CORRESPONDENCE

UNILATERAL EXTRADURAL BLOCKADE IN OBSTETRICS

Sir,—The hypothesis advanced by Narang and Linter [1] concerning the cause of unilateral extradural blockade is intriguing. However, I must take exception to their mathematics. Using the data from their table I, I have calculated the percentage of unsatisfactory blocks at each S–EDS (skin–extradural space distance) (table I).

Spearman’s rank correlation remains the same, but the authors’ statement that there is a greater than 40% chance of producing an unsatisfactory block if the extradural space has not been identified before a depth of 6 cm, must be challenged. Calculations from their data (table I) put this figure at 33.7%.

S. ZBINDEN
Zurich

REFERENCE

Sir,—Drs Narang and Linter [1] attempted to shed some light upon failed extradural blocks in obstetric practice and postulated that lateral placement of the catheter was responsible, resulting from deviation of the tip of the needle from the midline. Use of the paramedian approach may result in lateral placement of the catheter as or more frequently than the midline approach. However, in a prospective study of 165 patients, no difference was found in the incidence of failed blocks between the midline and paramedian approaches [2].

In obese patients, with a large skin to extradural space distance (and therefore greater likelihood of lateral placement of the catheter) there may be difficulty in location of the midline. The paramedian approach relies less upon identification of the midline [3] and, since the needle tip is angled medially after contact with the lamina propria, is unaffected by the skin to extradural space distance for placement of the catheter. Therefore, for patients in whom a large skin to extradural space distance is anticipated, the paramedian approach may be preferred.

R. M. GRIFFIN
London

REFERENCES

Sir,—Dr Zbinden is correct. Unfortunately we made a transcription error and substituted failure/success % instead of failure/total % in column four of table I.

Our own preference for difficult obstetric extradurals is with the patient sitting. Scrupulous attention should be paid to monitoring a midline approach.

S. P. K. LINTER
Southampton

PRICK TESTS TO DIAGNOSE ANAPHYLAXIS

Sir,—Following our study of prick tests in the diagnosis of anaphylaxis caused by general anaesthetics [1] we have simplified the method. In 34 patients (30 female, 4 male; ages 7–73 yr) who were referred to our department between April and June 1987 for skin tests to anaesthetics, we have compared prick tests with neuromuscular blockers (suxamethonium, gallamine, alcuronium, pancuronium and vecuronium) with prick tests that were made with a mixture of these same five drugs (final concentration = 1:5 of each).

Eleven patients who had an anaphylaxis (group 1) and 23 patients who had been previously anaesthetized without anaphylaxis (group 2), were considered by the anaesthetist to be “at risk”.

In group 1, prick tests were positive (weal ≥ 5 mm, flare ≥ 10 mm) for at least one of the five neuromuscular blockers in all patients and positive with the mixture in 10. Prick tests were all negative in group 2 and so the results of the prick tests with the mixture were in agreement with those from the

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<th>Table 1. Numbers of unsatisfactory blocks</th>
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<td>S–EDS (cm)</td>
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individual tests in 33/34 patients (Chi squared with Yates' correction = 24.8, P < 0.001).

This work shows that there was no haptenic inhibition with the mixture—that is, a neuromuscular blocker that did not give a positive result itself in a particular patient did not inhibit a positive reaction from another blocker in the same patient—and that a single prick test with a mixture of the compounds may be used to detect allergic patients (1 in about 3000) more easily and cheaply than with individual tests with each drug.

F. LEYNADIER
Paris

REFERENCE

CARDIAC ARREST FOLLOWING I.V. VERAPAMIL MAY BE RELATED TO CONCOMITANT DIGOXIN THERAPY AS WELL AS HALOTHANE

Sir,—The recent case report published in the Journal [1] attributed cardiac arrest during halothane anaesthesia to the drug interaction between halothane and verapamil. I would like to suggest that another drug which the patient was reported to be receiving might also be responsible for this unfortunate occurrence.

Exactly the same course of events has been reported in a patient who was being chronically treated with digoxin and received the same dose, 5 mg, of verapamil i.v., but who was not anaesthetized with halothane [2]. Although halothane can interfere in A-V conduction, interaction with verapamil is not as prominent as that seen with enflurane. Both our laboratory [3,4] and Kapur and colleagues [5] have reported this. However, in both instances very high plasma concentrations of verapamil were necessary for the interaction (greater than 200 ng ml⁻¹). In addition, the interaction of verapamil given i.v. in more than twice the dose reported in the letter [1] has been associated with minimal haemodynamic effects with MAC concentrations of all three inhalation anaesthetics [4,5]. Only the markedly invasive, highly instrumented preparation of Ramsay and colleagues [6] and the anecdotal observations of Pryς-Roberts [7] have suggested a deleterious interaction between clinical concentrations of halothane and i.v. verapamil.

Obviously, a potent drug such as verapamil must be used with some caution at all times, and particularly during anaesthesia with the cardiac depressant inhalation anaesthetics. However, in my opinion, the conclusions of Moller are not justified by either his case report or a careful review of the literature [8].

R. G. MERIN
Houston

REFERENCES

Sir,—Thank you for the opportunity to reply to the letter by Dr Merin.

I agree that the concomitant treatment with digoxin might have influenced the occurrence in the patient receiving i.v. verapamil followed by halothane anaesthesia, all three interfering with the A-V conduction.

However, also based upon the results of Chelly and colleagues [1] obtained in dogs, which was published after I submitted my letter, I still feel that my conclusion is valid: caution should be exercised in the simultaneous use of the two drugs—and especially so, if given in high doses.

I. W. MOLLER
Copenhagen

REFERENCE

DEADSPACE AFTER CORRECTIVE CARDIAC SURGERY

Sir,—Yates, Lindahl and Hatch [1] found a large increase in physiological deadspace after corrective cardiac surgery in children with left to right (LR) shunting. They suggested altered pulmonary wall tension and "different working conditions for the hypertrophic arteries" after correction as an explanation.

Fletcher and Jogi [2] reported similar findings in 13 children and adults, 10 of whom had atrial or ventricular septal defects (ASD). The changes were caused by variation in the alveolar deadspace fraction. Its mean value increased from 0.14 to 0.25 after cardiac bypass, and then decreased to 0.21 (P < 0.01).

The increase in alveolar deadspace was associated with a flat phase III of the single breath test for carbon dioxide. This relatively unusual combination of findings (increased deadspace is usually associated with an increased slope) is also seen in pulmonary air embolism [3]. Therefore, we wondered if pulmonary air embolism was occurring during surgery. In two