the abdominoplasty masked the level of block, as tested over the abdomen, and could have led to a mistaken belief that the epidural was not working or that a larger increment of local anaesthetic was required.

These two cases are unusual in the presentation of their regional block. However, knowing the anatomy of the one case and the past surgical history in the other it is easy to postulate a mechanism for the clinical picture.

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Vasopressors and hypotension

To the Editor:

We read with interest Dr. P. Morgan's review entitled "The role of vasopressors in the management of hypotension induced by spinal and epidural anaesthesia" (Can J Anaesth 1994; 41; 404-13) and would like to make the following comments.

There was no mention of the role played by the venous system in the genesis of hypotension during spinal blockade. Greene and Brull,¹ Macrae and Wildsmith² and our own findings³ all report that venous capacitance is increased during spinal blockade. Greene and Brull's¹ view was that venodilation causes a reduction in venous return to the heart and this is one of main factors causing hypotension. They strongly recommend the use of head-down tilt during spinal blockade to improve venous return. Our experience with elderly patients receiving subarachnoid block is that a moderate decrease in central venous pressure occurs, on average 2.5 cm H₂O, and this can be adequately reversed, in most cases, by giving 8 ml · kg⁻¹ colloid during the induction phase of the block.³

Dr. Morgan contradicts himself when discussing the use of ephedrine. He mentions that ephedrine has both α and β -adrenergic agonist actions and the "vasoconstriction is almost balanced by vasodilatation, and peripheral resistance is usually little changed." Later he states that ephedrine is capable of increasing cardiac output and of increasing peripheral resistance. We think that Dr. Morgan alludes to the fact that agonists with both α and β stimulating activity, such as ephedrine, are poor vasoconstrictors because of the opposing effects of simultaneous α (vasoconstriction) and β (vasodilatation) stimulation on the arterial beds.⁴ We found the same results in treating hypotension during subarachnoid block with ephedrine infusion in 30 elderly patients.⁵

Dr. Morgan refers to metaraminol as having both α and β stimulatory effects. This is misleading as the clinical effects of metaraminol are predominantly alpha. We have used metaraminol during subarachnoid block for several years and consider the dose of 5 mg *iv* to be excessive, despite being frequently quoted.^{6,7} In the non-obstetric elderly patient undergoing subarachnoid block, our current practice, after giving a moderate volume of colloid (8 ml · kg⁻¹), is to treat hypotension with a bolus of 0.5-1.0 mg (0.1-0.2 mg · kg⁻¹) followed by an infusion of 10 mg in 20 ml of normal saline run at 5-10 ml · hr⁻¹. Our experience with this dose is that metaraminol is highly effective at restoring preload and afterload during subarachnoid block but has little demonstrable effect on heart rate or contractility.⁸

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