Spread of Spinal Anaesthesia with 0.5% Bupivacaine: Influence of the Vertebral Interspace and Speed of Injection

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Summary

Three millilitres of plain 0.5% bupivacaine were injected intrathecally at two different spinal interspaces (L2/3 and L4/5) and at two different speeds (15 and 30 sec) in four groups of ten patients. Injection at L2/3 over 15 sec produced a significantly higher mean maximum spread of analgesia (T6.4) when compared to injection at L4-5 over 15 sec (T10.3) (P<0.05). Over the same interspace L2/3, injection over 15 sec also produced a higher level of spread as compared to the 30 sec group (P<0.05). At 15 min there was a greater fall in blood pressure in the L2/3 15 sec group when compared to the other groups (P<0.01). There was a further decrease in the blood pressure in L2/3 15 sec and L4/5 30 sec groups after 30 minutes of blockade (P<0.01). Therefore close monitoring of cardiovascular parameters must be continued for at least 30 min in spinal anaesthesia with bupivacaine.

Key Words: Spinal anaesthetic technique, Local anaesthetic bupivacaine

Introduction

The use of plain bupivacaine for subarachnoid anaesthesia has been studied previously outside Malaysia. Factors thought to influence intrathecal spread such as total dose, volume, patient position and effect of baricity have been studied extensively. The importance of the site and speed of injection is still controversial. The purpose of our study was to determine the influence of spinal interspace and speed of injection on the spread of 3ml of 0.5% plain bupivacaine.

Material And Method

We studied 40 patients, all ASA 1-2, age between 18-30 years scheduled to undergo surgery of the lower extremities. The patients were allocated randomly to one of the four groups (Table I). Informed consent was obtained from the patients and the study was approved by the Hospital Ethical Committee.

Premedication consisted of Tab Diazepam 5-10 mg an hour before the procedure. All patients were preloaded with 500ml of Hartman’s solution, infused over 30 mins before subarachnoid injection.
SPREAD OF SPINAL ANAESTHESIA WITH 0.5% BUPIVACAINE

Table I
Spinal interspace and speed of injection in four groups of patients receiving plain bupivacaine

<table>
<thead>
<tr>
<th>Group</th>
<th>No.</th>
<th>Spinal Interspace</th>
<th>Speed of Injection (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>10</td>
<td>L2/3</td>
<td>15</td>
</tr>
<tr>
<td>Group B</td>
<td>10</td>
<td>L2/3</td>
<td>30</td>
</tr>
<tr>
<td>Group C</td>
<td>10</td>
<td>L4/5</td>
<td>15</td>
</tr>
<tr>
<td>Group D</td>
<td>10</td>
<td>L4/5</td>
<td>30</td>
</tr>
</tbody>
</table>

Table II
Patients' data - mean (SD)

<table>
<thead>
<tr>
<th></th>
<th>Group A L2/3 15 seconds</th>
<th>Group B L2/3 30 seconds</th>
<th>Group C L4/5 15 seconds</th>
<th>Group D L4/5 30 seconds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>29.3 (9.8)</td>
<td>36.2 (13.8)</td>
<td>29.6 (11.3)</td>
<td>36.0 (14.1)</td>
</tr>
<tr>
<td>Ht (cm)</td>
<td>169.8 (6.0)</td>
<td>163.1 (8.3)</td>
<td>169.1 (8.4)</td>
<td>166.3 (6.0)</td>
</tr>
<tr>
<td>Wt (kg)</td>
<td>56.2 (8.4)</td>
<td>56.2 (8.4)</td>
<td>62.1 (15.5)</td>
<td>61.5 (10.9)</td>
</tr>
</tbody>
</table>

Spinal anaesthesia was performed with the patient in the lateral horizontal position. The patient was turned supine horizontal immediately after the injection. A 25G spinal needle was used and 3ml of 0.5% plain bupivacaine was used as the local anaesthetic in all patients.

The spines of the vertebra were counted from both cranial and caudal directions, and palpation of the iliac crest was made to confirm the position of the fourth lumbar vertebra. All punctures were made in the midline and aspiration of spinal fluid 0.2ml was made at the beginning and at the end of the injection. Assessment of patients was done by another anaesthetist using a 23G needle to assess sensory level. Motor block was scored using the Bromage motor scale.

Assessment was made at five, 10, 30 and 60 minutes and then at 30 minute intervals until recovery of L1 spinal segment. Visible contraction of the rectus femoris muscle was taken as the time of recovery from motor block. Intravenous ephedrine (3mg increments) was given if the blood pressure fell by more than 25 per cent of the baseline reading.

Data was analysed using Student's t-test for comparison between groups and one way analysis of variance within groups. All sensory levels were converted to a score before employing the above analysis. P<0.05 was considered statistically significant.

Results
There was no difference between the four groups with respect to age, height and weight (Table II).
Fig. 1: Spread of sensory blockade from the intrathecal injection of 0.5% plain bupivacaine

Fig. 2: Mean onset time of analgesia after intrathecal injection of 0.5% plain bupivacaine 3ml at level L2/3 (■) and L4/L5 (●) over 15 sec {**p < 0.05}

Note: The mean onset time of analgesia was faster at 15, 30 and 60 minutes (P<0.05) at spinal interspace L2/3 if the speed of injection was over 15 seconds compared to spinal interspace L4/5.

At different spinal interspaces and speed of injection over 15 seconds, the mean maximum spread of analgesia was dermatome T6.4 in group A as compared to T10.3 in group C (p<0.01). However, there was no difference in mean maximum spread of analgesia between group B and group D when speed of injection was over 30 seconds (Fig. 1).

At the same spinal interspace L2/3 but with different speeds of injection either over 15 or 30 seconds, group A (15 seconds) had a greater cephalad spread when compared to group B (30
Fig. 3: Mean onset time of analgesia after intrathecal injection of 0.5% plain bupivacaine 3ml at level L2/L3 (■) vs L4/L5 (♦) over 30 sec

Note: There was no difference in mean onset time between L2/L3 and L4/L5 if the speed of injection was over 30 seconds

Fig. 4: Mean onset time of analgesia after intrathecal injection of 0.5% plain bupivacaine 3ml at level L2/L3 over 15 sec (■) and L2/L3 over 30 sec (♦) (** P< 0.02)

Note: The mean onset time was faster at 15 and 30 minutes in spinal interspace L2/3 if the speed was 15 seconds compared to 30 seconds
seconds) which was statistically significant (p<0.05). There was no difference in cephalad spread at lower spinal interspace L4/5 regardless of speed of injection.

At higher spinal interspace L2/3 and with a faster speed of injection over 15 seconds (group A) there was a wider range of spread as compared to the other three groups (Fig 6).

The mean onset of analgesia was faster at spinal interspace L2/3 at 15, 30 and 60 minutes compared to spinal interspace L4/5 if the speed of injection was over 15 seconds (Fig 2). There was no difference if the injection was over 30 seconds (Fig 3).

The mean onset of analgesia was also significantly faster if given at a faster speed (over 15 seconds) and at higher spinal interspace L2/3 (Fig 4).

Injection at a lower spinal interspace L4/5 deferred the time of onset of analgesia even with faster speed of injection (Fig 5).

In group A, B and D, 100 per cent motor blockade was achieved in all patients by 60 minutes. In group C only 80 per cent of patients achieved this degree of motor blockade. However this difference was not statistically significant.

There was a greater fall in blood pressure in group A patients compared to the other three groups at 15 and 30 minutes (Table III). Four patients in group A received ephedrine with doses ranging from 3-12mg because of fall in systolic blood pressure by more than 25 per cent of baseline level. The decreases in blood pressure was still significant at 30 minutes of blockade in group A (p<0.01) and group D (p<0.05). There was no significant change in heart rate (Table III).

![LEVEL T6 T8 T10 T12 L2 L4 S1 0 10 20 30 40 50 60 TIME AFTER INJECTION (min)](image)

**Fig. 5:** Mean onset time of analgesia after intrathecal injection of 0.5% plain bupivacaine 3ml at level L4/L5 over 15 sec (■) vs 30 sec (★)

*Note: The mean onset time was not different eventhough the speed of injection was at 15 seconds compared to 30 seconds at spinal interspace L4/5.*
Fig. 6: Maximum spread of analgesia in 40 patients after the injection of 0.5% bupivacaine 3ml

Table III
Heart rate and blood pressure changes before and after spinal anaesthesia. Mean (SD)

<table>
<thead>
<tr>
<th></th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
<th>Group D</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>L2/3 15</td>
<td>L2/3 30</td>
<td>L4/5 15</td>
<td>L4/5 30</td>
</tr>
<tr>
<td>Heart rate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(beat/min)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before</td>
<td>79.5 (19.8)</td>
<td>77.8 (13.0)</td>
<td>78.8 (15.4)</td>
<td>86.3 (20.9)</td>
</tr>
<tr>
<td>At 15 min</td>
<td>79.4 (25.2)</td>
<td>73.6 (11.1)</td>
<td>81.7 (17.3)</td>
<td>77.5 (14.3)</td>
</tr>
<tr>
<td>At 30 min</td>
<td>69.6 (15.8)</td>
<td>71.5 (12.30)</td>
<td>77.3 (14.6)</td>
<td>68.2 (17.0)</td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before</td>
<td>123.8 (15)</td>
<td>124.4 (13)</td>
<td>115.0 (13)</td>
<td>129.2 (18)</td>
</tr>
<tr>
<td>At 15 min</td>
<td>102.3 (10) *</td>
<td>116.0 (15)</td>
<td>114.4 (13)</td>
<td>121.5 (17)</td>
</tr>
<tr>
<td>At 30 min</td>
<td>103.3 (12) *</td>
<td>111.5 (15)</td>
<td>116.0 (18)</td>
<td>107.2 (22) **</td>
</tr>
</tbody>
</table>

(P value: for significance between groups)
* P < 0.01
** P < 0.05
Discussion

There are few techniques which produce so widespread an effect in response to the administration of so little drug as the subarachnoid administration of local anaesthetic agents. The importance of spinal interspace on spread of analgesia has been studied by M. Tuominen et al recently. They compared the spread of analgesia at different interspace and found that at spinal interspace (L2/3), a much higher analgesic level could be achieved than at spinal interspace L4/5. However, Osler et al found that the results were otherwise. They showed that spinal interspace did not influence the spread of analgesia. Our results showed that injection at lower spinal interspace level will result in lower and more restricted spread of analgesia.

At higher spinal interspace L2/3, the range of spread was higher and wider.

The faster the injection speed, combined with higher spinal interspace level will invariably result in higher analgesia level and hence a greater fall in blood pressure.

The mechanism of this effect may be due to deposition of local anaesthetic solution in the lumbar cerebrospinal fluid (CSF) collection if the injection site is low. This collection of CSF may buffer the initial spread of local anaesthetic. If the subarachnoid injection is given at higher spinal interspace and at faster speed, there will be a greater spread along the more limited space between dura and spinal cord or caudal equina. Foelschow et al studied the effect of removing CSF before spinal injection and found that spread was more extensive and variable, implying that the buffering capacity of the lumbar CSF had been impaired.

The injection at L2/3 spinal interspace at 15 seconds resulted in greater fall in blood pressure as compared to the other groups. There was a further fall in the blood pressure after 30 minutes of blockade. Therefore close monitoring must be continued for at least 30 minutes in bupivacaine spinal anaesthesia.

References


