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. (Br. J. Anaesth. 1993; 71: 807-809)

KEY WORDS

It is thought that the distribution of local anaesthetic solution in the cerebrospinal fluid is complete within 15-20 min [1-3]. However, after subarachnoid administration of hyperbaric 0.5 % bupivacaine [4, 5] and other hyperbaric local anaesthetic solutions [4], the spread of the block has been shown to change further 45-60 min later, when the patients were moved to another position. With plain 0.5 % bupivacaine, the cephalad spread of spinal block occasionally continues beyond 60 min [6-11]. We have observed recently two patients who had severe transient bradycardia and hypotension after re-positioning 80 and 125 min after subarachnoid injection of local anaesthetic. In both patients, the level of spinal anaesthesia was noticed to have increased by four to five segments (to T1 and to T6, respectively) compared with the levels at 60 min. We have therefore studied the effect of a 30° head-up tilt position on the level of spinal anaesthesia in the immediate postoperative period—that is, more than 60 min after the subarachnoid injection of plain 0.5% bupivacaine.

PATIENTS AND METHODS

We studied 40 ASA I–II patients, 18-60 yr, undergoing orthopaedic surgery of the lower limb (table I). The study was approved by the Institutional Ethics Committee and informed verbal consent was obtained. Patients with cardiovascular diseases were excluded. The patients received diazepam 5–15 mg orally 1–1.5 h before induction of spinal anaesthesia. When required during surgery, diazepam 2.5–5 mg was given for sedation, or fentanyl 0.05–0.1 mg i.v. for analgesia. A midline lumbar puncture was performed at the L3-4 interspace using a 27-gauge needle with the patient in the lateral horizontal position. Plain 0.5 % bupivacaine 3 ml at room temperature was injected. Thereafter the patients were turned gently to the supine horizontal position.

The cephalad spread of analgesia was assessed by pinprick. The segmental level of analgesia was recorded 60 min after injection of local anaesthetic, at the end of surgery and again after the patient was moved into bed (table I).

In the recovery room, the patient remained in the supine horizontal position for 15 min before the start of the study. All patients had a segmental level of the block in the range L1-T5 and the spinal block was stable or decreasing at the beginning of the test. The upper half of the patient's body was then tilted to a precalibrated, 30° head-up position. One of the
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].

Use of the sitting position during and after injection of plain bupivacaine (slightly hypobaric) produces a higher final level (by up to four segments) of block than use of the lateral horizontal position [13]. However, extending the duration of sitting from 2.5 to 7.5 min has not been found to influence the spread of the block [14], but a slow continuous increase was noticed for 30 min in all patients. This agrees with our present finding of changes up to 30 min, indicating late redistribution of the drug within the CSF. A slowly progressing extension of the block (promoted mainly by diffusion) has been observed in several studies [6–11] in which the time to the maximal level of analgesia was 60 min, or even later, with patients in a horizontal position. The continuous increase in the spread for the entire 30-min period in two of our patients (fig. 1) was similar to the diffusional type of distribution seen usually in the early phase of the block. In contrast, in the four other patients with extensions of block, the spread was probably caused by temporary cephalad displacement of CSF which contained a concentration of bupivacaine just sufficient to cause a block.

Only two of the patients had a decrease in heart rate and arterial pressure at the beginning of the 30-min sit-up test. The level and intensity of sympathetic block in relation to the level of sensory block may vary. Preganglionic sympathetic denervation occurs earlier and with smaller concentrations of

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**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 chemo- 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 horizontal position for patients during recovery and avoidance of unnecessary movements, when plain bupivacaine has been used for spinal anaesthesia.

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