This is based on the incorrect assumption that the central compartment is homogeneous and that instantaneous mixing occurs within it [6].

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IDENTIFICATION OF THE INTERPLEURAL SPACE
Sir,—We were interested to read the paper by Dr Scott in which he described a technique to locate the interpleural space using a saline infusion [1]. The same principle is applied in the "falling column" sign—a method we described previously to identify the interpleural space, and thus should minimize the incidence of pneumothorax from needle misplacement. The cases of pneumothorax reported in the literature were associated with mechanical ventilation [3,4] or, in one case, unexpected movement by the patient [5]. We prefer, therefore, to insert interpleural catheters in awake, spontaneously breathing patients. The interpleural pressure remains negative throughout the ventilatory cycle and the need for disconnection and infusion methods provide a clear and distinct end-point for identifying the interpleural space, and thus should minimize the incidence of pneumothorax from needle misplacement.

It would appear that both the falling column and saline infusion methods provide a clear and distinct end-point for identifying the interpleural space, and thus should minimize the incidence of pneumothorax from needle misplacement. The cases of pneumothorax reported in the literature were associated with mechanical ventilation [3,4] or, in one case, unexpected movement by the patient [5]. We prefer, therefore, to insert interpleural catheters in awake, spontaneously breathing patients. The interpleural pressure remains negative throughout the ventilatory cycle and the need for disconnection of the ventilator is obviated.

The use by Dr Scott of the catheter sheath adaptor through which the catheter passes is to be commended, as it would appear to eliminate altogether the risk of entry of air into the interpleural space.

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HEADACHE AFTER CONTINUOUS SPINAL ANAESTHESIA
Sir,—We read with interest the paper by Kestin and colleagues [1] comparing continuous spinal with extradural anaesthesia for elective Caesarean section. The use of continuous spinal anaesthesia for these patients is not new, but it has never achieved great popularity, presumably because of the high incidence of postoperative spinal headache; Giuffrida and colleagues [2] quoted an incidence of 16% using a 21-gauge needle. It was disappointing to note that the use of a 26-gauge needle and 32-gauge spinal catheter was associated with a 10% incidence of headache.

We observe, however, that it is possible to thread the 32-gauge catheter through a 24-gauge pencil-pointed Sprotte needle. This is a variation on the older Whitacre needle. A recent study found no postoperative headache in a series of 55 obstetric patients given spinal injections through these needles [3]. This, rather than smaller, diamond-tipped needles, may be a more fruitful avenue of research in attempting to reduce further the incidence of headache, while maintaining the undoubted advantages of a continuous spinal anaesthetic technique.

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Sir,—Thank you for the opportunity of replying to the letter from Drs Milligan and Carp. In our study, there were two of 20 mothers in the spinal group with a headache typical of that after lumbar puncture. The headaches were mild, discovered only by direct questioning, and the mothers did not need to restrict their activity. Two other mothers in the spinal group, and three in the extradural group had headaches that were not typical of lumbar puncture headache, but had symptoms at least as severe as the two “spinal” headaches. In our small study, spinal headaches were not a significant clinical problem.

The 32-gauge catheter can be threaded through a 24-gauge Sprotte needle, but the hub design of those currently available is not suitable for this purpose. Modified 24-gauge Sprotte needle catheters will be available from the manufacturer (Rusch U.K.) in the next few months. There are technical difficulties in threading the 32-gauge catheter through 26-gauge needles using a midline approach [1]. In our study, we had no difficulty using an oblique paraspinous approach. A 24-gauge Sprotte needle may overcome these technical difficulties and reduce the incidence of spinal headache, but this remains to be determined.

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REFERENCE

MIDAZOLAM AND ERYTHROMYCIN

Sir,—I was interested to read the clinical report by Hiller and colleagues describing unconsciousness associated with the use of midazolam before erythromycin given for antibiotic prophylaxis before adenoidectomy [1]. The plasma concentration of midazolam was greater in this patient than in six other children who did not receive erythromycin. Erythromycin has been shown previously to inhibit the metabolism of theophylline [2] and alfentanil [3].

It has been determined that elimination of the immunosuppressive drug cyclosporine, depends on its metabolism in the liver by cytochrome P450 II A [4]. Cytochrome P450 II A also catalyses the N-demethylation of erythromycin and the erythromycin breath test (the ability of the patient to demethylate erythromycin to produce exhaled 14C-carbon dioxide after i.v. 14C-N-methyl-erythromycin) has been used as a predictor of blood concentrations of cyclosporine in patients [4,5]. When the erythromycin breath test was administered to 30 hospital inpatients, there was a four- to six-fold range of breath test values, indicating marked inter-individual variability [4]. Thus for cytochrome P450 II A there appears to be a broad unimodal distribution of enzyme activity in the population, in contrast with the bimodal distribution of enzyme function seen in debrisoquine genetic polymorphism, in which the ability to metabolize debrisoquine is impaired in 8-10% of the Caucasian population (poor metabolizers).

There is in vitro evidence that midazolam also is metabolized by cytochrome P450 II A [6,7]. Midazolam itself shows considerable variation in its pharmacokinetic parameters, possibly resulting partly from genetic polymorphic drug metabolism. I suggest, therefore, that the increased concentration of midazolam in the patient described in the case report by Hiller and colleagues was a result of inhibition of cytochrome P450 II A, perhaps in a patient with reduced cytochrome P450 II A function.

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