sufficient to remember that the value of the normal minute volume in litre min⁻¹ is at least the square root of the weight in kilograms. Thus for a T-piece, a child weighing 9 kg will require a fresh gas flow rate of 3 litre min⁻¹, a child of 16 kg will require 4 litre min⁻¹ and so on, giving a range of flow rates for paediatric patients of 3–8 litre min⁻¹.

May I conclude by complimenting Dr Hatch and his colleagues on having moderated their views on the need for excessively high gas flows with the T-piece systems (Lindahl, Charlton and Hatch, 1984) and trust that my views on the issue have been clarified.

G. Meakin
Manchester

REFERENCES