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Sedation depends on the level of sensory block induced by spinal anaesthesia

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Summary

We have investigated the relationship between the extent of spinal block and occurrence of sedation. In a first series of 43 patients, the distribution of sedation score (measured on the Ramsey scale) was related to the extent of spinal block (pinprick). In a second series of 33 patients, the relationship between sedation score and spinal block persisted after injection of midazolam 1 mg. This study confirmed that high spinal block was associated with increased sedation. (*Br. J. Anaesth*. 1998; **81**: 970–971).

Keywords: anaesthetic techniques, subarachnoid; hypnotics benzodiazepine, midazolam; sedation

It is stated frequently that patients with spinal block may be drowsy, although they may not have received any sedative drugs.[1] It has been demonstrated also that high spinal anaesthesia increases sensitivity to the sedative effect of midazolam,[2–4] suggesting that spinal or epidural block has a sedative action. Therefore, we have conducted a study to investigate the effect of spinal block on patient awareness and to relate this effect to extent of block.

Methods and results

Initially, we studied 43 unpremedicated patients undergoing general surgery under spinal anaesthesia (mean age 57 (range 17–91) yr). Hyperbaric 0.5% bupivacaine was used for spinal anaesthesia in the dose range 5–20 mg, according to the surgical procedure requirements of the anaesthetist in charge. The level of sensory block was evaluated by pinprick at 5, 10, 20, 30 and 45 min after administration of bupivacaine. Sedation was estimated at the same time using the Ramsey scale (1 = anxious and agitated; 2 = cooperative and tranquil; 3 = drowsy but responsive to command; 4 = asleep but responsive to a glabellar tap; 5 = asleep with a sluggish response to tactile stimulation; 6 = asleep and no response) by an independent observer unaware of the extent of block.[4] Monitoring included heart rate, arterial pressure and Sa_{O_2}. Hypotension (defined as a decrease in systolic arterial pressure less than 90 mm Hg) was treated by ephedrine 3–12 mg i.v.

We then studied a second group of 33 patients (mean age 58 (range 25–83) yr) also undergoing surgery under spinal anaesthesia using 0.5% hyperbaric bupivacaine in the same dose range. After completion of spinal block, these patients were given an i.v. bolus of midazolam 1 mg. Sedation scores were determined before and 5, 10, 20, 30 and 45 min after administration of midazolam. Patients in this group were monitored as before.

In both groups we examined the relationship between maximum sedation score and maximum extent of sensory block and age. Data were analysed using a contingency table. $P<0.05$ was considered significant.

The maximum upper levels of sensory block were T2 and L3. A significant relationship was found between the maximum extent of sensory block and level of sedation in the first series of patients (fig. 1A) ($P<0.03$) and in the second series after administration of midazolam (fig. 1B) ($P<0.05$). Three patients in the first group and one in the second group experienced hypotension without worsening of sedation score. There were no episodes of oxygen desaturation. There was no relationship between age and extent of block.

Comment

Although sedation scores were not distributed in the complete range of the Ramsey scale, we have shown an association between the level of sensory block and degree of sedation. There was no association with age. I.v. administration of midazolam increased sedation scores but did not change the relationship between the extent of sensory block and degree of sedation. In agreement with previous studies,[2–4] and with daily clinical practice, we found that, especially when the block extended to the upper thoracic segments, after a small dose of midazolam (1 mg), some patients (27%) were unresponsive to verbal stimulation and only reacted to a light glabellar tap.

High spinal block may impair awareness because of hypotension or arterial oxygen desaturation. There were no such events in our study. A direct effect of local anaesthetic on the brainstem may explain sedation. Thus to avoid significant plasma absorption of local anaesthetic, we studied spinal instead of epidural anaesthesia. Nevertheless, Inagaki and colleagues have demonstrated that i.v. lidocaine did not produce sedation while epidural lidocaine did,[6] suggesting that the sedative effect was more likely to be caused by the anaesthetic block rather than the

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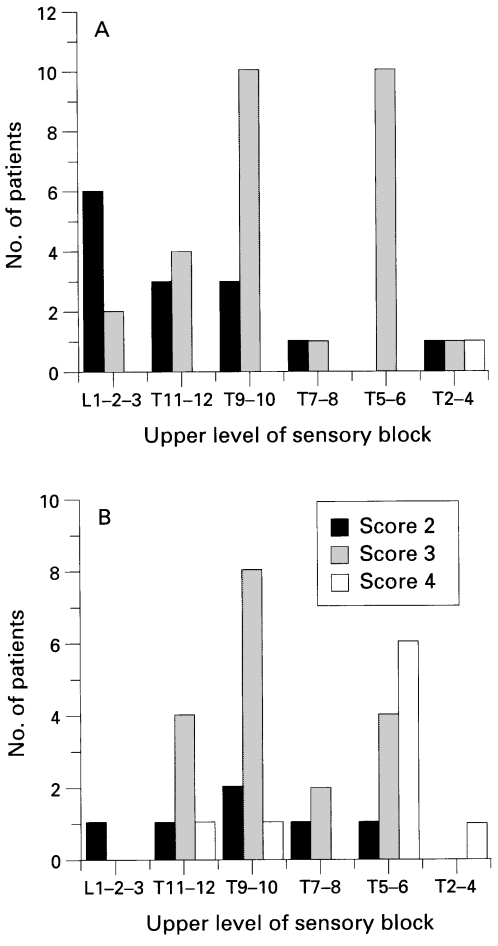

Figure 1 A: Sedation scores according to the level of sensory block ($n=43$). B: Sedation scores after administration of midazolam according to the level of sensory block ($n=33$).

effect of the local anaesthetic on the central nervous system.

Spinal block results in loss of proprioceptive inputs from muscles and joints which are thought to contribute to maintenance of awareness. Inagaki and colleagues also suggested that a decrease in nociceptive stimulation was implicated in the increased sensitivity to anaesthetic agents.[6] The lack of warm and cold sensations could also be partly implicated.

In summary, we have shown that high spinal block was associated with sedation. This confirms that sedation must be carefully titrated in patients with high central block.

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