Bilateral L1 and L2 dorsal root ganglion blocks for discogenic low-back pain

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Background. It is possible that interruption of nociceptive input from intervertebral discs can be modulated through bilateral L1 and L2 dorsal root ganglia (DRG) blockade. In order to test this hypothesis, we prospectively collected data from patients with low-lumbar pain, accurately diagnosed as discogenic using provocation discography.

Methods. Twelve patients were recruited with a mean (SD) symptom duration of 13.7 (8.2) years. Bilateral DRG blocks of L1 and L2 were performed using methylprednisolone 80 mg, clonidine 75 μg and 0.5% bupivacaine 4 ml in each patient.

Results. Analysis of Brief Pain Inventories showed no significant change in pain scores.

Conclusion. We conclude that blocks of this nociceptive pathway in humans using bilateral DRG blocks has no therapeutic value.

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It has recently been discovered that information from intervertebral disc innervation ascends in the sympathetic chain and then enters the spinal cord through the L1 and L2 dorsal root ganglia (DRG; Figs 1 and 2). This has led to investigations of therapeutic interventions to reduce afferent signals in patients with low-back pain.1-4 Nakamura and colleagues reported positive results with unilateral selective local anaesthesia of the L2 nerve root5 and Simopoulos and colleagues with radiofrequency lesioning of the L2 ramus communicans.6 However, patient numbers in these reports were small and the diagnostic criteria used by Nakamura and colleagues were inadequate to determine whether or not true discogenic pain was being studied. In low-back pain, clinical data augmented with findings of magnetic resonance imaging cannot lead to a reliable diagnosis and diagnostic provocation discography is required to prove whether pain is of discogenic origin.7 We carried out this prospective investigation to test whether bilateral L1 and L2 DRG blocks would be a potentially useful intervention for patients with low-lumbar discogenic pain, diagnosed accurately using prior provocation discography.

Methods

After the approval of Local Research Ethics committee, patients with chronic long-term stable or recurrent low-back pain who attended our Pain Clinic between July 2006 and March 2007 were assessed to participate in the study. All had had a negative response to epidural steroids, and other causes of low-back pain had been excluded using MRI scanning and negative responses to medial branch blocks and or sacroiliac joint blocks. All had been accurately diagnosed as having discogenic pain using provocation discography of the lower lumbar intervertebral levels carried out within 1–2 yr of recruitment to the study according to International Spinal Intervention Society (ISIS) recommendations,7 with the exception that pressure monitoring had not been used because of a lack of equipment availability. The leading author and operator (J.R.) is very experienced in this diagnostic technique and particular care was taken not to over-pressurize any disc, thereby producing a false-positive diagnosis. In all patients the level(s) of disc pain involved were L3/4, L4/5, L5/S1, or all.
Written consent was obtained from all recruited patients. A Brief Pain Inventory (BPI, with permission) was completed pre-procedure with the aim of repeating this at monthly intervals post-intervention until pain relief ceased. After pre-procedure completion of the form patients underwent bilateral L1 and L2 DRG blocks according to ISIS guidelines. All injections were performed by a single practitioner (J.R.). Particular care was taken to ensure that optimal contrast medium flow was observed into the intervertebral foramen outlining the DRG at each level. In total, methylprednisolone 80 mg (Depo-Medrone, 40 mg ml⁻¹, Pharmacia Ltd, Milton Keynes, UK), clonidine 75 μg (Catapres, 150 μg ml⁻¹, Boehringer Ingelheim Ltd, Bracknell, UK) and 0.5% bupivacaine 4 ml (Marcain, AstraZeneca UK Ltd, Luton, UK) were injected per patient, being evenly distributed among the four ganglia. After operation, patients remained supine for 1 h with cardiovascular monitoring and went home the same day.

Criteria excluding patients from the study were: discogenic pain rostral to the L2-3 level, refusal to participate, mental incapability of understanding the aims or methods proposed, allergy to any of the drugs, pregnancy, and obesity.

Assessment of the BPI data and data entry was by separate practitioners (N.C./A.J.S.). Statistical analysis was undertaken using Stata, Release 9.2. Using the Shapiro–Wilk test for normal data on the pain score differences, only the ‘pain score right now’ difference appeared as a reasonably normal distribution (P > 0.01 for the other three differences). Therefore, for consistency the Wilcoxon matched-pairs signed-ranks test was used for all four pain score differences. Bonferroni correction for pain score results gives a P-value threshold for significance of 0.0125. Recruitment ceased at 12 patients because there appeared to be no evidence of any meaningful effect. A retrospective power analysis was performed, for which it was judged that the minimum clinical important change would be 1 unit change on any one of the four pain measures. For 80% power, using a Bonferroni-corrected alpha of 0.0125 and a two-tailed test, 12 subjects would be required. Our study was therefore adequately powered to find an effect size of this magnitude.

With regard to the activity of daily living scores using the Shapiro–Wilk W test, not all the variables approximated a normal distribution; hence for consistency, a Wilcoxon matched-pairs signed-ranks test was used.

**Fig 1** ‘Traditional’ understanding of lumbar intervertebral disc afferent pathways. The sinuvertebral nerve, being a branch of the anterior ramus and the sympathetic chain was originally thought to transmit all the afferent information (e.g. from the L5/S1 intervertebral disc) via the anterior ramus directly into the cauda equina.

**Fig 2** Afferents from the sinuvertebral nerve travel outside the spine, up the sympathetic chains before entering the neuroaxis via the L1 and L2 dorsal root ganglia. Anterior rami afferents also entering the neuroaxis at these levels carry afferent input from the skin of the groins. The false-localizing symptom of groin pain with low lumbar discogenic pain (e.g. from the L5/S1 in the diagram) is thus explained.¹⁻⁴

**Results**

All patients approached agreed to take part and all completed the study. Twelve patients (eight males) were recruited and completed the BPI pre-procedure and 1 month later. Mean (sd) patient age was 45.2 (7.9) years and preoperative symptom duration 13.7 (8.2) years.

Pain patterns drawn on a human diagram predominantly involved the lower back extending in five drawings to the groin and hip and in two drawings into the leg to the ankle. Post-procedure there was no noticeable shrinkage of the pain regions represented on the human diagram and in seven instances these areas had visibly increased. No significant differences in pain scores pre-procedure vs...
Table 1 Mean (SD) differences in pain scores pre- and 1 month post-procedure

<table>
<thead>
<tr>
<th></th>
<th>Pre-procedure</th>
<th>Post-procedure</th>
<th>Difference (1 month)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Worst pain score in last 24 h (0–10)</td>
<td>7.3 (1.5)</td>
<td>6.8 (1.9)</td>
<td>0.6</td>
</tr>
<tr>
<td>Least pain score in last 24 h (0–10)</td>
<td>4.6 (1.5)</td>
<td>4.5 (1.9)</td>
<td>0.1</td>
</tr>
<tr>
<td>Average pain score (0–10)</td>
<td>6.1 (1.2)</td>
<td>6.3 (1.2)</td>
<td>−0.2</td>
</tr>
<tr>
<td>Pain score right now (0–10)</td>
<td>6.3 (1.1)</td>
<td>6.3 (1.4)</td>
<td>−0.08</td>
</tr>
</tbody>
</table>

Table 2 Mean (SD) pain interference scores regarding daily life in the last 24 h pre-procedure and at 1 month post-procedure

<table>
<thead>
<tr>
<th></th>
<th>Pre-procedure</th>
<th>Post-procedure</th>
<th>Difference (1 month)</th>
</tr>
</thead>
<tbody>
<tr>
<td>General activity</td>
<td>6.8 (1.7)</td>
<td>6.8 (1.5)</td>
<td>0</td>
</tr>
<tr>
<td>Mood</td>
<td>6.1 (1.5)</td>
<td>6.25 (1.6)</td>
<td>−0.25</td>
</tr>
<tr>
<td>Walking ability</td>
<td>5.6 (2.3)</td>
<td>5.8 (2.0)</td>
<td>−0.25</td>
</tr>
<tr>
<td>Normal work</td>
<td>7.2 (1.7)</td>
<td>6.9 (1.6)</td>
<td>0.3</td>
</tr>
<tr>
<td>Relations with other people</td>
<td>5.3 (2.7)</td>
<td>4.4 (2.8)</td>
<td>0.8</td>
</tr>
<tr>
<td>Sleep</td>
<td>5.7 (2.8)</td>
<td>5.8 (2.2)</td>
<td>0</td>
</tr>
<tr>
<td>Enjoyment of life</td>
<td>6.7 (2.4)</td>
<td>6.1 (2.5)</td>
<td>0.6</td>
</tr>
<tr>
<td>Ability to concentrate</td>
<td>5.2 (2.1)</td>
<td>5.0 (1.6)</td>
<td>0.2</td>
</tr>
<tr>
<td>Appetite</td>
<td>4.1 (2.5)</td>
<td>3.7 (2.8)</td>
<td>0.42</td>
</tr>
</tbody>
</table>

1 month post-procedure were found when considering pain at its worst in the previous 24 h, pain at its least in the previous 24 h, pain on average and pain right now (having accounted for multiple testing of pain scores; Table 1). Usage of analgesic medications was unaffected except in one patient in whom it had increased. No statistically significant differences were found before the intervention and at 1 month in: ability to perform activities of daily living, walking, mood, sleep, and personal relations. Changes in enjoyment of life approached significance but it was felt that owing to the small mean difference between pre- and post-procedure scores, and the absence of an effect on other measures that could explain a meaningful difference on this measure, this was of little clinical importance (Table 2). Data collection ceased at 1 month because of these universally negative outcomes.

Discussion

Low-back pain is a prominent cause of morbidity in industrialized counties and carries with it severe economic consequences. Many forms of patient management are offered but outcome data frequently remain unimpressive.10 Surgical treatment of low-back pain, more so than for radiculopathic pain (e.g. sciatica), is controversial12 and carries risks and complications collectively summarized by the term the ‘failed back surgery syndrome’. Alternative methods of management are needed and, as in other surgical spheres, minimally invasive disc procedures, for example percutaneous nucleoplasty, intradiscal electrothermal therapy, have been developed. Patients were recruited to this study having been accurately diagnosed as having low-lumbar discogenic pain but having been subsequently refused funding by the National Health Service for minimally invasive disc treatments.

Pain arising from the intervertebral disc, or discs (discogenic pain), is probably the most common identifiable reason for low-back pain.13 Understanding the nerve supply to the intervertebral disc potentially opens up pathways to interruption, with consequent pain improvement. A stimulus for investigation into intervertebral disc nerve pathways was the long-standing clinical conundrum of groin pain in association with low-back pain. With no known relationship between the innervation of the groin and the lower lumbar intervertebral discs, upper lumbar pathology was avidly, and frequently erroneously sought.

The intervertebral disc is surrounded by a network of nerve fibres which form into ventral and dorsal plexuses within the anterior and posterior longitudinal ligaments. The rami communicantes and the sinuvertebral nerves interconnect with these plexuses alongside the lateral borders of the intervertebral discs.1–3 These sympathetically-related nerves are thought to have a nociceptive function either directly or by the sensitisation of nociceptors and altered sympathetic activity within the spinal cord.4 5 Stimulation of any of the lower lumbar intervertebral discs in the rat has been observed to result in plasma extravasation (sweating) within its L2 dermatome.1 Nakamura and colleagues hypothesized that the afferent pain pathways for the lower intervertebral discs were predominantly via the sympathetic nerve afferents from the sinuvertebral nerves in the L2 nerve root.5 Morinaga showed that the anterior portion of the L5/6 disc in the rat is innervated from the L1 and L2 DRG (Fig. 2), as opposed to being transmitted entirely by the anterior ramus into the cauda equina (Fig. 1), explains the presence of groin pain as a false-localizing sign. The L1 and L2 DRGs hence become legitimate targets for afferent blocks.

Our negative results require exploration. First, it is quite possible that the intervention used was not capable of producing long-term pain relief. The duration of action of bupivacaine augmented with clonidine and depo-steroid may have been too short for the first data collection which started at 1 month. Questioning of patients, however, refuted even short-term benefit. This was in contrast to the results of Nakamura and colleagues who showed an average of 20.7 days (1.5 h–100 days) using 1.5 ml of plain 1% lidocaine.5 The reason we hoped infiltration around the DRG in our study might have been long-lasting was because we used a bilateral two-level technique using combinations of drugs with a longer duration of action. Clonidine can extend local anaesthetic action and having
blocking spontaneous discharge. Bupivacaine is longer acting than lidocaine. Studies reporting on the efficacy of selective nerve root blocks in radiculopathic pain show long-term highly effective pain relief, well beyond the pharmacological durations of action of the injectates. Although the design of our study may have been inadequate to demonstrate a very short-term symptom improvement, the potential usefulness of this as a treatment for discogenic low-back pain is obviously negative. It is possible that a prolongation of effect through pulsed radio-frequency lesioning may have been more effective, but the lack of even transient pain relief makes this unlikely.

A second possible reason for lack of efficacy is that as much of the anatomical work of disc innervation has been carried out in the rat, it is possible that species differences may be operative.

Thirdly, the amount of nociceptive information from the intervertebral disc entering the neuroaxis directly via the anterior primary ramus (as opposed to travelling up the sympathetic chain and then entering the neuroaxis at the L1 and L2 levels) cannot be known in any individual. It is possible in our patients that the predominant nerve pathway was not via the sympathetic chain (as shown in Fig. 2).

Fourthly, our diagnostic criteria using provocation discography did not involve intradiscal pressure measurement as recommended by ISIS, because of a lack of equipment availability. The experienced discographer was careful to avoid over-pressurization but this was only judged empirically.

We conclude that infiltration around the DRG of L1 and L2 is an ineffective intervention in chronic discogenic low-back pain. Our study data are insufficient to challenge the discovery of sympathetic chain pain transmission of nociceptive intervertebral disc afferents as opposed to the traditionally accepted segmental pathway via the cauda equina.

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References

419