
Sir,—We thank Professor Sjoberg, Mr Kjellgren and Dr Gupta for their comments. Our study [1] was conducted in normal human volunteers. The intention was to mimic, as closely as possible, the clinical situation in which nitrous oxide is administered in oxygen, without the introduction of a third gas, for example nitrogen. Therefore, we feel that our results reflect accurately what occurs in routine clinical practice. In studies using normal human volunteers, Kety and Schmidt [2] and Lambertsen and colleagues [3] did indeed show a decrease in cerebral blood flow when 100% oxygen was inspired. However, the accompanying hyperventilation and hypocapnia could alone have been responsible for the documented changes in cerebral haemodynamics [2]. Our volunteers did not hyperventilate.

While we accept that greater concentrations of oxygen may cause global cerebral vasodilatation and certainly cortical vasodilatation in animal studies, the situation in normal humans is far less clear and warrants further investigation.

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Pressure in the extradural space

Sir,—I read with interest the letter by Guedj [1] describing an ingenious device for detecting negative (subatmospheric) pressure within the extradural space. However, Telford and Holloway [2] have clearly shown that, in the lumbar region at least, the pressure in the extradural space is always positive (supra-atmospheric). An artefactual negative pressure is produced only as a result of tenting of the dura by the blunt Tuohy needle.

This would suggest that techniques which rely on eliciting a negative pressure to locate the extradural space are more likely than those using loss of resistance to result in accidental dural puncture.

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Sir,—The question of the pressure in the extradural space, first raised by Janzen in 1926, has been a subject of much controversy, especially regarding negative pressure. Does it really exist, and if so, does it occur naturally or is it caused as an artefact by the devices used to identify the space?

Bromage, after a comprehensive study of the literature, concluded in 1978 that the pressure in the extradural space in the lumbar region is positive unless the patient lies head-down in the lateral position; whereas negative respiratory pressure is transmitted to the thoracic extradural space [1].

Many methods of identifying the extradural space have been described, some based on the existence of negative pressure (such as the hanging-drop technique of Gurtierez), others depend upon recognition of sudden release of resistance to injection of fluid (such as the "technique du mandrin liquide" proposed by Sicard, later popularized by Dogliotti and called the "loss of resistance test").

However, every technique has advantages and drawbacks. For example, in the Gurtierez technique, the sucking of the drop of liquid is elicited in 82% and the hanging-drop technique lacks reliability in the lumbar region. On the other hand, the loss of resistance test is difficult to use in the thoracic and cervical regions.

The Macintosh balloon indicator, on which our device is based, seems to resolve most problems. As the pressure in the indicator balloon is always greater than that of the extradural space, regardless of the spinal level (thoracic, cervical or lumbar) or of the patient's position, abrupt deflation indicates entry into the extradural space in all patients. A further advantage of this technique is that both hands of the anaesthetist can be used for careful needle advancement.

Finally, we do think that a small percentage of failures (i.e. accidental dural puncture) is less related to the technique, than to the skill and experience of the practitioner.

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Respiratory depression after extradural fentanyl

Sir,—Following earlier correspondence [1], we wish to comment further on respiratory depression after extradural fentanyl. Three cases [2-4] have now been documented in the obstetric anaesthetic literature and although it is tempting to speculate on common factors and the mechanisms involved, the important message is that it can occur and it can occur relatively late after extradural injection. Drs Chrubasik, Chrubasik and Black were mistaken when they stated that one of the patients we reported received diamorphine in addition to extradural fentanyl [1]. In our opinion the events described in these three obstetric patients are not explained on the basis of additional systemic drug administration and direct access of fentanyl to the brain stem caused by rostral spread in cerebrospinal fluid is the likely mechanism.

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Sir,—We apologise to Dr Noble and colleagues for misrepresenting the treatment of one of their reported cases of ventilatory depression after extradural fentanyl. I.v. supplements of diamorphine (or other opioid) were given to some of the patients reported in their study and, in particular, to two in the group in which two patients needed naloxone. We clearly put two and two