EFFECT OF FLUID PRELOADING ON CARDIOVASCULAR VARIABLES AFTER SPINAL ANAESTHESIA WITH GLUCOSE-FREE 0.75% BUPIVACAINE

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Subarachnoid anaesthesia causes hypotension from sympathetic nervous block which may be detrimental if excessive. At least one death has been reported following profound hypotension associated with spinal anaesthesia [1]. Despite the common practice of administering i.v. fluid before spinal anaesthesia, the efficacy of this manoeuvre in maintaining arterial pressure has not been evaluated in non-parturients undergoing elective surgery. The present study was undertaken to compare the effect of fluid preload with that of no preload on arterial pressure in ASA I patients. Glucose free 0.75% bupivacaine was used to evaluate its characteristics and value for lower abdominal or lower limb surgery. Data for 0.5% bupivacaine has been documented previously [1-5].

MATERIALS AND METHODS

We studied 40 ASA group 1 patients younger than 70 yr and scheduled for elective lower abdominal or lower limb surgery involving minimal blood loss under spinal anaesthesia. No patient was receiving concurrent medication. The study was approved by the hospital Ethics Committee and informed consent was obtained from patients who were allocated randomly to two groups (A and B). Group A (no preload) received Hartmann’s solution 1 ml min⁻¹ to keep an i.v. cannula patent, whilst group B (preload) received Hartmann’s solution 1000 ml in the 15 min immediately before spinal puncture. After overnight fasting, diazepam 10 mg was administered orally 2 h before operation. In the anaesthetic room, the resting arterial pressure was recorded using a Dinamap 845 automatic pressure recorder with printer [6]. A 14-gauge i.v. cannula was inserted into a forearm vein under local anaesthesia. Lumbar puncture was performed using a midline approach with a 25-gauge spinal needle at the L3–4 interspace with the patient in the sitting position. When a free flow of cerebrospinal fluid (CSF) had

SUMMARY

We studied the effect on systemic arterial pressure of fluid preloading with 1 litre of crystalloid fluid before spinal anaesthesia in 40 patients undergoing minor lower abdominal or lower limb surgery. Fluid was given at a rate of either 1 ml min⁻¹ (no preload group), or 1000 ml in the 15 min (preload group) immediately before induction of spinal anaesthesia with 3 ml of 0.75% glucose-free bupivacaine. There was no difference between the groups in the character of anaesthesia or motor block in the lower limbs. The cephalad spread of analgesia ranged from L1 to C8. Analgesia was insufficient for surgery without supplementary analgesia in three patients in each group. The group not given a fluid preload had significantly lower arterial pressures (P < 0.05) when anaesthesia extended above the T5 dermatome. The mean time before the lowest arterial pressure was recorded was twice as long in the preloaded group as in the non-preloaded group. Glucose-free 0.75% bupivacaine did not give a reliable extent of anaesthesia for lower abdominal surgery.
been obtained, 0.75% glucose-free bupivacaine 3 ml was injected at a rate of 1 ml every 5 s without barbotage. CSF 0.1 ml was re-aspirated to confirm that the tip of the needle was still in the subarachnoid space. After 2 min the patient was returned to the horizontal supine position. Systolic, mean and diastolic arterial pressures and heart rate were recorded at 1-min intervals for 30 min and then every 15 min. Bradycardia or hypotension to 80 mm Hg systolic, or symptoms of nausea, sweating or faintness were treated with atropine 0.5 mg i.v. or ephedrine 15 mg i.v. according to clinical judgement. The segmental spread of analgesia was assessed by loss of touch sensation to the blunt end of a Sherwood 25-gauge dental needle at 2, 5, 10, 15, 20 and 30 min, and then every 15 min until complete return of sensation. Motor block was assessed simultaneously by recording movement of the lower limbs according to a modified Bromage scale [7]: 0 = no paralysis; 1 = inability to raise the outstretched leg; 2 = inability to flex the knees; 3 = total paralysis of the lower limb.

The patients were assessed on the morning after surgery to evaluate any late complications of the spinal block.

Statistical analysis of the spread of block was undertaken using the Fischer Exact Test and Wilcoxon's Rank Summation Test. The correlation between the spread of block and the decrease in arterial pressure was analysed with linear regression analysis. Cardiovascular variables were analysed using Student's t test for unpaired data. P values < 0.05 were regarded as significant.

**RESULTS**

There were no significant differences between the physical characteristics of each group.

**Onset, duration and spread of analgesia**

There was a loss of sensation detectable within 2 min in all patients. The mean level of sensory loss reached the T11 dermatome after 5 min in both groups (fig. 1), and was maximal after 40 min in the unpreloaded and 30 min in the preloaded group (fig. 2). These results were not significantly different. The final spread of analgesia was wide, ranging from L1 to C8 (table I). The offset was similar in the two groups, with a mean time to complete loss of anaesthesia of 285 min. Analgesia was insufficient in three patients in each group (all...
undergoing inguinal hernia repair). Supplementation with papaveretum, local infiltration or, in one subject, recourse to general anaesthesia, was necessary in these patients.

**Motor block**

The onset of motor block in the lower limbs commenced between 4 and 7 min after subarachnoid injection and was complete in all cases after approximately 15 min. The interval before straight leg raising was possible was approximately 270 min; there was no significant difference between the groups.

**Cardiovascular changes**

The mean control systolic and diastolic arterial pressures were similar in both groups. In all patients, arterial pressure decreased and in all but two (one in each group) the heart rate decreased also. The mean values of the lowest recorded systolic and diastolic pressures were significantly lower in the unpreloaded group at both 0–30 min and 30–180 min (table II). The correlation between the lowest recorded pressure and the extent of anaesthetic block ranged from −0.3 to −0.63.

In order to examine this further, each patient group was subdivided into patients in whom the final spread of block was at T5 or above (high blocks), and those in whom the block was below this level (low blocks).

At 0–30 min, the unpreloaded patients with high blocks had minimum systolic and diastolic pressures that were significantly lower than those in the preloaded patients, whereas in patients with low blocks, only the minimum diastolic pressure was significantly lower, again in the unpreloaded group (table II). At 30–180 min the minimum systolic and diastolic pressures were also lower in unpreloaded than in preloaded patients with high blocks. There was no difference in the minimum systolic or diastolic pressures of preloaded and unpreloaded patients with low blocks (table II).

When the arterial pressures were analysed at incremental times from 0 to 180 min, mean systolic and diastolic pressures were significantly lower at 15 min in unpreloaded patients with high blocks than in corresponding preloaded patients (fig. 3A). Differences after this time were probably masked by patients in the unpreloaded group treated with ephedrine or atropine. In patients with low blocks, mean diastolic pressures were significantly lower in unpreloaded patients than in preloaded patients at 10–30 min, and the mean systolic pressure was significantly lower at 20 min (fig. 3B).

The lowest arterial pressure was recorded later than 30 min in only four unpreloaded patients (17%), whilst in the preloaded group arterial

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**Table I. Upper limit of the spread of the subarachnoid block. Number of patients in each group reaching grouped segmental levels**

<table>
<thead>
<tr>
<th>Segmental level</th>
<th>Preloaded (n = 18)</th>
<th>Unpreloaded (n = 22)</th>
<th>Total (n = 40)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Higher than T3</td>
<td>6</td>
<td>9</td>
<td>15</td>
</tr>
<tr>
<td>T6–T4</td>
<td>4</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td>T9–T7</td>
<td>3</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>T12–T10</td>
<td>4</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>Lower than T12</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>

**Table II. Mean (SEM) lowest values of arterial pressure recorded**

<table>
<thead>
<tr>
<th>Time from insertion of block (min)</th>
<th>0–30</th>
<th>30–180</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic (mm Hg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unpreloaded group</td>
<td>99.9 (3.7)</td>
<td>100.7 (3.9)</td>
</tr>
<tr>
<td>Preloaded group</td>
<td>109.6 (2.9)</td>
<td>115.3 (3.3)</td>
</tr>
<tr>
<td>P</td>
<td>&lt; 0.025</td>
<td>&lt; 0.005</td>
</tr>
<tr>
<td>Unpreloaded group (high blocks)</td>
<td>94.5 (5.0)</td>
<td>95.0 (5.5)</td>
</tr>
<tr>
<td>Preloaded group (high blocks)</td>
<td>110.1 (4.5)</td>
<td>113.6 (3.0)</td>
</tr>
<tr>
<td>P</td>
<td>&lt; 0.05</td>
<td>&lt; 0.025</td>
</tr>
<tr>
<td>Unpreloaded group (low blocks)</td>
<td>110.7 (2.7)</td>
<td>109.3 (2.3)</td>
</tr>
<tr>
<td>Preloaded group (low blocks)</td>
<td>117.0 (3.9)</td>
<td>109.1 (3.6)</td>
</tr>
<tr>
<td>P</td>
<td>ns</td>
<td>ns</td>
</tr>
</tbody>
</table>
FLUID PRELOADING AND SPINAL ANAESTHESIA

FIG. 3. Variation in arterial pressure in patients with blocks at the level of T5 or above (A), and in those with blocks below the level of T5 (B). Preloaded group: ■ = systolic; ◆ = diastolic. Unpreloaded group: □ = systolic; ◇ = diastolic. *P < 0.05.

FIG. 4. Number of patients in whom minimum arterial pressure was recorded at various times from insertion of the block.

pressure continued to decrease after 30 min in 12 patients (66%) (fig. 4). The median time for a maximal decrease in arterial pressure was 20 min (range 10–150 min) in the unpreloaded group and 60 min (range 10–180 min) in the preloaded group (P < 0.05). Thirteen of 40 patients (32.5%) needed atropine or ephedrine. Of these, 10 were in the unpreloaded group, of whom nine had high blocks; the other three were in the preloaded group, of whom two had high blocks. Thus 84.6% of patients requiring treatment had high blocks.

Adverse reactions

In one patient in the unpreloaded group, sudden bradycardia associated with a systolic pressure of 46 mm Hg was observed after 1 h during a period of traction on the spermatic cord. The patient felt nauseated, sweaty and faint. Atropine 0.5 mg and ephedrine 15 mg were administered and complete recovery followed.

One patient developed a post spinal headache of moderate severity 4 h after the block; recovery followed without treatment.

DISCUSSION

This study has demonstrated variation in the spread of block following a standardized technique using glucose-free 0.75% bupivacaine. Previous work recorded a mean spread to T4 following 0.75% bupivacaine, two segments higher than in this study [5]. The range of spread in the present study was from L1 to C8. If T6 is the level of block considered optimal for lower abdominal surgery, 24 of our patients (60%) had excessive cephalad spread and 15% had inadequate analgesia. This unpredictable spread suggests that 0.75% plain bupivacaine is not reliable for lower abdominal surgery when administered as described, as has been observed previously [8].

The median time interval for maximal cephalad spread to occur in our series was approximately 35 min. This is longer than observed in previous studies in which 3 ml of 0.5% solution was used [3, 4, 9, 10]. Bengtsson, Malqvist and Edstrom also found a longer period for maximum cephalad spread using 3 ml of 0.75%, or 4.5 ml of 0.5% solution [11], suggesting that the increased dose may be an important factor.

Although it is common practice to administer fluids before extradural or spinal anaesthesia, our results suggest that many patients receive them
unnecessarily. A previous study found that, in 31% of parturients, the systolic arterial pressure decreased more than 30 mm Hg after extradural bupivacaine, despite preloading with 500 ml of dextran solution [12]. Crystalloid fluid and albumin solutions have also been shown to be useful in parturients [13, 14], in whom inferior vena caval compression may complicate the findings. Others have studied vasopressors, but their control patients received a fluid preload which did not prevent hypotension [15–17]. One study observed a decrease in arterial pressure of 24.7% and 15.2% with 0.75% bupivacaine and 0.5% amethocaine, respectively, despite 500–800 ml of crystalloid preload [18].

It has been suggested previously that the extent of hypotension may be related to the spread of block [12, 13, 19, 20]. In our study there was a tendency towards a more stable arterial pressure after preloading in patients in whom the block extended to T5 or above; below T5, early differences disappeared later because the mean time to recording of the lowest arterial pressure was twice as long in the preloaded group. This presumably resulted from redistribution of fluid from the intravascular compartment [14, 21, 22].

Eleven of the 13 patients needing treatment for hypotension had high blocks, two having received fluid preload. Only one patient in each group required treatment when the block was below T5. It is unlikely, therefore, that the mean lowest arterial pressures recorded in patients with low blocks were significantly changed by treatment for hypotension.

The three preloaded patients given fluid preloads and requiring vasoactive drugs were treated later in the study. Delayed hypotension has been noted before [23]. In our study vasopressors were given to patients in both groups more than 1 h after the block.

In conclusion, we suggest that a fluid preload may be of value in reducing the maximum decrease of arterial pressure, but only in patients with blocks extending above the T6 dermatome. Furthermore, we found that vasopressors were always effective in treating hypotension when 1 litre of crystalloid fluid was ineffective.

REFERENCES

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5. Tuominen M, Kalso E, Rosenberg PH. Effects of posture on the spread of spinal anaesthesia with isobaric 0.75% or 0.5% bupivacaine. British Journal of Anaesthesia 1982; 54: 313–318.
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