CORRESPONDENCE


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, Ketty 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 vasodistraction and certainly cortical vasoconstriction 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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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 together.