Incidence of bradycardia during recovery from spinal anaesthesia: influence of patient position

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Summary

We administered 0.5% plain bupivacaine 4 ml intrathecally (L2–3 or L3–4) in three groups of 20 patients, according to the position in which they were nursed in the post-anaesthesia care unit (PACU): supine horizontal, 30° Trendelenburg or hammock position (trunk and legs 30° elevated). Patients were observed until anaesthesia descended to less than S1. The incidence of severe bradycardia (heart rate <50 beat min⁻¹) in the PACU was significantly higher in patients in the Trendelenburg position (60%) than in the horizontal (20%, P<0.01) or hammock (10%, P<0.005) position. After 90 min, following admission to the PACU, only patients in the hammock position did not have severe bradycardia. In this late phase, the incidence of severe bradycardia in the Trendelenburg group was 35% (P<0.005) and 10% in patients in the supine horizontal position. In four patients, severe bradycardia first occurred later than 90 min after admission to the PACU. The latest occurrence of severe bradycardia was recorded 320 min after admission to the PACU. We conclude that for recovery from spinal anaesthesia, the Trendelenburg position should not be used and the hammock position is preferred. (Br. J. Anaesth. 1998; 81: 723–726).

Keywords: anaesthetic techniques, subarachnoid; position, effects; heart, heart rate; complications, bradycardia

Moderate bradycardia occurs regularly during spinal anaesthesia.¹ The incidence of intraoperative severe bradycardia requiring therapy has been reported²³ and cardiac arrest has occurred.⁴ However, there are no published data on the incidence of severe bradycardia during recovery from spinal anaesthesia.

Decreased venous return to the heart secondary to sympathetic block leads to a decrease in right atrial pressure and pressure in the great veins as they enter the right atrium; this is assumed to contribute to bradycardia during spinal anaesthesia.⁶ The Trendelenburg position is associated with a higher venous return⁷ which might result in a decreased incidence of severe bradycardia. The incidence and time of occurrence of severe postoperative bradycardia during recovery from spinal anaesthesia, and the influence of differences in venous return in the Trendelenburg, horizontal and hammock positions were examined in this study.

Patients and methods

We studied 60 patients, ASA I or II, undergoing arthroscopy of the knee under spinal anaesthesia after obtaining approval from the Ethics Committee of the Faculty of Medicine, University of Graz, Austria and written informed consent from all patients. Patients with characteristics known to influence the incidence of intraoperative severe bradycardia from spinal anaesthesia were excluded (i.e. heart rate (HR) <60 beat min⁻¹, age >50 yr and <20 yr, ASA >II, cardiovascular disease or medication with β-adrenergic blocking drugs).³⁴ Pregnancy was also an exclusion criterion.

All patients received oral premedication with midazolam 7.5 mg, 1 h before induction of spinal anaesthesia. Continuous monitoring in the operating theatre and in the post-anaesthesia care unit (PACU) consisted of ECG, HR and pulse oximetry, in addition to non-invasive systolic and diastolic arterial pressure measurements (every 5 min). Data were recorded automatically onto a computer using Hewlett Packard DocVue. In the operating theatre, patients received i.v. infusion of 6% hydroxyethyl-starch 500 ml before induction of spinal anaesthesia. This was followed by slow infusion of Ringer’s solution 500 ml. Plain bupivacaine 0.5% (4 ml) was injected into the subarachnoid space at L2–3 or L3–4 with the patient in the lateral horizontal position. After injection of local anaesthetic, the patient was immediately positioned horizontally and supine for surgery.

Severe bradycardia was defined as HR <50 beat min⁻¹ requiring treatment with atropine 0.5 mg i.v., repeated as necessary. Severe hypotension was defined as a decrease in systolic arterial pressure (SAP) to less than 70% of the patient’s initial SAP before induction of spinal anaesthesia, requiring treatment with ephedrine 25 mg i.v. Moderate bradycardia and moderate hypotension were defined as statistically significant decreases in these variables not requiring treatment.

After surgery, patients were transferred to the PACU and allocated randomly to one of three groups of 20 patients each according to the position in which they were to be nursed in the PACU: horizontal supine, hammock (legs and trunk 30° elevated) or 30° Trendelenburg. Randomization was accomplished by predetermined numbers; in the operating
theatre both the patient and examiner were blinded to group allocation; in the PACU, this was not possible because the position of the patient was obvious. Monitoring and therapy continued as before. The dermatomal level of anaesthesia (no sensation to pin-prick with the sterile tip of a safety pin after a strong push onto the skin) was recorded at the time of admission to the PACU and at 15-min intervals. Patients remained in the PACU until anaesthesia descended to below S1. Duration of spinal anaesthesia was defined as the time between intrathecal injection of bupivacaine and the moment when the anaesthetic level had descended to below S1.

Data are expressed as number, percentage, mean or median (SD, confidence limits or percentile). Comparisons of several means and continuous data were made using two-way ANOVA, and chi-square analysis with the Bonferroni–Dunn procedure. Exact binominal confidence limits were used for comparison of incidents. P < 0.05 was considered statistically significant. The statistical computer package used was NCSS 97, except for the prospective power analysis, where PASS 6.0 was used; both were run on Win NT 4.0 Workstation using a Pentium PC.

**Results**

There was no significant difference in mean age, weight, height, sex ratio or ASA status between the three groups (table 1). All patients had satisfactory block for surgery and did not require supplementary medication. Median level of anaesthesia at the time of admission to the PACU was L3 (25th – 75th percentile: L2–L4) in all groups. There was no significant difference in duration of spinal block or duration of stay in the PACU (table 1). During the observation period, no patient had an oxygen saturation less than 95%; oxygen was not administered to any patient. No patient fell asleep or had respiratory obstruction.

### Table 1: Comparison of patient characteristics (mean (SD or range) or number).

<table>
<thead>
<tr>
<th></th>
<th>Hammock position (n = 20)</th>
<th>Horizontal position (n = 20)</th>
<th>Trendelenburg position (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex (M/F)</strong></td>
<td>8/12</td>
<td>7/13</td>
<td>11/9</td>
</tr>
<tr>
<td><strong>Age (yr)</strong></td>
<td>40 (21–47)</td>
<td>37 (26–50)</td>
<td>36 (20–50)</td>
</tr>
<tr>
<td><strong>Height (cm)</strong></td>
<td>171 (11)</td>
<td>172 (10)</td>
<td>169 (10)</td>
</tr>
<tr>
<td><strong>Weight (kg)</strong></td>
<td>73 (11)</td>
<td>76 (11)</td>
<td>71 (12)</td>
</tr>
<tr>
<td><strong>ASA</strong></td>
<td>1.2 (0.3)</td>
<td>1.2 (0.4)</td>
<td>1.3 (0.2)</td>
</tr>
<tr>
<td><strong>Initial heart rate (beat min</strong>⁻¹)</td>
<td>73.8 (8.1)</td>
<td>74.3 (9.1)</td>
<td>73.7 (9.3)</td>
</tr>
<tr>
<td><strong>Initial SAP (mm Hg)</strong></td>
<td>130.9 (16.1)</td>
<td>133.0 (14.5)</td>
<td>131.8 (21.5)</td>
</tr>
<tr>
<td><strong>Initial DAP (mm Hg)</strong></td>
<td>69.0 (11.3)</td>
<td>72.5 (10.7)</td>
<td>67.8 (14.5)</td>
</tr>
<tr>
<td><strong>Duration of spinal block (min)</strong></td>
<td>320 (86)</td>
<td>308 (52)</td>
<td>325 (56)</td>
</tr>
<tr>
<td><strong>Duration of stay in PACU (min)</strong></td>
<td>212 (56)</td>
<td>196 (53)</td>
<td>225 (19)</td>
</tr>
<tr>
<td><strong>No. of patients with ISB</strong></td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td><strong>No. of patients with ISH</strong></td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

In the operating theatre, all patients were in the horizontal supine position, where two patients in each group had an episode of severe bradycardia (table 1). In the PACU, patients in all groups had moderate bradycardia, and this continued until the end of the observation period. Between groups, however, there was no difference in the mean decrease in HR. In the PACU, there was significant variation in the incidence of severe bradycardia in terms of incidence during the total stay in the PACU and incidence later than 90 min after admission to the PACU (table 2).

The time of occurrence of severe bradycardia in the PACU and anaesthetic level at which it occurred are documented in figure 1. After successful treatment of severe bradycardia during the first 90 min after admission to the PACU, some patients continued to have episodes of severe bradycardia, with the latest occurring after 320 min. In one patient in the horizontal group and in three patients in the Trendelenburg group, an episode of severe bradycardia occurred later than 90 min after admission to the PACU where none had occurred previously; one patient had his first episode of severe bradycardia 255 min after admission to the PACU.

Both patients in the Trendelenburg group who had severe bradycardia in the operating theatre also had severe bradycardia in the PACU; none of the patients with intraoperative severe bradycardia in the other groups (two patients in each group) had severe bradycardia in the PACU. All patients with intraoperative severe bradycardia were treated successfully with atropine 0.5 mg. One patient in the hammock group and one patient in the horizontal group had intraoperative severe hypotension, treated successfully with ephedrine 0.25 mg (table 1). On admission to the PACU, the HR of all patients treated in the intraoperative period with atropine or ephedrine was below the initial value before induction of spinal anaesthesia. Neither of the two patients with intraoperative severe hypotension had severe hypotension or severe bradycardia after operation. The number of patients with intraoperative severe bradycardia or intraoperative severe hypotension, however, was insufficient for statistical analysis on the influence of intraoperative use of atropine or ephedrine on the incidence of severe bradycardia in the PACU. All patients with intraoperative severe bradycardia or intraoperative severe hypotension were included in the PACU analysis.

In the PACU, mean SAP was lower than the initial
value before induction of spinal anaesthesia and remained so until the end of the observation period. However, severe hypotension requiring therapy did not occur in the PACU. Between groups there was no difference in mean changes in SAP. There was no difference between mean diastolic arterial pressure in the PACU and pre-induction values in any group.

**Discussion**

In the operating theatre, where all patients were in the supine horizontal position, the incidence of severe bradycardia did not differ between groups, and was similar to previous studies."""We can assume that the risk factors for severe bradycardia during spinal anaesthesia were evenly distributed between groups.

For comparison with previous studies, we defined severe bradycardia as HR <50 beat min⁻¹. This value was thought to be an appropriate endpoint for intervention in this carefully defined group of patients: other patients may require earlier intervention or may safely tolerate this HR. Similar numbers of patients in the three groups were treated with atropine and ephedrine in the operative period and it is unlikely that these treatments influenced the differences in the incidence of bradycardia between groups.

In the PACU, patients in the 30° Trendelenburg position had a higher incidence of severe bradycardia than patients in the horizontal or hammock position. This observation seems to contradict the proposed mechanisms for bradycardia during spinal anaesthesia: according to Greene,"""9 diminished venous return secondary to sympathetic block activates great vein and right atrial cardiac receptors that both, by reflex mechanisms and directly, slow the HR. Although patients in the Trendelenburg position have greater venous return,"""7 they had a higher incidence of severe bradycardia. The level of moderate bradycardia did not differ between groups in our study. Therefore, a decrease in venous return most probably was not the primary cause for moderate or severe bradycardia during spinal anaesthesia in our patients.

In patients with higher levels of sympathetic block, including levels T1–4, cardiac accelerator denervation also slows HR."""9 In our patients, moderate bradycardia in the PACU continued until the end of the observation period with an anaesthesia level below S1 more than 5 h after subarachnoid injection of bupivacaine. Cardiac accelerator block most probably did not play a role in the mechanism of bradycardia in the PACU. Assuming a differential block of six segments between sensory and sympathetic block,"""10 only one patient had a sensory level high enough to block one cardiac accelerator segment at the time of admission to the PACU. In the later phase of recovery it can be assumed that no patient had cardiac accelerator block at a time when all patients had moderate bradycardia, and when some had episodes of severe bradycardia.

Recent studies using HR variability demonstrated modulation of sympathovagal balance during spinal anaesthesia. During the onset of spinal anaesthesia, disruption of sympathetic neural pathways leads to a decrease in sympathetic activity and a reflex reduction in parasympathetic activity. Over time, reflex parasympathetic inhibition is withdrawn. Although in volunteers without spinal block, the autonomic system attempts to maintain sympathovagal balance,30 30 min after spinal injection, decreased sympathetic activity and increased parasympathetic activity in the cardiac autonomic nervous system were observed. As acetylcholine released at the vagal nerve endings diminishes the release of noradrenaline from neighbouring sympathetic terminals, decreased cardiac sympathetic activity could be the result of both direct and indirect effects.

Studies of the influence of the Trendelenburg position in volunteers without spinal anaesthesia have not shown an effect on HR."""7 Furthermore, the Trendelenburg position in volunteers without spinal anaesthesia is not associated with significant alteration of dominant parasympathetic cardiac control in comparison with the resting supine position."""13 However, increased venous return in a patient under spinal anaesthesia with coexisting decreased sympathetic and increased parasympathetic activity could have a different effect compared with volunteers with normal sympathovagal balance and a tendency to maintain this balance."""15 Time from injection until the anaesthetic level descended to below S1 was more than 5 h. As moderate bradycardia continued until this time, and some patients also had episodes of severe bradycardia after 5 h, we can assume that sympathovagal imbalance was still present at the end of the observation period. Because a smaller dose of bupivacaine resulted in a lower level of spinal block, it might be speculated that such a smaller dose results in a lesser degree and shorter duration of sympathovagal imbalance, together with a smaller incidence of severe bradycardia. However, in a prospective study of risk factors for severe bradycardia during spinal anaesthesia, peak block height had a very weak correlation with occurrence of severe bradycardia and no correlation with the magnitude of bradycardia. Sympathovagal imbalance apparently only seems to have a weak cor-
In summary, patients in the Trendelenburg position had a high incidence of severe bradycardia. We suggest that this position should not be used during recovery from spinal anaesthesia. Severe bradycardia occurring later than 90 min after admission to the PACU was observed in patients who had bradycardia treated successfully in the first 90 min and in patients who had not had severe bradycardia before. This observation is particularly important, as in normal clinical practice some of these patients would have been discharged from the PACU before the occurrence of the first or a subsequent episode of severe bradycardia. Only patients in the hammock position did not have severe bradycardia in this late phase of recovery from spinal anaesthesia. Although the difference in the incidence of severe bradycardia between the hammock and horizontal groups was not significant, in comparison with the Trendelenburg group, only patients in the hammock group had a significantly lower incidence of severe bradycardia occurring later than 90 min after admission to the PACU. Therefore, the hammock position can be considered to be the optimal position for recovery from spinal anaesthesia, especially after discharge from the PACU.

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References