Rate of injection through Whitacre needles affects distribution of spinal anaesthesia

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A prospective, randomized, double-blind study was performed to investigate whether altering the rate of injection of local anaesthetic through a Whitacre needle had any effect on the spinal block achieved. Twenty patients scheduled for elective urological surgery under spinal anaesthesia received an injection of 3 ml of 0.5% plain bupivacaine either by hand (fast) over 10 s (18 ml min⁻¹) or by infusion pump (slow) over 3 min (1 ml min⁻¹). All patients were in the sitting position both during insertion of the spinal needle and for 3 min after the start of spinal injection, and they then changed to the supine position. The slow injection group achieved peak sensory block earlier, after a median interval of 20 (95% confidence interval 12.5–30) min vs 30 (22.5–45) min (P<0.05) for the fast group. The level of peak sensory block was similar: T3.5 (T2–T4.5) vs T4 (T1.5–T6.5). The time to lowest mean arterial pressure occurred earlier in the slow group, at 10 (8 to 18) vs 20 (15–31) min (P<0.05). Duration of the motor block was shorter in the slow group: 180 (152±242) vs 270 (225±300). We conclude that a slow spinal injection of plain bupivacaine results in a block of more rapid onset and recovery.

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There are many factors that effect the distribution of local anaesthetics within the intrathecal space. Factors that the anaesthetist has some control over include the physical characteristics of the injectate and the technique of injection.

The recent reintroduction of side-port atraumatic spinal needles has changed the practice of spinal anaesthesia. These needles have a side opening, which can effect the distribution of local anaesthetic agents. Also, the flow patterns of fluids injected through these needles have different properties when compared with the conventional Quincke type. In vitro work has suggested that flow patterns become turbulent from Whitacre needles at injection rates that are much faster and more clinically relevant than those obtained with Quincke needles of the same gauge.

A prospective, randomized, double-blind study was performed to investigate whether altering the rate of injection of the local anaesthetic through a Whitacre needle had any effect on the characteristics of spinal block achieved.

Methods

Twenty patients scheduled for elective transurethral prostate or bladder surgery under spinal anaesthesia were recruited. Patients were aged 18–80 yr, ASA I–III and had no neurological deficits or contraindications to spinal anaesthesia. The study was approved by the hospital Ethics Committee and all patients gave informed consent.

Patients were premedicated 1–2 h before surgery with temazepam 0.3 mg kg⁻¹ to the nearest 5 mg. On arrival in the anaesthetic room, non-invasive blood pressure, pulse oximetry and ECG monitoring were applied and an i.v. infusion of Ringer lactate solution (10 ml kg⁻¹) was administered over 15–20 min. Patients were randomized (sealed envelope) by computer-generated random list (Kwikstat 3.01; TexaSoft, Cedar Hill, Texas, USA) to receive either the fast (18 ml min⁻¹) or slow (1 ml min⁻¹) injection rate of spinal anaesthesia.

Lumbar puncture was performed in the midline at the L2,3 interspace under sterile conditions with the patient in the sitting position using a Becton Dickinson Whitacre 25G spinal needle with the orifice pointing cephalad. Once cerebrospinal fluid (CSF) had been identified, 3 ml of 0.5% plain bupivacaine was injected either by hand over a 10 s period (fast) or via a constant infusion pump over 3 min (slow). Patients in the fast group remained seated for a total of 3 min before being gently positioned supine, so that both groups experienced the same duration of positional effects. Oxygen was administered via a face mask at 5 litre min⁻¹.

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and sedation with midazolam 1–2 mg increments was given as clinically appropriate. Ephedrine was given if required in 3–6 mg increments to maintain mean arterial blood pressure within 20% of baseline, and i.v. fluids were given to replace estimated surgical blood loss and aid restoration of blood pressure.

Blood pressure, height of sensory block using temperature sensation with ethyl chloride spray and motor block (modified Bromage 4-point scale) were recorded by a blinded investigator at frequent intervals (0, 5, 10, 15, 20, 30, 45, 60, 90, 120 min and hourly thereafter) until complete recovery. Blood pressure was measured every 2.5 min for the duration of the surgery. The dose of ephedrine and i.v. fluid administration was recorded.

Results were analysed by the Student’s $t$, $\chi^2$ and Mann–Whitney test as appropriate (Minitab 10.5 for Windows 95). A $P$ value of <0.05 was regarded as significant. As there were no previous data on which to base a power calculation, this was considered to be a pilot study and so an empirical number of 20 patients was chosen.

Table 1 Patient characteristics expressed as mean (sd) or ratio. No significant differences

<table>
<thead>
<tr>
<th></th>
<th>Fast (n=9)</th>
<th>Slow (n=11)</th>
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<tbody>
<tr>
<td>Age (yr)</td>
<td>74 (67–81)</td>
<td>74 (66–81)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>165 (12)</td>
<td>165 (8)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>71 (17)</td>
<td>72 (10)</td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>7/2</td>
<td>11/0</td>
</tr>
<tr>
<td>Surgical time (min)</td>
<td>34 (10)</td>
<td>37 (12)</td>
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Table 2 Median (95% CI) times in minutes for onset and recovery of sensory and motor blocks. *Significant at $P<0.05$ by Mann–Whitney test

<table>
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<tr>
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<th>Fast (n=9)</th>
<th>Slow (n=11)</th>
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<tbody>
<tr>
<td>Level of peak sensory block</td>
<td>T4 (T1.5–T6.5)</td>
<td>T3.5 (T2–T4.5)</td>
</tr>
<tr>
<td>Onset of peak sensory block</td>
<td>30 (22.5–45)</td>
<td>20 (12.5–30)*</td>
</tr>
<tr>
<td>Full recovery of sensory block</td>
<td>300 (240–330)</td>
<td>240 (240–270)*</td>
</tr>
<tr>
<td>Full recovery of motor block</td>
<td>270 (225–300)</td>
<td>180 (152–242)*</td>
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Fig 1 Onset of median (95% CI) sensory level (loss of temperature sensation) after administration of 0.5% bupivacaine 3 ml injected at the speed of 1 ml min$^{-1}$ (slow) or 18 ml min$^{-1}$ (fast). *Significant at $P<0.05$ by Mann–Whitney test.
epidural and was presumably delivered cephalad more quickly and demonstrated a higher spread when using the slower rate of injection. The local anaesthetic in its original direction before the momentum ran out. Conversely, a fast injection produced a turbulent pattern that whipped back from the surface, causing the distribution to be less directional and more mixed and diluted. A mathematical model produced matching results. In an *in vitro* model, a slow injection was shown to produce a greater spread of local anaesthetic than a faster turbulent flow. With a slow injection, the injectate tended to adhere to and travel further along any surface it contacted in its original direction before the momentum ran out. Similarly, in our study, a slow injection (1 ml min⁻¹) of 0.5% plain bupivacaine through a 25G Whitacre spinal needle with the patient sitting up and the orifice pointing cephalad produced initially a greater spread of local anaesthetic than a faster injection rate has been used in the clinical management of the cephalc direction. The local anaesthetic agent was presumably delivered cephalad more quickly and remained concentrated in the posterior compartment of the CSF, where the sensory nerve roots are. This resulted in a compression of turbulence, resulting in more mixing and dilution. Such a slow injection rate would be so tedious and difficult to perform that its clinical use would be unlikely. However, the flow pattern from a Whitacre needle of the same gauge becomes turbulent at an injection rate of 2 ml min⁻¹. This injection rate has been used in the clinical management of patients in a couple of studies. In a *in vitro* model, a slow injection was shown to produce a greater spread of local anaesthetic than a faster turbulent flow. With a slow injection, the injectate tended to adhere to and travel further along any surface it contacted in its original direction before the momentum ran out. Conversely, a fast injection produced a turbulent pattern that whipped back from the surface, causing the distribution to be less directional and more mixed and diluted. A mathematical model produced matching results. Similarly, in our study, a slow injection (1 ml min⁻¹) of 0.5% plain bupivacaine through a 25G Whitacre spinal needle with the patient sitting up and the orifice pointing cephalad produced initially a greater spread of local anaesthetic than a faster injection rate has been used in the clinical management of the cephalic direction. The local anaesthetic agent was presumably delivered cephalad more quickly and remained concentrated in the posterior compartment of the CSF, where the sensory nerve roots are. This resulted in a more rapid onset of peak sensory block. Plain 0.5% bupivacaine is slightly hypobaric at body temperature and so it will ‘float’ in the CSF. The fact that the peak sensory block levels were similar in the two groups implies that equal amounts of drug eventually reached the same cephalad level in the posterior CSF compartment. All the patients remained sitting upright for the same period of time and were then positioned supine, so there were no differing gravitational effects. The bupivicaine would have floated upwards along the cephalad border of the thoracic kyphosis (while the patient was supine) to a higher level until diluted enough with the CSF to become thoroughly mixed and no longer affected by gravity. The clinical height of the sensory block then depends on there being the minimal concentration of local anaesthetic required to block a sensory spinal root. The time for return of normal sensory function in the slow injection group (Table 2) is probably an artefact as the recovery profile of the sensory block was identical for both groups until 180 min. The statistical significance of the measurement at 240 min is almost certainly a chance effect. It is not possible to explain how a sensory block of similar height produced by the same agent could wear off more quickly in one group than in the other. The onset time of peak sympathetic block was inferred from the time to achieve the lowest mean arterial pressure. The anaesthetist involved (MGS) attempted to maintain the mean arterial pressure within 20% of the baseline level by the early use of fluids and ephedrine. The reduction of blood pressure was more rapid in the slow group. Patients in the slow group required more ephedrine; this probably reflects the fact that the patients had less time to compensate for the haemodynamic changes. The reason for the more rapid sympathetic block is unknown. The sympathetic efferents that maintain vasoconstriction accompany the anterior motor roots, and the onset of motor block was if anything slightly delayed in the slow group. However, sympathetic afferent fibres accompany the dorsal sensory roots, and blockade of these might affect the activity in the sympathetic efferent fibres. The motor block tended to develop more slowly (though not significantly so) and wore off significantly faster in the slow injection group. This aspect would be a useful clinical advantage in the shorter or daycase type procedure. This may be because, with more cephalic distribution, there was less drug reaching the lower lumbar and sacral motor nerve roots. There are many studies in the literature concerning slow injection rates through spinal catheters after the appearance of the cauda equina syndrome, but very few concerning unidirectional atraumatic needles. Holman and colleagues studied various injection rates through needles in a spinal cord model and recorded the distribution of hyperbaric dye using a digital video image technique. He found that the injection rate had a significant effect on peak dye concentrations, and this could be modelled empirically by an inverse exponential formula. A slow injection directed caudally (2 and 4 ml min⁻¹) produced much higher concentrations of dye in the sacral distribution, whereas a fast injection produced lower concentrations of dye because of turbulence, resulting in more mixing and dilution. Interestingly, no further dilution occurred once a rate of 6 ml min⁻¹ was exceeded, and this was similar whether the needle was a 24G Sprotte or a 27G Whitacre. Therefore, it is
not surprising that Neigh and colleagues did not detect a difference in patients when he used a 22G Whitacre needle with the orifice pointing cephalad. \(^1^8\) He injected hyperbaric tetracaine at 12 or 60 ml min\(^{-1}\); both rates are in the turbulent range and therefore produce maximum mixing. \(^5^\) \(^1^7\)

Two other clinical studies looking at injection rates through Whitacre needles did show a difference in block characteristics between slow (1.2 ml min\(^{-1}\)) and fast rates (30 ml min\(^{-1}\)). \(^1^1^\) \(^1^2^\) They both differed from our study because the patients were lying on their side and the needle orifice was pointing upwards, i.e., laterally to the spinal cord. Horlocker found that a slow injection of 0.3% bupivacaine (hypobaric) resulted in a peak sensory level that was four dermatomal segments lower. \(^1^1^\) Atchison and colleagues also found that a slow injection of hypobaric tetracaine produced a lower block level, which took longer to regress than the fast rate. \(^1^2^\) However, our in vitro observations support these findings, as with a slower injection there would be more flow in the direction of the orifice and consequently less lateral (cephalad and caudad) spread when the orifice is pointed perpendicular to the longitudinal axis of the spinal cord. \(^1^3^\)

Studies using hyperbaric solutions of local anaesthetic, even when using appropriately different injection rates, have not shown a difference in anaesthetic profile. \(^1^9^\) \(^2^0^\) This is probably because the baricity of conventional ‘hyperbaric’ solutions is so excessive that gravity effects override all other effects. \(^2^1^\)

References

6. Pitkanen M. Slow injection of 0.5% plain bupivacaine increases level. Reg Anesth 1992; 17 Suppl 26: 35: 28