We studied 27 patients with low back pain and unilateral L5 or S1 spinal nerve root pain. Significant radiological changes were restricted to the symptomatic root level, when compared with controls. Low back and leg pain were graded on a visual analogue scale. Dermatomal quantitative sensory tests revealed significant elevations of warm, cool and touch perception thresholds in the affected dermatome, compared with controls. These elevations correlated with root pain (warm v L5 root pain; \( r = 0.88, p < 0.0001 \)), but not with back pain. Low back pain correlated with restriction of anteroposterior spinal flexion (\( p = 0.02 \)), but not with leg pain.

A subset of 16 patients underwent decompressive surgery with improvement of pain scores, sensory thresholds and spinal mobility. A further 14 patients with back pain, multilevel nerve root symptoms and radiological changes were also studied. The only correlation found was of low back pain with spinal movement (\( p < 0.002 \)). We conclude that, in patients with single level disease, dermatomal sensory threshold elevation and restriction of spinal movement are independent correlates of sciatica and low back pain.

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Spinal pain is the most common musculoskeletal symptom. Between 50\% and 80\% of the population suffer at least one disabling bout of low back pain (LBP) during a lifetime. It has been estimated that during 1993 the cost of back pain to the British National Health Service exceeded £481 million, while indirect costs of social security and lost work exceeded £5 billion.1 Following an episode of LBP, 90\% of sufferers recover within three months, about 10\% remain disabled after three months and approximately 3\% have surgical treatment.2 Of the associated symptoms, pain radiating to the leg, sensory changes and motor weakness are the most important, and pain in the leg the most common.2 Pain or numbness in the legs due to compression of a spinal nerve root is often associated with lumbar degenerative disorders, such as disc herniation or spinal canal stenosis. Mechanical nerve root compression may not explain all cases of sciatica. It has been suggested that material from the nucleus pulposus induces neurological symptoms by mechanisms other than simple mechanical compression.3 Several studies have shown biomechanical interactions between disc tissue and nerve roots; chemical substances in the epidural space may penetrate the root sheath contributing to or triggering ‘neuropathic’ pain.4-6

The bedside neurological examination contributes to the diagnosis, particularly if deep tendon reflexes are affected, but muscle weakness or atrophy are relatively uncommon at presentation.7 Imaging may also be of limited value as a normal CT of the spine can be found in 29\% of symptomatic patients, and many abnormalities have been observed on MRI in symptomatic patients.8 Electromyographic tests, which are used to evaluate nerve fibres of large diameter, may be normal in the presence of severe pain.

Quantitative sensory testing (QST) has been used to assess the function of small and large nerve fibres; warmth is mediated by the unmyelinated C fibres, cool sensation by the thinly myelinated A fibres and C fibres and sensation of light touch by the large myelinated A\( _β \) fibres. Several studies have investigated sensory deficits in patients with sciatica, with varying results.9,10 To our knowledge, there has been no objective study relating a patient’s assessment of pain to sensory abnormalities. Recovery of sensory nerve function after surgical decompression has been reported to depend on several factors, including duration of symptoms, nerve fibre size and time after operation.11

The purpose of our study was to relate reported pain with dermatomal sensory nerve dysfunction in patients with low back pain and unilateral single level sciatica. We also sought
Patients and Methods

The study group comprised 27 patients who presented with LBP and unilateral single nerve root symptoms (Quebec Task Force grading 3 to 4) in the L5 or S1 dermatomes. The patients had had symptoms for a mean of two years (0.15 to 10). All patients who had previous spinal surgery, co-existing neurological disorders or were skeletally immature were excluded from the study. Most patients had been treated previously with physiotherapy and were using regular analgesia; 15 patients were taking non-steroidal anti-inflammatory drugs, nine were also taking opiates and 12 patients a combination of the two; three were additionally taking a morphine derivative. A further 14 patients with multilevel nerve root symptoms (graded similarly 3 to 4) and with a mean of 9.4 years’ history of LBP (4 to 30) were also studied; ten volunteers who worked at the hospital and had no previous history of LBP acted as controls and underwent quantitative sensory testing. Clinical data for the patients and controls are shown in Table I. Written informed consent was obtained from all subjects. The study had ethical approval.

A detailed history was taken from all patients followed by a full neurological and orthopaedic examination. All had MR or CT scans of their lumbo-sacral spine; 24 had nerve root compression from a bulging or protruding disc on MRI and three had degenerative facet joints on CT. These appearances were restricted to the symptomatic root level. None had weakness or diminished deep tendon reflexes in the relevant areas. The 14 patients with multilevel symptoms had clinical and radiological evidence of canal stenosis or nerve root compression at more than one level. Most had reduced deep tendon reflexes at one or more levels and weakness attributed to one or more nerve root. All graded their pain on a visual analogue scale (VAS) and underwent dermatomal sensory testing and analysis of spinal movement.

All patients were asked to detail their site of pain on body charts and to grade their level of LBP and leg pain as it was on the day of interview, as it usually was and the worst it had been in the last week. An average of these assessments was taken as their VAS.

QST was carried out in a silent room at an ambient temperature between 20° and 23°C by methods that have been described previously. The sites of testing were at the mid-points of the L4, L5 and S1 dermatomes according to the MRC memorandum. Quantitative thermal thresholds were determined by the Marstock method with a thermistor used according to the Peltier principle (Thermostat type 1, Somedic AB, Stockholm, Sweden). The baseline temperature was 30°C with a rate of change of 1°C. The limits of temperature were 10°C and 50°C. Detection thresholds for cool and warmth were recorded as a mean of four tests. The threshold of touch perception was measured using monofilaments or ‘hairs’, numbered 1 to 20 according to stiffness and directed at 90° to the surface of the skin to the point at which each would bend. Correct identification of touch in five consecutive tests was taken as the threshold. The relationship between the hair number and log 10 force is linear, enabling statistical analysis.

The range of movement of the lumbar spine was recorded using a computerised triaxial potentiometric analysis system (CA-6000 Spinal Motion Analyser, OSI, Hayward, California). A series of three constrained movements into flexion and extension (anteroposterior movement) were undertaken at the subject’s preferred pace, within the range of comfort. The range was then calculated as the mean of these three readings. The accuracy and repeatability of this system has been established previously.

Of the 27 patients with unilateral sciatica, 16 underwent discectomy or nerve root decompression carried out by the same surgeon (SPFH), and had the above tests performed by the same investigator (NAQ), at intervals of six weeks (n = 16), six months (n = 15) and one year (n = 11) after surgery.

The elevation of threshold for each dermatome was calculated from the mean threshold at the same dermatome for controls. The elevation of sensory threshold for each modality was correlated with LBP and leg pain scores (VAS) using Spearman’s correlation coefficient. Unpaired t-tests (Mann-Whitney U test) were used to calculate the levels of significance between QST thresholds and controls. Spearman’s correlation coefficient was used to determine significance between pain scores and sensory thresholds using the GraphPad Prism biostatistical analysis package (version 3.0).
for PC, GraphPad Software, San Diego, California) on a personal computer. For the post-operative follow-up study, RANOVA was used.

**Results**

All 27 patients with single level pathology experienced LBP with radiation down a leg; 14 patients experienced leg pain localised to the L5 dermatome and 13 had evidence of S1 sciatica (clinical and imaging findings). The mean leg pain score in patients with single level symptoms was higher than the mean LBP score ($p = 0.2$), but the results did not reach significance ($p = 0.09, r = 0.29$, Table I). In patients with multilevel involvement, back pain scores were higher than leg pain scores, although this was not significant ($p = 0.49$). There was no correlation between these scores ($p = 0.37$).

The results for the warm detection thresholds in single level patients are shown in Figures 1a and b. The leg affected with sciatica was compared with the unaffected leg in patients with unilateral symptoms in L5 or S1 dermatomes. There was a marked elevation of the warm perception threshold in the symptomatic dermatome of the affected side, compared with the controls. The warm threshold was also significantly raised in neighbouring dermatomes of the affected leg. There was also a trend towards elevation in the non-affected leg, but this was not statistically significant. In the multilevel group, dermatomic warm detection thresholds (WDTs) were compared with equivalent dermatomes in the controls. All were significantly raised with the WDTs in the S1 dermatomes having the greatest difference compared with controls (mean 9.6°C v 30°C, $p = 0.0007$).
The results for the cool perception threshold in single level patients are shown in Figures 1c and d. The L5 and S1 sciatica patients had significantly raised cool thresholds compared with controls in the affected dermatome. The cool threshold was also increased in neighbouring levels, but unlike the warm threshold, it did not reach significance. Similarly, the dermatomal cool detection thresholds (CDTs) in the multilevel patients were compared with the equivalent dermatomes in the controls. The CDTs were found to be higher in the patients compared with the controls, although this was less significant than the WDT measurements (5.6°C v 2.1°C, p = 0.043).

Figures 1e and f illustrate light touch thresholds in single level patients where monofilament numbers are equivalent to log 10 forces. Light touch thresholds were raised significantly in the respective dermatomes affected by sciatica, and also in S1 dermatomes in patients with L5 sciatica. The light touch threshold in the multilevel patients was also found to be significantly raised in all dermatomes compared with those in the controls. In single level patients with either L5 or S1 root symptoms, the WDT was found to correlate significantly with the leg pain score of the affected dermatome, but not with the LBP score (Fig. 2). There was also significant correlation between the change in cool threshold in the S1 dermatome and the touch threshold in the L5 dermatome, with leg pain scores (p = 0.02 and p = 0.006, respectively). These were not so marked as the changes in the warm threshold. Further, when ‘typical pain scores over the last week’ were correlated with the warm threshold, a good correlation was observed (L5; p = 0.002, r = 0.8; S1 = 0.008, r = 0.7), but this was not so with LBP scores (L5, r =0.3; S1, r =0.4). The significance was maintained when a patient with a leg pain score of four or more (moderate to severe pain) was compared with the change in warm threshold at the affected level (p < 0.0001, r = 0.8). In multilevel patients, no significant relationship was found between sensory modalities and leg or back pain scores.

The measurements of pain-free spinal flexion were then compared with low back and leg pain VAS scores as shown in Figure 3. There was a significant correlation with low back pain scores, but not with leg pain scores. Multilevel patients showed similar results (Fig. 4a).

For the cohort of 16 patients who underwent surgical treatment (discectomy and nerve root decompression), the
mean leg pain VAS (1.4 ± 2.4) and LBP VAS (1.8 ± 2.4) scores were significantly lower compared with pre-operative levels at one-year follow-up (LBP VAS, p = 0.0005; leg pain VAS, p < 0.0001). Figure 4a shows changes in the warm threshold after operation, with the affected dermatome value collated. Warm thresholds were significantly different from the pre-operative level at all stages (pre-operative v controls, p < 0.0001, difference = 3.9, confidence interval (CI) 2.1 to 5.9). At one-year follow-up, there was no significant difference from controls. Cool thresholds were decreased significantly from their pre-operative level at one-year (Fig. 4b, p = 0.0067, difference = 2.83, CI 0.81 to 4.84). Light-touch
thresholds (Fig. 4c) were significantly higher at the pre-operative stage, than for the controls and decreased over the assessment period, having fallen significantly at one-year (p = 0.0001, difference = 2.5, CI 1.5 to 3.52). Pain-free flexion (Fig. 4d) improved over the assessment period, being significantly different from the pre-operative level at six months (p = 0.05, difference = 11.28, CI 0.02 to 22.55) and at one year (p = 0.019, difference = 15.89, CI 2.7 to 29.8).

Discussion

We found that dermatomal sensory thresholds to warm, cool and touch stimuli are elevated in patients with LBP and single level unilateral symptomatic radioculopathy. In general, the increase in threshold was most pronounced in the dermatome of the symptomatic nerve root, less so in the ipsilateral neighbouring dermatomes, even less in the dermatome contralateral to the symptomatic one and least in the dermatome of the symptomatic nerve root, less so in the single level unilateral symptomatic radioculopathy. In general, the increase in threshold was most pronounced in the dermatome of the symptomatic nerve root, less so in the ipsilateral neighbouring dermatomes, even less in the dermatome contralateral to the symptomatic one and least in the contralateral neighbouring dermatomes. These findings confirm previous reports.9 The warm threshold was raised significantly in neighbouring non-symptomatic dermatomes, suggesting that while the warm threshold may be a sensitive marker for nerve root involvement, an additional ‘trigger’ may be required to produce pain at the symptomatic level. We report, for the first time, that the degree of elevation of the warm sensory threshold in affected dermatomes was highly and linearly correlated with root pain scores, but not with back pain scores. Conversely, back pain scores correlated closely with the pain-free spinal range of flexion (p = 0.02), but not with leg pain scores. The cool threshold was significantly raised only in the involved dermatome, which may show this to be a more specific marker than the warm threshold. It also correlated with leg, but not with back pain scores. Touch thresholds showed a similar pattern.

The affected root or dermatome had the maximum elevation of the sensory perception thresholds, with the threshold for warmth being more sensitive, and for cold more specific. The level of root pain correlated more closely with the degree of elevation of the warm sensory threshold. Assessment of all three sensory modalities should be considered in predicting the affected dermatome; fibres of every calibre may need to be involved to produce pain, reflecting a more severe lesion. Combining the range of spinal movement with the back pain score can help to distinguish neuropathic (nerve root) from nociceptive (low back) pain. The pathological mechanisms underlying the relationship of sensory threshold elevation to spinal root pain in such patients are unknown. Demyelination or ion channel re-distribution and consequent delay in nerve conduction may be responsible for the elevated sensory threshold and a trigger, either mechanical or chemical, may produce pain in proportion to the pathology. The following observations would support this suggestion:

1) Elevated sensory thresholds alone cannot be related to pain, as these are significantly elevated in non-symptomatic dermatomes. Our evidence for this reflects previous studies.9,10

2) In our sub-set of patients who had decompressive surgery, sensory thresholds were improved, often within weeks of surgery, and were indistinguishable from healthy controls at one year after surgery, confirming the report by Nygaard et al.11 Relief of leg pain accompanied improvement of sensory thresholds, which suggests that re-myelination or ion channel re-distribution has occurred, and argues against significant axonal damage (axotomy).

3) In a study on pig’s cauda equina, autologous nucleus pulposus applied to the nerve roots reduced nerve conduction velocities even in the absence of compression.20 This would support the possibility of ion channel re-distribution at the affected level demonstrated by Bucknill et al.21

4) Nerve compression alone does not usually cause pain, and some degree of irritation or inflammation may be required to produce or trigger symptoms.22,23

5) Evidence for chemical and mechanical triggers exists in the form of biochemical interactions between disc tissue and nerve roots, chemical substances in the epidural space which may penetrate the root sheath and mechanical stimuli, particularly nerve stretching.4-6,24,25

There are few studies of human tissue from such patients. In more complex or severe cases with central axotomy, there may be other factors which may preclude any simple relationship between elevated dermatomal sensory thresholds and radiating leg pain. One study of biopsy specimens from nerve roots obtained during surgery showed that chronic nerve root compression is associated with a decrease in the number of large myelinated fibres.26

Animal models of radiculopathy often show hypersensitivity to non-noxious stimuli, termed allodynia. Although this may be seen in patients with sciatica, it is uncommon. No patient in our study had allodynia in the legs, and its absence has been reported by others.27 It is possible that patients with allodynia have a predominantly inflammatory lesion without significant root compression or axonal damage, which would usually lead to elevated sensory thresholds. Partial nerve root damage with central spinal cord changes (sensitisation or disinhibition) may be a cause of allodynia. Thus, animal models which lead to allodynia may be informative about only a small number of patients with back and nerve root pain. In one study, nucleus pulposus material applied to the nerve roots of rats, produced allodynia in the lower leg.28 In a rat lumbar radiculopathy model, mechanical allodynia was observed on both ipsilateral and contralateral sides after reinjury.29 Repeated nerve root injury leads to potentiation of central sensitisation and the development of chronic lumbar radiculopathy. An experimental dog model showed that chronic compression of a lumbar nerve root results in a decrease in the number of large myelinated nerve fibres and an increase in thinly myelinated fibres around the peripheral part of the nerve root.29

In our study, patients with multilevel lumbar degenerative disease and complex symptoms had probably sustained
severe axonal damage. In previous studies of spinal movement in patients with LBP, including those with multiple level pathology and complex symptoms, we found that the range of flexion improved at the six-month stage. In this study our patients’ spinal flexion appeared to have improved at the six-week stage, and continued to improve beyond six months. Other studies have noted a strong correlation between LBP and vertebral movement.32

We believe, that dermatomal sensory thresholds are a useful diagnostic tool in the evaluation of patients with radiating low back and spinal nerve root pain. Dermatomal sensory threshold elevation and restricted spinal movement appear to be independent correlates of sciatica and LBP. It is, thus, possible to distinguish nociceptive and neuropathic components of pain and to consider different mechanisms in groups of patients with LBP and nerve root pain.

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