EFFECT OF LATE POSTURE CHANGE ON THE LEVEL OF SPINAL ANAESTHESIA WITH PLAIN BUPIVACAINE

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SUMMARY

We studied 40 patients, 18-60 yr, undergoing orthopaedic surgery of the lower limb under spinal anaesthesia. A midline lumbar puncture was performed in the L3-4 interspace using a 27-gauge needle with the patient in the lateral horizontal position. Plain bupivacaine 3 ml at room temperature was injected. The cephalad level of analgesia was assessed by pinprick 60 min after injection of local anaesthetic, at the end of surgery and again after the patient was moved into bed. All patients had a segmental level of the block of L1-T5 at the beginning of the study. The upper half of the patient's body was then tilted to a 30° head-up position. Segmental spread was subsequently assessed by pinprick at 5-min intervals for 30 min. In six of the 40 patients (15%), increased cephalad spread of spinal analgesia occurred. The mean time from induction of spinal anaesthesia was shorter in these six patients (mean 92 min, range 80-115 min) than in the patients whose block did not change or was decreasing during the 30-min test (mean 119 min, range 83-210 min) (P < 0.05). We conclude that the patient should remain in the supine horizontal position until recovery from the spinal block.

KEY WORDS

investigators assessed the segmental spread of spinal analgesia to pinprick at 5-min intervals for 30 min. The subjective sensations of the patient were also recorded. Arterial pressure (oscillotonometry) and heart rate were measured at 5-min intervals during surgery and the study and at 10-min intervals thereafter. ECG was monitored continuously. After the study, the patients were again placed horizontal.

Mann-Whitney U test was used to test the difference between times from the subarachnoid injection of local anaesthetic to the study.

RESULTS
In six of the 40 patients (15%), increased cephalad spread of spinal analgesia was observed when the patients were placed in a 30° head-up position (fig. 1). The mean time from induction of spinal anaesthesia to the sit-up test was shorter in those six patients with an increase in the block (mean 92 min, range 80–115 min) than in the patients whose block did not change or was decreasing during the 30-min test (mean 119 min, range 83–210 min) (P < 0.05). The largest individual increase in spread was four segments. Two patients had a subjective sensation of an extension in block. One patient, who had an increase of one segment of the block (T12–T11) developed bradycardia (slowest heart rate 37 beat min⁻¹) and hypotension (smallest systolic pressure 75 mm Hg) and received atropine 0.5 mg i.v.

Five of the 40 patients had an extension of the block (two segments) after they were moved from the operation table to the bed. Only one of these patients belonged to the group of six patients who had extension of the block during the sit-up test, while the others had a regressing block. None of these five patients had any haemodynamic disturbances. During the 30-min test, the level of spinal analgesia regressed in 29 patients and there was no change in five others. One of the patients, whose block regressed from T7 to T8 in 10 min after the change to sitting became hypotensive (systolic pressure 93/64 mm Hg) and received atropine 0.5 mg i.v.

To our surprise, in six patients, the level of spinal analgesia increased up to four segments when the patient was placed 30° head-up 80–115 min after the injection of bupivacaine. Thus it seems that there was still sufficient unbound bupivacaine in CSF to produce sensory block, probably by mechanically induced displacement of CSF in a cephalad direction. It is generally believed that the baricity of an injected solution influences the spread of local anaesthetic molecules for only approximately 30 min [12]. In spite of this, it has been shown that a change in the position of patients during spinal block with hyperbaric local anaesthetic solutions affects the spread of the block relatively late after induction [4, 5]. Block level has been found to extend by up to eight segments 60 min after administration of hyperbaric bupivacaine 4 ml when the patient was turned from the sitting to the supine horizontal position [5]. Also, the lateral position for 45 min after injection of 2 ml of hyperbaric 0.5 % bupivacaine, 5 % lignocaine or 4 % mepivacaine first produced unilateral anaesthesia, which changed to almost symmetrical bilateral anaesthesia when the patient was moved to the supine horizontal position [4].

DISCUSSION
There were no major haemodynamic changes during surgery in any of the patients, and surgical blood loss was minimal. Nine patients received sedatives during induction of anaesthesia or surgery. One belonged to the group of six patients who had an extension of the block during the study. Only one patient received fentanyl (0.05 mg); he had an extension of block (one segment). In the recovery room, no sedatives or fentanyl were given before the tilt test.

The course of spinal anaesthesia during operation and recovery after the tilt test were uneventful.

FIG. 1. Changes of segmental level of spinal anaesthesia in six patients during the study.
local anaesthetic in the CSF than block of somatic sensory fibres [15]. Greene [17] suggested, therefore, that the sympathetic block extends two to four segments cephalad to the level of the sensory block. In contrast, Bengtsson, Löfström and Malmqvist [16] and Malmqvist and colleagues [17] have shown that the distribution of the sympathetic block may not exceed that of the sensory block during spinal anaesthesia. A change to the sitting position during sympathetic block of the lower half of the body may suddenly decrease the venous return to the heart. Acute activation of cardiac ventricular chemos- and mechanoreceptors (Bezold–Jarisch reflex) caused by the rapid decrease in ventricular volume has been suggested to be the aetiology of bradycardia in such instances. Atrial [18] or pacemaker stretch receptor reflexes [19] may also be involved. An extending sympathetic block possibly reaching the high thoracic level may impede the normal physiological compensatory cardiac response via unopposed vagal input. Unexpected bradycardia and even cardiac arrest have been documented in association with spinal anaesthesia without changes in position in patients with relatively high blocks [20–22]. As seen in our study, such cardiovascular complications may occur also when the sensory block reaches only the lower thoracic level.

Because the head-up tilt 80–115 min after subarachnoid injection increased the cephalad spread of the spinal block by one to four segments in six of our patients, we recommend the use of the supine posture change and level of spinal anaesthesia without changes in position in order to avoid unnecessary movements, when plain bupivacaine has been used for spinal anaesthesia.

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