Block of the sacral segments in lumbar epidural anaesthesia

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Background. Block of the first sacral segment is often delayed in lumbar epidural anaesthesia. The addition of either epinephrine or sodium bicarbonate to the local anaesthetic enhances the efficacy of epidural block. We assessed the block of lumbo-sacral segments in lumbar epidural anaesthesia adding epinephrine and/or bicarbonate to lidocaine.

Methods. Twenty-seven patients undergoing lumbar epidural anaesthesia with lidocaine 2%, 17 ml at L4-5 or L5-S1 were randomly divided into three groups. Plain lidocaine, lidocaine with 1:200 000 epinephrine or lidocaine–epinephrine–bicarbonate was administered via an epidural catheter. The pain threshold after repeated electrical stimulation was used to assess the sensory block at the L2, S1, and S3 segments. Motor block was evaluated using the Bromage scale.

Results. Patient characteristics were comparable between the groups. The pH of lidocaine in the lidocaine–epinephrine–bicarbonate group was significantly higher than that in other groups. Pain thresholds at the S1 and S3 segments in the lidocaine–epinephrine–bicarbonate group were significantly higher than those in the lidocaine–epinephrine group. However, differences in the pain threshold at the L2 segment between groups were insignificant. The time to onset of sensory block at the S1 and S3 in the lidocaine–epinephrine–bicarbonate group was significantly shorter than that in the lidocaine group. Pain threshold by pinprick test was approximately within the 30–50 mA range.

Conclusion. A combination of lidocaine, bicarbonate, and epinephrine increases the pain threshold over the sacral segments.


Keywords: anaesthetics local, lidocaine; anaesthetic techniques, epidural

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The cranial spread of lumbar epidural anaesthesia is somewhat more extensive than the caudal spread. Indeed, the block of the fifth lumbar and first sacral segments by anaesthetics is often delayed or incomplete. It has been reported that the delay in onset at L5 and S1 segments may be because of the large size of this nerve root. However, there is a large degree of inter-individual variability in root size. These reports suggest that the onset of the block of the caudal sacral segments may be later than that of the lumbar segments.

It has been reported that the mean time to achieve complete sacral anaesthesia with lidocaine hydrochloride 2% was 21 min following caudal epidural block. If a lumbar epidural successfully blocks the sacral segments within 21 min then there is no specific requirement to perform caudal epidural block. The addition of sodium bicarbonate to local anaesthetic enhances the effects of block whilst the addition of epinephrine (5 µg ml⁻¹, 1:200 000) to lidocaine hydrochloride 2% markedly affected the extradural caudal spread (L2-3). Therefore, the addition of sodium bicarbonate and epinephrine to lidocaine would be predicted to accelerate both the onset and caudal spread of lumbar epidural anaesthesia.

Electrical stimulation has been used to assess the degree of regional block as this method allows quantification of analgesia and induces temporal summation. Induction of temporal summation may render this test closer to clinical pain. Temporal summation occurs when the repetition of a peripheral stimulus causes increased and prolonged firing of dorsal horn neurones (central sensitization) thereby increasing pain perception. Central sensitization is likely to play an important role in the physiopathology of acute pain.
and chronic pain syndromes, including postoperative and neuropathic pain.\textsuperscript{13}

We compared the clinical efficacy [onset, sensory block of cephalad (L2 segment) and caudal region (S1 and S3 segments), motor block, and sympathetic block] within 20 min following lumbar epidural anaesthesia with either: (i) lidocaine alone, (ii) lidocaine–epinephrine, or (iii) lidocaine–epinephrine–sodium bicarbonate. We assessed analgesia using repeated electrical stimulation.

Methods

The sample size was based on estimations of the primary efficiency endpoint/pain threshold after repeated electrical stimulation. A difference of 15 mA in pain threshold was determined to be of clinical interest and a standard deviation of 12 mA has been reported.\textsuperscript{14} Nine patients per group were considered necessary to achieve statistical significance ($\alpha=0.05$, $1–\beta=0.8$).

After institutional review board approval and written, informed consent, patients (ASA physical status I–II, 16–65 yr of age) undergoing elective lower abdominal surgery, lower extremity surgery, vaginal surgery, or haemorrhoidectomy with epidural anaesthesia were enrolled in this prospective double-blind study. Randomization was performed by means of sealed envelopes.

The patients were divided into three groups: (i) lidocaine, (ii) lidocaine–epinephrine, and (iii) lidocaine–epinephrine–bicarbonate. The anaesthetic solutions were as follows: plain lidocaine (20 ml of xylcaine 2\% (Astra Zeneca, Osaka, Japan) plus 2 ml of saline 0.9\%); lidocaine–epinephrine (20 ml of xylcaine 2\% plus epinephrine (1:200 000) plus 2 ml of saline 0.9\%); lidocaine–epinephrine–bicarbonate (20 ml of xylcaine 2\%, plus epinephrine (1:200 000) plus 2 ml of sodium bicarbonate 8.4\%, being added immediately before administration). All solutions were given via an epidural catheter. The anaesthetic solutions were maintained at room temperature and the operating room temperature was maintained at 24–26°C.

Patients were premedicated with an i.m. injection of atropine 0.5 mg and midazolam 2 mg, 30 min before admission into the operating room. After insertion of an i.v. catheter, non-invasive arterial pressure, ECG, and arterial saturation were monitored. A pulse oximeter probe was applied to a big toe. Plethysmographic waveforms were recorded before and after epidural injection and every 5 min for 20 min (Datex AS/3\textsuperscript{TM}, Helsinki, Finland). Plethysmographic waveforms reflect the changes in blood pulsation or flow at the site of measurement.\textsuperscript{15} The amplitude of pulse oximeter waveforms on the toe significantly increases after lumbar epidural anaesthesia\textsuperscript{16} and the increase in the pulse amplitude from the toe may aid in the early detection of successful epidural block.\textsuperscript{16,17}

All epidural punctures were performed at the L4-5 or L5-S1 interspace. The epidural space was identified while patients were in the lateral position by using an 18 G Tuohy needle with its bevel facing cranially and a loss of resistance technique using saline. A 20 G catheter was inserted 3–5 cm into the epidural space. After negative aspiration, 3 ml of a solution containing lidocaine 2\% with epinephrine was administrated as a test dose. If after 2 min there was no evidence of intravascular or subarachnoid injection, an additional 14 ml was injected over a 1.5 min period. The time at which the study drug injection ended was termed ‘time 0’ for the purpose of subsequent patient assessment. The pH of lidocaine was determined by using a pMETER (Shimadzu, Kyoto, Japan).

In order to evaluate the pain threshold, we used four stimuli at 2 Hz (i.e. train-of-four) with TOF GUARD\textsuperscript{TM} (Biometer International, Odense, Denmark), which was originally used to evaluated the neuromuscular block. These stimuli evoke temporal summation.\textsuperscript{14,18} Bipolar surface Ag–AgCl electrodes were placed at the dermatomes L2 (ventral femoral region) as an index of cephalad spread, S1 (the lateral malleolus for stimulation of the sacral nerve) and S3 (gluteal region at the left paravertebral line) as an index of caudal spread.

Subjective pain thresholds were recorded as follows. A 200 µs, square wave impulse was used as a single stimulus. In order to determine the threshold for temporal summation, the single stimulus was repeated four times at 2 Hz and current intensity was increased from 5 mA in steps of 1–5 mA until summation (perception of the last stimulus being perceived as painful) was observed or a maximum current of 60 mA was reached in which case threshold was defined as 60 mA. The test was repeated at dermatomes L2, S1, and S3 at 5, 10, 15, and 20 min following administration of lumbar epidural anaesthesia.

Sensitivity to pinprick was tested at L2, S1, and S3 dermatomes 2 cm from the sites of electrical stimulation. The pinprick test was performed by pricking the skin twice with a 26 G needle at an interval of approximately 0.3 s. Cold sensitivity was tested with a gel bag that was kept in a freezer and applied to a 5 cm diameter area for 3 s. The pinprick test was repeated at the dermatomes L2, S1, and S3 for 5, 10, 15, and 20 min following administration of lumbar epidural anaesthesia. The onset of sensory block was defined as the time from ‘time 0’ to the abolition of the pinprick response. The level of anaesthesia was assessed in the anterior mid-sagittal line at 20 min by loss of cold sensation as described above.

Motor block was recorded using the Bromage scale (0=full flexion of feet and knees, 1=just able to move knees, 2=able to move feet only, and 3=unable to move feet or knees) with assessments performed at 5, 10, 15, and 20 min following administration of lumbar epidural anaesthesia. Each patient was hydrated with acetate Ringer’s solution 10 ml kg\textsuperscript{-1} during the first hour.

Analysis of patient characteristics was by the Kruskal–Wallis test. Wilcoxon’s signed-rank test was used for differences of haemodynamic data within the groups. The pain threshold response was compared using the
repeated measures, one-way analysis of variance (ANOVA). The amplitude of plethysmographic waveforms and Bromage scale values were compared using the Kruskal–Wallis test. A Bonferroni adjustment was made for multiple comparisons if significant results were obtained. Statistical analysis for the onset of sensory block was performed by utilization of survival analysis (Kaplan–Meier curves). The log rank test was used for comparisons among the groups. A P-value <0.05 was considered statistically significant. All data except the level of anaesthesia are presented as mean (SD). The level of anaesthesia is represented as median and range.

Results

Twenty-seven patients were enrolled in this study with no significant differences in height, anaesthesia level, weight, age, gender, and fluid administration between the groups (Table 1). The pH of lidocaine in the lidocaine–epinephrine–bicarbonate group was significantly higher than that in the lidocaine and lidocaine–epinephrine groups (P<0.01). There were no significant differences in mean arterial pressure and heart rate between the groups. However, heart rate significantly increased within the lidocaine–epinephrine and lidocaine–epinephrine–bicarbonate group after 20 min (Table 2). There was no difference in Bromage scale values and the amplitude of the plethysmographic waveforms between the groups. However, they significantly increased within each group after 20 min (Table 2).

The pain threshold at the S1 and S3 segments in the lidocaine–epinephrine–bicarbonate group was significantly higher than that in the lidocaine–epinephrine group whilst the pain threshold at the S3 segment in the lidocaine–epinephrine–bicarbonate group also was significantly higher than that in the lidocaine group (Fig. 1). The pain threshold at the S1 segment at 20 min was lower than that at the L2 and S3 segments in all groups (Fig. 1). In the lidocaine–epinephrine group, the pain threshold at the S1 segment at 20 min was significantly lower than that at the L2 and S3 segments (P<0.01).

The onset of sensory block by the pinprick test at the S1 and S3 segments in the lidocaine–epinephrine–bicarbonate group was significantly more rapid than that in the lidocaine group [16.6 (4.2) vs 19.0 (2.4) min, 12.5 (6.6) vs 16.5 (4.9) min; P<0.05; Fig. 2]. However there was no difference in

| Table 1 Patients characteristics. Values are mean (SD). Cephalad and caudal level are median [range]. Fluid administration volume was the first hour from induction. **P<0.01 compared with both lidocaine group and lidocaine–epinephrine group. |
|----------------------------------|----------------|----------------|
|                                  | Lidocaine      | Lidocaine–epinephrine | Lidocaine–epinephrine–bicarbonate |
|                                  | group (n=9)    | group (n=9)            | group (n=9)                        |
| Height (cm)                      | 160.3 (8.3)    | 157.1 (5.5)            | 160.1 (10.3)                       |
| Cephalad level                   | T10 [T3-L4]   | T9 [T4-L1]             | T10 [T4-L3]                        |
| Caudal level                     | S4 [S1-5]     | S5 [S4-5]              | S5 [S5]                           |
| Weight (kg)                      | 52.7 (5.5)     | 55.62 (8.8)            | 53.8 (11.1)                        |
| Age (yr)                         | 36.9 (9.4)     | 41.6 (9.5)             | 45.4 (16.5)                        |
| M/F                              | 5/4            | 4/6                    | 3/6                                |
| pH                               | 6.51 (0.05)    | 6.64 (0.06)            | 7.42 (0.07)**                      |
| Liquid administration (ml kg⁻¹ h⁻¹) | 8.9 (4.0)    | 9.2 (4.8)              | 9.8 (3.9)                          |
| Surgery type                     | General surgery | 1                     | 1                                  |
|                                  | Gynaecological | 4                     | 5                                  |
|                                  | Orthopaedics   | 4                     | 3                                  |

| Table 2 Haemodynamic data, Bromage scale and pulse amplitude multiples. Values are mean (SD). *P<0.05, **P<0.01, and **P<0.01 after 20 min compared with before induction in each group. †P<0.05 after 20 min compared with before induction in each group. |
|----------------------------------|----------------|----------------|
|                                  | Before induction | 20 min after epidural injection |
|                                  | Lidocaine group |             |                               |
| MAP (mm Hg)                      | 94.8 (10.1)     | 91.7 (8.5)   |
| HR (beats min⁻¹)                | 85.2 (13.8)     | 91 (18.1)    |
| Bromage scale                    | 0               | 1.4 (0.5)*   |
| Pulse amplitude multiples       | 1               | 4.6(2.6)*    |
|                                  | Lidocaine–epinephrine group |             |                               |
| MAP (mm Hg)                      | 88.2 (17.7)     | 84.1 (12.6)  |
| HR (beats min⁻¹)                | 76.1 (14.1)     | 93.7 (14.5)**
| Bromage scale                    | 0               | 1.6 (0.7)*   |
| Pulse amplitude multiples       | 1               | 3.4 (2.0)†   |
|                                  | Lidocaine–epinephrine–bicarbonate group |             |                               |
| MAP (mm Hg)                      | 89.3 (11.5)     | 81.8 (12.9)  |
| HR (beats min⁻¹)                | 70.7 (14.7)     | 88.8 (17.9)**
| Bromage scale                    | 0               | 1.8 (0.4)**  |
| Pulse amplitude multiples       | 1               | 2.8 (2.8)†   |

MAP=mean arterial pressure; HR=heart rate.
Discussion

We have demonstrated that the use of lidocaine with added bicarbonate and epinephrine for lumbar epidural anaesthesia at L4/5 or L5/S1 enhanced the pain threshold of the S1 and S3 segments and blocked the sacral segments within 21 min (mean=16.6 min). The change of pH resulting from the addition of bicarbonate to lidocaine results in an increase in the extraneural amount of non-ionized lidocaine, which is the form that diffuses through the lipid phase of the neural membrane. An increase in the non-ionized fraction of the local anaesthetic thereby facilitates improved nerve penetration and a more rapid onset of nerve block. Epidural epinephrine results in reduced vascular uptake of local anaesthetic, which increases its concentration and a consequent reduction in local anaesthetic clearance from the epidural space. Epidural epinephrine also provides segmental hypoalgesia. The reason for this is that epinephrine was absorbed into the cerebrospinal fluid and bound to α₂-adrenoreceptors. An increase in non-ionized lidocaine because of bicarbonate, activation of the α₂-adrenergic system and an effect on drug redistribution by inducing vasoconstriction because of epinephrine may contribute enhanced block of the sacral segments.

Block of the S1 segment in the lidocaine and lidocaine–epinephrine groups was delayed in this study. Our results indicate that the block of S3 is easier than that of S1 whilst the S3 segment is blocked as rapidly as the L2 segment. The data indicate that, with the exception of the S1 segment, the caudal spread of lumbar epidural anaesthesia using a catheter technique at L4/5 or L5/S1 is comparable with cephalad spread. Lumbar epidural anaesthesia produced sufficient block of the sacral segments except S1. The delayed block of S1 may be explained by the large size of this nerve root compared with those of other segments. These results stress that particular attention must be paid to the block of the S1 segment during the evaluation block of sacral segments. The S4 and S5 segments were not examined in our study because Axelsson and colleagues reported that the S3 segment could be blocked just as rapidly as both the S4 and S5 segments.

In previous reports, anaesthetic level was usually evaluated by pinprick or cold testing. The information obtained by these tests is usually limited to two responses, that is the presence or absence of sensation. The stimulation provided by pinprick or cold may be weaker than that of surgery. It has been reported these tests do not necessarily predict pain-free skin incision. Quantification of analgesia is needed in order to evaluate the block more accurately. It may be possible to quantitively assess the degree of sensory block by measuring the threshold of pain sensation by our repeated electrical stimulation. We believe that repeated electrical stimulation might be a more reliable method than pinprick or cold testing. Our results indicated that absence of pinprick sensation corresponded to a pain threshold of approximately 30–50 mA.

Fig 1 Changes of the pain threshold. Values are mean (SD).

L2 segment

S1 segment

S3 segment

Fig 1 Changes of the pain threshold. Values are mean (SD). L2 segment: there were no significant differences among the groups. S1 segment: *P<0.05 compared with the lidocaine–epinephrine group. (Repeated measures ANOVA, post hoc test; Bonferroni adjustment.) There was a statistically significant difference between the S1 segment and both the L2 and S3 segments at 20 min in the lidocaine–epinephrine group (P<0.01, symbol not shown). S3 segment: *P<0.05 compared with the lidocaine–epinephrine group. **P<0.01 compared with the lidocaine group. (Repeated measures ANOVA, post hoc test; Bonferroni adjustment.)

the onset of sensory block at the L2 segment between the groups [lidocaine group: 15.0 (4.1), lidocaine–epinephrine group: 11.0 (5.7), lidocaine–epinephrine–bicarbonate group: 12.5 (5.3) min; Fig. 2].
It is not clear why the addition of epinephrine to lidocaine did not enhance the epidural block in this study. Murphy and colleagues\(^9\) reported that the caudal spread of analgesia was improved with an epinephrine-containing solution. In their report, the complete block of sacral segments may have required 36 min. Curatolo and colleagues\(^21\) have reported that epidural epinephrine produces segmental hypoalgesia. Evaluation was performed 30 min after the administration of the solution in their study. These reports suggest that epinephrine might produce an enhancement of the block.
over a period of 30 min. However, we assessed the extent of
the block only to 20 min after administration of the local
anaesthetic and this may explain why we did not detect a
significant difference.

We conclude that a combination of lidocaine, bicarbon-
ate, and epinephrine increases the pain threshold of the
sacral segments. We must pay particular attention to the
block of the S1 segment during evaluation. Lumbar epidural
anaesthesia with the lidocaine±epinephrine±bicarbonate
combination provides an acceptable alternative in cases
where caudal epidural anaesthesia is difficult to perform.

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