THE AFFERENT PATHWAYS OF DISCOGENIC LOW-BACK PAIN

EVALUATION OF L2 SPINAL NERVE INFILTRATION

SHIN-ICHIRO NAKAMURA, KAZUHISA TAKAHASHI, YUZURU TAKAHASHI, MASATSUNE YAMAGATA, HIDESHIGE MORIYA

From Chiba University, Chiba, Japan

The afferent pathways of discogenic low-back pain have not been fully investigated. We hypothesised that this pain was transmitted mainly by sympathetic afferent fibres in the L2 nerve root, and in 33 patients we used selective local anaesthesia of this nerve.

Low-back pain disappeared or significantly decreased in all patients after the injection. Needle insertion provoked pain which radiated to the low back in 23 patients and the area of skin hypoalgesia produced included the area of pre-existing pain in all but one. None of the nine patients with related sciatica had relief of that component of their symptoms.

Our findings show that the main afferent pathways of pain from the lower intervertebral discs are through the L2 spinal nerve root, presumably via sympathetic afferents from the sinuvertebral nerves. Discogenic low-back pain should be regarded as a visceral pain in respect of its neural pathways. Infiltration of the L2 nerve is a useful diagnostic test and also has some therapeutic value.

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Low-back pain is often diffuse and ill-defined; there may be little associated tenderness. The nature of this pain is similar to that of visceral pain as described by Ness and Gebhart (1990). Discogenic low-back pain is considered to have afferent pathways in the sinuvertebral nerves, mainly originating from the ventral rami of the spinal nerves (Wiberg 1949; Pedersen, Blunck and Gardner 1956; Bogduk, Tynan and Wilson 1981; Bogduk 1983), but many patients with disc herniation complain of sciatica without low-back pain. This suggests that the spinal nerve roots are being compressed proximal to the branching point of the sinuvertebral nerves and that the afferent pathways for discogenic low-back pain are not in the spinal nerves at the same level.

There is considerable evidence to show that the lumbar sympathetic afferents do play a role in the transmission of low-back pain. This can be summarised as follows:
1) sympathetic afferents can transmit pain (Echlin 1949; Ruch 1955; Gillette, Kramis and Roberts 1994);
2) low-back pain is induced by stimulation of the lumbar sympathetic trunk (White and Sweet 1955);
3) it disappears after a lumbar sympathetic ganglion block (Brena et al 1980; El Mahdi, Abdel Latif and Janko 1981); and
4) the sinuvertebral nerves originate only from the sympathetic trunk (Groen, Baljet and Drukker 1990).

The sympathetic trunk originates from the myelomeres of only T1 to L2 (Johnson and Spalding 1974; Williams et al 1989a), and Foerster (1933) showed that L2 is the dermatome corresponding to the low back. It is known that in rats the sympathetic nerves also originate from the T1 to L2 myelomeres (Coggeshall et al 1977; Gabella 1995).

In recent papers we have reported the following findings in rats:
1) plasma extravasation is induced in the groin skin, which is in the L2 dermatome, by stimulation of the lower lumbar intervertebral discs regardless of the level stimulated (Takahashi et al 1993);
2) the sensory neurones which innervate the anterior portion of the L5/6 disc arise only from the L1 and L2 dorsal root ganglia (Morinaga et al 1996:in press); and
3) the posterior part of the lumbar intervertebral discs is innervated by fibres derived only from the sympathetic trunks bilaterally and multisegmentally (Nakamura et al 1996).

From this evidence, we hypothesised that pain arising from the lower lumbar intervertebral discs was transmitted mainly through the sympathetic afferent fibres contained in the L2 spinal nerve root. To confirm this, we performed

S. Nakamura, MD, PhD
K. Takahashi, MD, PhD
Y. Takahashi, MD, PhD
M. Yamagata, MD, PhD
H. Moriya, MD, PhD
Department of Orthopaedic Surgery, School of Medicine, Chiba University, 1-8-1 Inohana, Chuo-ku, Chiba 260, Japan.

Correspondence should be sent to Dr S. Nakamura at the Department of Orthopaedic Surgery, Sakura National Hospital, 2-36-2 Eharadai Sakura-City, Chiba 285, Japan.

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Table I. Details of 33 patients with discogenic low-back pain

Case	Sex	Age (yrs)	Diagnosis	Level	Pain	
					Low-back	Other
1	M	47	Disc lesion	L4/5, L5/S	Bilateral	
2	M	24	Herniated nucleus pulposus	L4/5	Bilateral	
3	M	23	Disc lesion	L4/5	Right	
4	F	61	Disc lesion	L4/5, L5/S	Right	Buttock and thigh
5	F	52	Disc lesion	L3/4	Right	
6	M	64	Disc lesion	L4/5	Left	Buttock
7	M	57	Disc lesion	L4/5	Right	Groin
8	F	44	Disc lesion	L4/5, L5/s	Left	Thigh
9	F	21	Herniated nucleus pulposus	L5/S	Left	
10	M	41	Herniated nucleus pulposus	L4/5	Right	Thigh and leg
11	M	45	Disc lesion	L4/5, L5/s	Left	Groin
12	M	20	Herniated nucleus pulposus	L5/S	Left	Thigh and leg
13	M	47	Herniated nucleus pulposus	L5/S	Right	Buttock and leg
14	M	23	Herniated nucleus pulposus	L4/5	Left	Buttock and leg
15	M	19	Disc lesion	L4/5	Right	
16	F	72	Disc lesion	L4/5	Right	
17	F	41	Herniated nucleus pulposus	L4/5	Right	Buttock
18	F	52	Disc lesion	L5/S	Right	
19	M	29	Herniated nucleus pulposus	L4/5	Bilateral	Right buttock and leg
20	M	42	Herniated nucleus pulposus	L5/S	Bilateral	Leg
21	M	29	Disc lesion	L4/5	Bilateral	
22	F	21	Herniated nucleus pulposus	L4/5	Bilateral	Left thigh
23	M	53	Herniated nucleus pulposus	L4/5	Left	Buttock and leg
24	M	45	Disc lesion	L4/5	Bilateral	
25	F	66	Herniated nucleus pulposus	L5/S	Left	Leg
26	M	16	Disc lesion	L4/5	Left	
27	M	44	Disc lesion	L4/5	Bilateral	
28	M	29	Herniated nucleus pulposus	L5/S	Right	
29	M	39	Disc lesion	L5/S	Left	
30	M	21	Herniated nucleus pulposus	L5/S	Right	
31	M	26	Disc lesion	L5/S	Right	
32	M	51	Herniated nucleus pulposus	L4/5	Bilateral	Right leg
33	F	47	Disc lesion	L4/5	Bilateral	

selected infiltration of the L2 nerve in a series of patients with chronic discogenic low-back pain.

PATIENTS AND METHODS

We studied 33 patients (23 male and 10 female) who had suffered from low-back pain for at least one month (Table I). Their average age was 39.7 years (16 to 72), and the average duration of low-back pain was 23.6 months (1 to 240).

The clinical diagnosis of discogenic pain was determined by physical examination, plain radiography and MRI. We excluded patients with severe osteoarthritis of facet joints, spondylolisthesis, or spondylolysis. Except for double lesions of L4/5 and L5/S1 we also excluded patients with multilevel disc degeneration and all those who had had previous spinal surgery. The involved levels were determined from MRI (Fig. 1a). The location and intensity of the low-back pain and the area of skin hypoalgesia revealed by a pin-prick test were recorded on a body diagram before infiltration.

Injection method. On the predominantly painful side, the skin was anaesthetised using 1.5 ml of 1% Lidocaine. A 22-gauge spinal-nerve-block needle was then advanced obliquely to the L2 spinal nerve under fluoroscopic control. The site of any pain provoked by this was recorded on a body diagram by the examiner. Then 0.5 ml of the contrast medium Iotorolan (Schering AG, Berlin, Germany) was injected to confirm the location and outlines of the spinal nerve (Fig. 1b); 1.5 ml of 1% Lidocaine was then injected. These blocks were all unilateral.

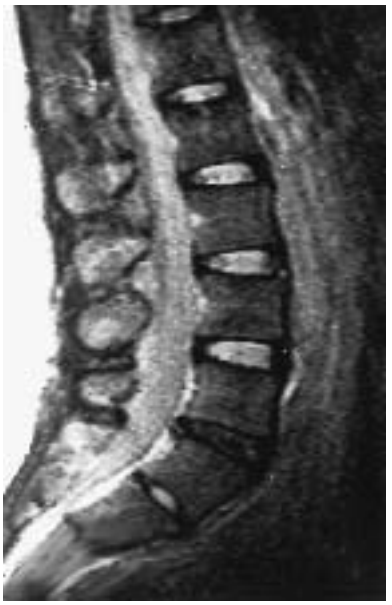

Fig. 1a

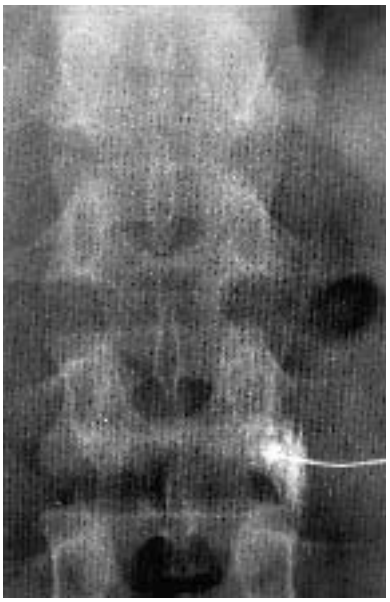

Fig. 1b

Case 31. T2-weighted MRI shows degeneration at L5/S1 (a). A radiculogram showing the site of injection of the right L2 spinal nerve (b).

At 15 minutes after infiltration, the low-back pain and area of hypoalgesia were reassessed for each patient using a visual analogue scale (VAS) (Huskisson 1974), with 20 points for the most severe pain ever felt and 0 for no pain.

The postural provocation of pain was evaluated by a 'flexion test', performed both before and 15 minutes after injection. Patients were asked to maintain a 30° flexed position of the spine. We recorded the pain-provocation time, defined as the period until there was a definite increase in pain, and the pain-endurance time, defined as the period for which the patient was able to maintain the posture, up to three minutes. If a patient was unable to continue the test for reasons other than low-back pain, such as sciatica, we recorded the intensity and location of such pain.

We used Student's paired *t*-test for statistical analysis. All the injections were given by one author (SN) and assessments were by two or more authors.

RESULTS

Needle insertion to the L2 nerve in 33 patients provoked pain in the ipsilateral low back in 23 and in the ipsilateral low back and the buttock in six, in all 29 within the area of pre-existing pain. Four patients had pain provoked in the anterolateral thigh. At about three to five minutes after the infiltration all the patients noted relief of low-back pain: 26 reported almost complete disappearance of the ipsilateral pain and the remaining seven had some relief of the pain by 15 minutes (Table II). On the VAS, ipsilateral low-back pain decreased on average from 10.0 ± 5.2 to 1.7 ± 3.4 ($p < 0.001$; Fig. 2).

Of the ten patients with bilateral low-back pain, five noted bilateral disappearance of the pain, four reported that the contralateral pain was unchanged although the ipsilateral pain had disappeared, and one patient had alleviation of contralateral pain with disappearance of the ipsilateral pain. Of the seven patients with buttock pain, all but one had complete disappearance of this pain and one had some decrease in pain. Five patients with pain in the anterolateral thigh noted complete relief after the procedure, as did two with inguinal pain

Of the nine patients with some leg pain, only three had partial relief and the other six had no change. Six of the 18 patients with tenderness between the spinous processes had complete relief, six some relief, and six had no change. Of the ten patients with a positive straight-leg-raising test, only

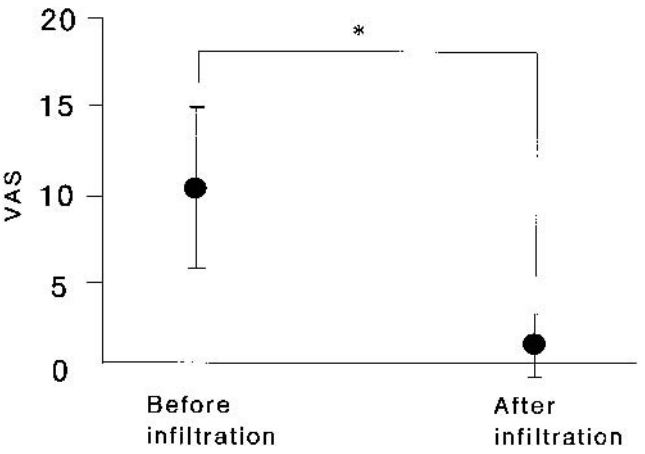

Fig. 2

Visual analogue scale ratings before and after infiltration of the L2 spinal nerve The bars represent the standard deviation (*= $p < 0.001$, Student's paired *t*-test).

Table II. Results of infiltration of the L2 spinal nerve in 33 patients

	Disappeared	Decreased	Unchanged
Low-back pain			
Ipsilateral (n = 33)	26	7	0
Contralateral (n = 10)	5	1	4
Buttock pain (n = 7)	6	1	0
Thigh pain (n = 5)	5	0	0
Inguinal pain (n = 2)	2	0	0
Leg pain (n = 9)	0	3	6
Tenderness			
Interspinous process (n = 18)	6	6	6
Paravertebral muscle (n = 11)	8	3	0
Superior clunial nerves (n = 13)	6	5	2
Sciatic nerve (n = 8)	2	1	5
Positive straight-leg-raising test (n = 10)	0	1	9

one patient showed an increase in the tolerated angle after injection. The pain relief after injection lasted for an average of 20.7 days (1.5 hours to 100 days), with 14 patients having relief for more than one week (Table III).

In the flexion test the average pain-provocation increased from 43.0 ± 38.7 s before injection to 120.1 ± 64.4 s after it ($p < 0.001$; Fig. 3). Sixteen patients, with an average pain-provocation time of 56.3 ± 46.0 s before injection, had no provocation of low-back pain after infiltration. One patient was unable to tolerate the flexion test for more than 40 s because of aggravated sciatica, but had relief from low-back pain.

The average pain-endurance time in the flexion test increased from 99.4 ± 65.2 s to 150.3 ± 47.7 s after injection ($p < 0.001$; Fig. 3), and 21 patients, with an average preinjection endurance time of 126.3 ± 65.2 s, were able to maintain the 30° flexed position for the whole three minutes.

After injection hypoalgesia appeared in the ipsilateral low back, buttock, and the anterolateral thigh in 31 of the 33 patients (Fig. 4). The hypoalgesic areas all included the areas of pre-existing low-back and buttock pain. One patient had an area of hypoalgesia only in the anterolateral thigh and another only in the low back and buttock. No patient had any areas of hypoalgesia on the contralateral side, and none had any apparent motor weakness.

The contrast medium was not seen to diffuse to neighbouring levels, or epidurally, and there were no complications related to the infiltration.

DISCUSSION

Irritation of the L2 spinal nerve provoked pain which radiated to the pre-existing painful area. After injection the low-back pain disappeared, but leg pain was not affected. The hypoalgesic area included the pre-existing painful area, and low-back pain on sustained flexion was significantly suppressed. All these findings suggest that the afferent pathways of discogenic low-back pain are mainly in the L2 spinal nerve.

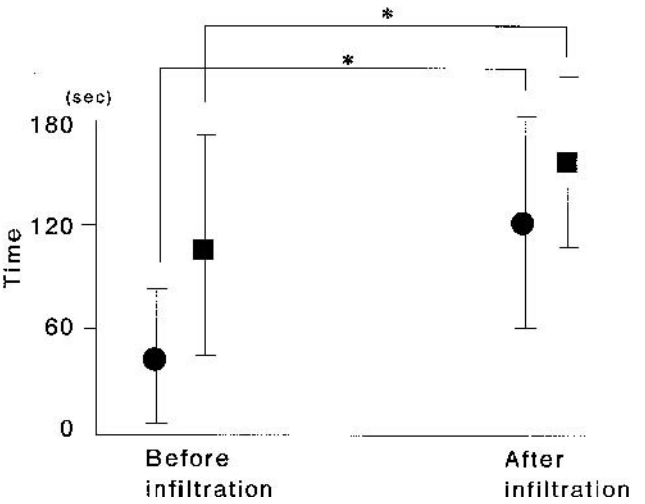

Fig. 3

The results of pain provocation and pain endurance in the flexion test. The bars represent the standard deviation. (● = pain provocation; ■ = *pain endurance; * = $p < 0.001$, Student's paired t*-test).

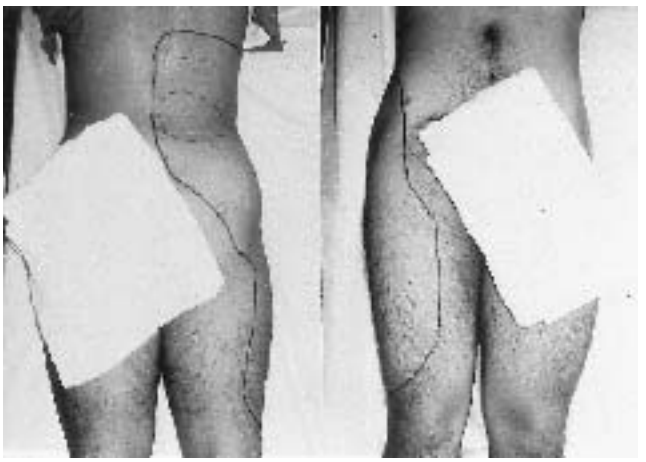

Fig. 4

Case 31. A typical area of hypoalgesia produced by infiltration of the L2 spinal nerve. The area surrounded by the dotted line indicates the area of back pain before injection.

Table III. Duration of the pain relief after infiltration of the L2 spinal nerve in 33 patients

	Duration				
	Under 3 hours	3 to 24 hours	1 to 7 days	1 to 4 weeks	Over 1 month
Number of patients	5	7	7	5	9

The intervertebral disc has long been regarded as the main source of the common form of low-back pain (Hirsch 1949; Wiberg 1949; Smyth and Wright 1958; Nachemson 1976; Mooney 1987). In addition, Kuslich, Ulstrom and Michael (1991) reporting intraoperative findings under local anaesthesia, found that low-back pain was reproduced by stimulation of the outer annulus or the posterior longitudinal ligament, while sciatica was induced by mechanical stimulation of inflamed nerve roots. They also reported that pre-existing low-back pain was not elicited by stimulation of the facet joints. Schwarzer et al (1994) used discography and facet joint block for patients with chronic low-back pain and reported that discogenic pain was more common than facetogenic pain.

Discogenic pain has been considered to be transmitted via the sinuvertebral nerves derived from the spinal nerve of the same segment (Wiberg 1949; Pedersen et al 1956; Bogduk et al 1981), but Wiberg (1949) reported that low-back pain could be evoked by stimulation of the intervertebral disc even after the corresponding two roots on the same side had been anaesthetised.

The sinuvertebral nerves which innervate the posterior parts of intervertebral discs have been reported to be recurrent branches of the spinal nerve (Spurling and Bradford 1939; Roofe 1940; Kojima et al 1990b), but others consider that both spinal (somatic) and sympathetic (visceral) nerves are involved (Wiberg 1949; Pedersen et al 1956; Stilwell 1956; Bridge 1959; Cloward 1960; Kimmel 1961; Edgar and Nundy 1966; Edgar and Ghadially 1976; Bogduk et al 1981; Bogduk 1983; Paris 1983). By contrast, the anterior part of the intervertebral discs has been reported to be innervated only by sympathetic nerves (Stilwell 1956; Bogduk et al 1981; Bogduk 1983; Paris 1983; Weinstein, Claverie and Gibson 1988).

It seems curious that different types of nerve innervate specific portions of the intervertebral discs. Groen et al (1990) reported that human sinuvertebral nerves were derived solely from the rami communicantes from the sympathetic trunks. We have verified in rats by using an acetylcholinesterase histochemical method with or without resection of the sympathetic trunks that the origin of nerves supplying the posterior part of lumbar intervertebral discs is in the sympathetic trunks (Nakamura et al 1996).

Human sympathetic nerves also originate from the T1 to L2 cord levels (Johnson and Spalding 1974; Williams et al 1989a) and are composed of both efferent and afferent fibres (Williams et al 1989a), including those proved to be nociceptive by physiological methods (Jänig and McLachlan 1986; Gillette et al 1994) or immunohistochemistry (Suseki et al 1996:in press). The sympathetic innervation of the lower trunk and the legs is mainly from the T10 to L2 myelomeres (Johnson and Spalding 1974), and therefore any sympathetic nerves innervating the lower lumbar discs should connect with L2 or higher levels. It has already been suggested that low-back pain from the anterior part of the intervertebral discs could be mediated by the sympathetic nervous system (Jinkins, Whittemore and Bradley 1989).

Low-back pain is usually perceived in the areas of the L1 or L2 dermatomes of Foerster (1933). The skin of the low back and buttocks is innervated by the superior clunial nerves, which are terminal cutaneous branches of the dorsal rami of L1, L2 and L3 spinal nerves. The dorsal rami of L4 and L5 do not have dorsal cutaneous branches (Bogduk 1983; Williams et al 1989b). Low-back pain cannot therefore be explained as being due to radiculopathy of L4 or L5; it could be regarded as pain referred to the L1 or L2 dermatomes. Some patients with low lumbar disc lesions complain of inguinal pain, the area of the L1 dermatome, and some of anterolateral thigh pain in the L2 dermatome (Takahashi, Takahashi and Moriya 1995). Very few patients have associated pain on the medial side of the thigh and leg, at the site of the L3 and L4 dermatomes.

We found that needle insertion produced pain which radiated to the low back and the buttock in some patients; these areas are innervated by the superior clunial nerves derived from the dorsal rami of the L2 spinal nerve. Other patients had referred pain in the anterolateral thigh, which is innervated by the lateral femoral cutaneous nerves derived from the ventral rami of the L2 spinal nerve. This explains the pain radiation to different areas.

Our results can be explained by the convergence projection theory (Ruch 1955). This is based on the fact that visceral and somatic afferent fibres have synapses to common posterior-horn cells (Cervero and Tattersall 1985; Ammons 1987; Cervero 1987; Yokota et al 1988). Pain caused by disease of abdominal organs may be perceived as if it comes from somatic tissues. One example is that pain during delivery is referred to the T10 to L1 dermatomes through 'sympathetic' hypogastric nerve fibres (Bonica 1984). Colicky low-back pain due to ureteric calculus is relieved by a thoracolumbar sympathetic block (MacLean, Carroll and Graves 1949). If discogenic pain is transmitted by sympathetic afferent fibres through the sinuvertebral nerves, this explains why some patients with lumbar radiculopathy have only leg pain, and also why low-back pain

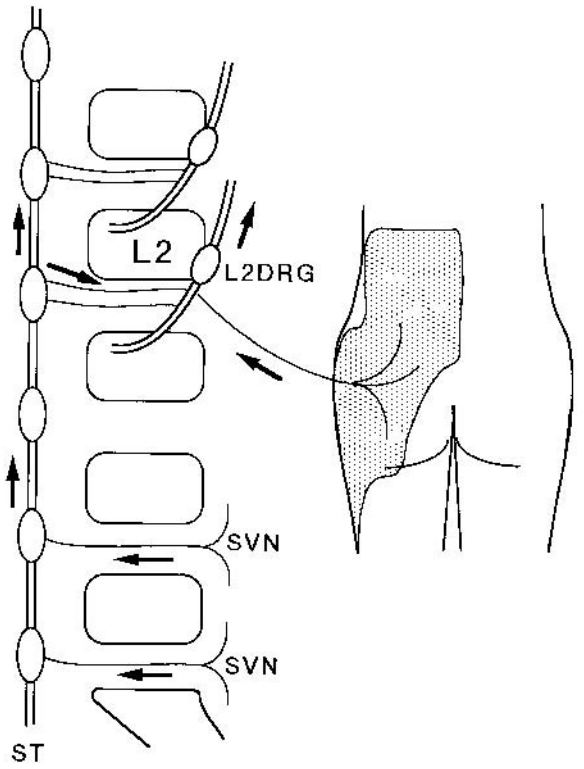

Fig. 5

Diagram to show the proposed afferent pathways of discogenic low-back pain. Pain from a lower lumbar disc is transmitted non-segmentally by visceral sympathetic afferent fibres, mainly from the L2 spinal nerve root. This results in referred pain in the L2 dermatome (DRG = dorsal root ganglion; SVN = sinuvertebral nerves; ST = sympathetic trunk).

can be produced by stimulation of an intervertebral disc even when the corresponding nerve root is blocked (Wiberg 1949).

Low-back pain is usually exacerbated in sustained sitting or forward bending of the trunk, both of which are known to increase intradiscal pressure (Nachemson 1976). Such pain is often relieved by lying on the side and gentle movement of the trunk; joint pain is usually made worse by such movement. Visceral pain can be induced by maintaining the intraluminal pressure of a hollow visceral organ above a certain pressure (Lipkin and Sleisenger 1957; Ness and Gebhart 1990); the latency from the onset of such a phasic stimulus to perception of pain by the patient is directly related to the intensity of the distending stimulus (Lipkin and Sleisenger 1957). We assume that the 'flexion test' which we used increased the intradiscal pressure and distended the outer layer of the annulus fibrosus. The reduction of this pain after infiltration implies that the low-back pain which we elicited originates mainly from the intervertebral discs.

Some of our patients reported bilateral relief of pain when hypoalgesia was restricted to the ipsilateral skin. We cannot fully explain this, but it may be due to anastomosis of the sinuvertebral nerves (Pedersen et al 1956; Kimmel 1961; Edgar and Ghadially 1976; Kojima et al 1990a; Nakamura et al 1996) or to integration of the sensation at higher levels (Ammons 1987). The persistence of tenderness between the interspinous processes after infiltration of the L2 nerve may indicate that the posterior elements have segmental innervation by the somatic spinal nerve (Bogduk 1983; Suseki et al 1996:in press).

Conclusions. We suggest that discogenic low-back pain is transmitted non-segmentally by visceral sympathetic afferents mainly through the L2 spinal nerve root, and that this may be perceived as referred pain in the L2 dermatome (Fig. 5). Discogenic low-back pain may be a type of visceral pain. Injection of the L2 spinal nerve root is a possible diagnostic tool and could be used for the conservative treatment of discogenic low-back pain.

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