Our experience over 4 yr with direct questioning of patients reassures us that hypnosis and haemodynamic stability are provided reliably by the second method. We are currently investigating the dose, and evaluating the haemodynamic effects of propofol for induction and maintenance of hypnosis in the presence of full opioid analgesia in patients undergoing cardiac surgery.

K. SHERRY
N. J. MASSEY
Sheffield

EXTRADURAL, SPINAL OR COMBINED BLOCK FOR OBSTETRIC SURGICAL ANAESTHESIA

Sir,—Dr Carrie, in an otherwise excellent review of regional anaesthesia in operative obstetrics [1], dismisses the practice of warming bupivacaine before extradural injection as being inconvenient and hardly merited because of a reduction of block onset time of only 20%. In this respect, he quotes our study [2] which recorded a reduction of 30% in onset time. Moreover, we demonstrated a 23% reduction in volume of local anaesthetic required to achieve satisfactory block, and a significant reduction in the incidence of shivering by injecting bupivacaine warmed previously to body temperature.

Maintaining a small supply of bupivacaine and 20 ml syringes in a thermostatically controlled warming cabinet (a standard fixture in any labour suite or operating theatre) does not impose any measure of inconvenience. There is no necessity to warm any other equipment and we continue to believe that this simple measure significantly improves the technique of extradural anaesthesia for Caesarean section.

D. A. DUTTON
J. E. HOWIE
Glasgow

REFERENCES

Sir,—I am grateful for the opportunity to thank Drs Dutton and Howie for their kind remarks about my review article [1] and to comment on the points they raise in their letter.

When discussing the effect of warming local anaesthetic solutions to increase the speed of onset of extradural block, I made reference not only to the work of Dutton and Howie [2], but also to that of Mehta and colleagues [3], whose reduction of onset time of block to T6 was only 21%. A "mean" of the two papers does give a reduction time of 25% so my figure of "about 20%" is only marginally low for the combined results, but underestimate by a few more per cent the 30% reported by Dutton and Howie. I apologize. Their observations on the effect of warming on reduction in volume of local anaesthetic and a reduced incidence of shivering were noted also by Mehta and colleagues, but my comments on the effect of warming were confined to the effect on rate of onset.

L. E. S. CARRIE
Oxford

COMPARISON OF DOPAMINE AND DOPEXAMINE

Sir,—The report by Stephan and colleagues [1] comparing the effects of dopexamine and dopamine on cardiovascular and renal haemodynamics gives rise to several areas of concern.

First, the doses of dopexamine and dopamine chosen were not comparable. The authors state that dopexamine has only 33% of the potency of dopamine at DA1 receptors, yet they chose to compare dopexamine 1, 2 and 4 μg kg"1 min"1 with dopamine 2.5 and 5 μg kg"1 min"1. Likewise, the dose ranges allow no direct comparison of cardiovascular variables between the groups. Changes in cardiac index and systemic vascular resistance occur with dopexamine at a low dose, whilst dopamine in greater doses does not cause a similar increase in cardiac index and decrease in systemic vascular resistance. Again, this may be predicted from the known receptor pharmacology of these agents. The authors comment on the acceptability of the increase in heart rate seen with dopexamine 4 μg kg"1 min"1, which resulted also in a 117% increase in cardiac index. We would conclude that, in this population of patients, this dose is unnecessary and excessive.

Second, the authors noted that the increase in renal blood flow was greater than that in cardiac index in those receiving dopamine, the reverse being the case with dopexamine. However, the increase in renal blood flow was not significantly greater with dopamine than with dopexamine, and the decrease in renal vascular resistance was greater with dopexamine.

All patients received calcium channel blockers and some were receiving β-blockers also. There is no comment on
CORRESPONDENCE

which agents were used, the doses, or when the last morning dose was given. This may be relevant for drugs with a long half-life, or if slow-release preparations have been administered. Furthermore, there is little discussion on possible effects of these agents on the results obtained. There is insufficient information to allow us to see how conclusions regarding the pharmacological receptors involved in changes in renal and cardiovascular haemodynamics were derived.

The authors state in the discussion that myocardial ischaemia has been reported with dopexamine, and incorrectly cite the work of Dawson and colleagues [2], who showed no alteration in myocardial oxygen demand or supply with increased rate-pressure products. Although two patients reported mild chest pain with 6 μg kg⁻¹ min⁻¹, there was no ECG evidence of myocardial ischaemia or a greater increase in myocardial oxygen consumption than the group mean. The absence of adverse effects of dopexamine on the myocardium has been observed by others [3]. However, the ST segment depression in this study, shown to occur in patients receiving dopamine [1] and dopexamine [2], emphasizes that caution should be exercised when using inotropic stimulation in patients with coronary artery disease.

G. Park
A. Burns
J. Pederson
Cambridge

REFERENCES


Sir.—We are aware of the fact that equipotent doses of dopexamine and dopamine are difficult to define because of differing receptor activities. This is the reason why we did not perform a statistical comparison between the groups. As we were more interested in renal than in cardiovascular effects of both drugs, we started with dopexamine 1 μg kg⁻¹ min⁻¹ and dopamine 2.5 μg kg⁻¹ min⁻¹ because these doses resulted in comparable increases in renal blood flow. Then we looked at the effects of twice these doses. Our results show clearly that, in contrast with dopamine, dopexamine increased renal blood flow mainly by an increase in cardiac index. Furthermore, we admit that (as with any other clinical study) we can only speculate on the different receptor potencies of both agents.

We observed that all patients were receiving maintenance doses of calcium channel blocking drugs and that two patients in the dopexamine group and three patients in the dopamine group were treated additionally with β-receptor antagonists. We stated that the last doses of all drugs were administered in the morning of the operation, but omitted to note that these drugs were nifedipine and atenolol. We did not comment on the doses, because oral doses do not allow any conclusions to be drawn on plasma concentrations.

We do not think that we cited the work of Dawson and colleagues incorrectly, because the common surface ECG is not always a sensitive marker of myocardial ischaemia. Berry and colleagues [1] examined the effects of selective regional myocardial ischaemia by occluding vessels during percutaneous coronary angioplasty. While ST elevation occurred in 84% of patients during occlusion of the left anterior descending artery and in 92% of patients during occlusion of the right coronary artery, only 32% of patients undergoing occlusion of the left coronary artery demonstrated ST elevation in the routine surface ECG; four of 19 patients of the latter group had only precordial ST depression, and nine (47%) patients had no ECG changes. Moreover, unaltered global myocardial oxygen consumption does not exclude regional myocardial ischaemia, which can be detected biochemically only by an impaired lactate balance, or even better, by sensitive markers of ischaemia, such as hypoxanthine [2].

H. Stephani
Göttingen

REFERENCES


MEMORY MECHANISMS

Sir,—I read with interest the Editorial in the November issue of the Journal, "Awareness and memory in anaesthetized patients". It contains a short review of memory mechanisms to which I wish to add the following comments.

First, the anatomical basis for memory formation was proposed long before Ramon y Cajal. There is, for example, an account in 1780 by the Italian anatomist Malacame [1] of an impressive experiment in which he trained one of two siblings of various species (dogs and several birds) and left the other sibling untrained. After a long period, he killed all the animals, examined the brains and reported that there were more folds in the cerebellum of the trained animals than in the untrained ones.

My second comment refers to the absence of any reference to cholinergic mechanisms within the hippocampus (and elsewhere in the forebrain) which mediate memory processes. As anaesthetists, we are very familiar with the effects of hyoscine on memory.

J. Ponte
London

REFERENCE