

The Tissue Origin of Low Back Pain and Sciatica:

A Report of Pain Response to Tissue Stimulation During Operations on the Lumbar Spine Using Local Anesthesia

Stephen D. Kuslich, MD,* Cynthia L. Ulstrom, RN, MBA,† and Cami J. Michael, Physician Assistant†

Over the past decade we have had the opportunity to perform more than 700 operations on the lumbar spine while using local anesthesia. The patient response to the stimulation of various tissues differed somewhat, but we observed common patterns of pain referral. This preliminary report summarizes our findings in 193 consecutive patients studied prospectively. We draw certain conclusions about the likely tissue origin of back and leg pain. Sciatica could only be produced by stimulation of a swollen, stretched, or compressed nerve root. Back pain could be produced by stimulation of several lumbar tissues, but by far, the most common tissue of origin was the outer layer of the annulus fibrosus and posterior longitudinal ligament. Buttock pain could be produced by the simultaneous stimulation of the annulus and the nerve root. The facet joint capsule very rarely generated low back pain. The facet synovium and cartilage surfaces of the facet were never tender.

What tissues are involved in the generation of low back pain and sciatica? Experts disagree on the answer to this question. The problem is not purely academic, because the medical and economic resources applied to

this problem are significant. It seems obvious that we would be more precise in our management of the patient if we possessed a more accurate understanding of what tissues are responsible for the pain, and those that are not.

A voluminous and diverse literature exists on the subject. A majority of that literature, however, consists of what we call "secondary publications," that is, opinions derived by deduction from a much less voluminous "primary research base."

A great deal of the confusion and controversy about the tissue origin of low back and leg pain results from these secondary publications, wherein the pain is presumed to arise from this or that tissue based on one's perception of the presence or absence of appropriate "nociceptive nerve endings." To illustrate this point, we note the opinions of Wyke, a British neurologist, who concluded that the disc is not an important source of low back pain, basing his opinion on a belief that suitable nerve endings are not present in that tissue.¹⁸

What about the role of muscular tissue? It is assumed by many authorities that weak and strained muscles are a common source of lumbar pain.¹⁵ If this assumption is true, why

* Clinical Assistant Professor, Department of Orthopaedic Surgery, University of Minnesota; Private Practice, Metropolitan Orthopaedic Associates, Ltd.; and Orthopaedic Surgeon, Metropolitan Mount Sinai Medical Center, Minneapolis, Minnesota

† Metropolitan Orthopaedic Association, Ltd., Minneapolis, Minnesota

then do we see backache in many patients who have strong muscles, and why does low back pain last so much longer than other commonly overused and strained muscles? A few isolated cases of compartment syndrome have been published and microscopic atrophic changes have been observed in some patients with low back pain, but we find no published accounts of muscle tears and hematomas in patients operated on for low back pain. Most observers have found very little evidence of muscle pathology that cannot be easily attributed to disuse.

The facet joint is regarded as a common source of pain by several authors.¹² Patients have undergone "facet injections" or even "facet denervations" in an effort to reduce the pain. These procedures are rarely more than temporary and partially effective. Can synovial folds become "pinched," and if so, is this a painful condition?

The available clinical and neuroanatomical evidence indicates that there must be some relationship between these pain syndromes and the process of degenerative disc disease.^{4,10} The exact relationship, however, remains unclear.

Hirsch³ reported the results of an experiment in which he stimulated various lumbar tissues in awake patients by the use of carefully placed needles. He noticed that he could produce low back pain in many individuals by stimulation of the posterior portion of the annulus of the disc. Furthermore, he was able to eliminate the pain by the injection of a minute volume of local anesthetic into that tissue. In some cases where the disc puncture itself was painless, he was able to reproduce the pain by applying pressure within the disc by the injection of saline.

A very ingenious experiment was performed by Smythe and Wright.¹³ These authors placed nylon threads into various lumbar tissues while performing certain lumbar spinal operations. During the postoperative period, they pulled on the threads and asked the patients to describe the location of any pain produced. This study indicated that the annulus fibrosus was the most common site of low back pain, and that the compressed nerve root was responsible for sciatica. The study also demonstrated that tension placed on a normal nerve root resulted in no pain.¹³

Recently, the diagnostic test known as discography is enjoying a resurgence in popularity. Most agree that the test is not sufficiently accurate or reliable to define all of the painful tissues, but there is little doubt that stimula-

tion of the pathologic disc can induce pain syndromes that closely resemble the painful conditions seen in clinical practice.^{2,7}

In 1948, Falconer and associates¹ published their observations made during the exploration of a small number of lumbar spines under local anesthesia. Murphy reported similar results in his small series of surgical cases similarly treated.⁹ These authors concluded that the annulus and the nerve root are the tender tissues.

Spurling and Grantham stated in 1940 that: "We have repeatedly had patients who complained of pain in the back during operation with local anesthesia when the annulus fibrosus was manipulated."¹⁴

Similarly, Wiberg in 1950, operating on some 200 patients using local anesthesia of the skin and muscles only, reported that "in most subjects, firm pressure on the posterior surface of the vertebral body causes no pain . . . On the other hand, touching the disc itself caused pain of lumbosacral distribution in nearly all cases."¹⁷

Roffe subsequently performed elegant dissections of the region, and by means of special staining techniques, he was able to identify a rich nerve supply to the outer annulus as well as the posterior longitudinal ligament.¹¹ These nociceptive fibers are connected to the central nervous system via the sinovertebral nerve, which was described in detail as early as 1850 by von Luschka.¹⁶

The present article describes the methods and results of our recent study designed to further elucidate which tissues are responsible for low back pain and sciatica.

METHODS

In Vivo Study of Pain Production in the Human Lumbar Spine

This article summarizes the results in 193 consecutive patients during the period from 1987 to 1990. All patients underwent decompression operations for herniated disc or spinal stenosis. We used an anesthetic technique known as "progressive local anesthesia," wherein each consecutive tissue is infiltrated as the operation proceeds according to the individual need for pain control. All patients were placed in the kneeling position on the Heffington frame.

We stimulated each successive tissue by means of mechanical force using blunt surgical instruments or by the application of elec-

trical current at low voltage using the unipolar cautery. We used an operating microscope in all cases because of its advantages in terms of improved lighting, magnification, and safety. The technical details of this method of lumbar nerve root decompression are contained in our previous publication on the subject.⁵

Table 1 lists the tissues tested in this manner.

The patients were fully awake or only lightly sedated. During the course of the operation we stimulated each tissue and asked the patient to report any painful sensation. The anesthetist recorded the response on a special form (Fig. 1).

RESULTS

Table 2 details our findings by tissue. It also provides specific numbers and percentages for each tissue tested. The number of tissues tested does not always equal 193, because we did not stimulate every tissue in every case because of the inconvenience or impossibility of accomplishing that task in all instances. For example, we did not expose every case to a sufficient degree to visualize a normal nerve root. We recorded the severity of pain using an analog scale from 0 to 5. Significant pain was defined as that pain which was high in intensity and closely corresponded to the pain felt preoperatively. We emphasize that this technique is performed gently and is, for the most part, a benign and well-tolerated operation. The pain stimulation produces a short-lived discomfort that is easily relieved by discontinuing the tissue stimulation or by the local administration of a small volume of 1% xylocaine.

Figure 2 indicates the tissue origin of clinically significant low back and leg pain.

Table 1. Tissues Tested for Pain Sensitivity by Mechanical Stimulation

Skin	Epidural fat
Fat	Nerve root dura
Fascia	Nerve root
Supraspinous ligament	Compressed
Interspinous ligament	Uncompressed
Spinous process	Annulus fibrosus
Muscle	Central
Lamina	Centrolateral
Ligamentum flavum	Lateral
Facet capsule	Nucleus
Facet synovium	Vertebral end plate

Table 3 summarizes the findings in order of pain sensitivity.

DETAILED OBSERVATIONS MADE DURING THE OPERATIONS

Lumbar Fascia

The lumbar fascia consists of a glistening, white, and moderately tense fibrous tissue overlying the lumbar paravertebral musculature. In most cases, the fascia may be touched or even cut without anesthetic. In occasional cases, we were able to produce some level of low back pain by stimulation along the central tissue connecting the spinous processes. This tissue is the so-called "supraspinous ligament." We emphasize, however, that this occurred only in rare cases. In addition, traction or cautery directly at the location of blood vessels or nerves piercing the fascia sometimes produced a sharp and localized discomfort.

Muscles

Gentle pressure never produced pain. Forceful stretching at the base of the muscles, especially at the site of blood vessels or nerves, or at its attachment to bone, usually produced a localized low back pain. This pain varied with the amount of pressure and stretch applied. The pain was described as sharp and rarely simulated the deep, dull ache of lumbago. We were unable to observe any evidence of gross pathologic changes in the muscle, and concluded that the pain was probably derived from local vessels and nerves, rather than the muscle bundles themselves.

Normal Nerve Root

The normal, uncompressed, or unstretched nerve root was completely insensitive to pain. It could be handled and retracted without any anesthetic. Forceful retraction over an extended period of time resulted in mild paresthesias but never any significant pain.

Compressed Nerve Root

Stimulation of the compressed or stretched nerve root consistently produced the same sciatic distribution pain as the patient had ex-

Table 2. Findings of the 193-Patient Study

TISSUE	NO. TESTED	NO. AND % SOME PAIN	SIGNIFICANT PAIN	ANATOMIC SITE OF PAIN
Lumbar fascia	193	32 (17%)	0.5%	Back
Paravertebral muscle	193	80 (41%)	0.0%	Back
Suraspinous ligament	193	49 (25%)	0.0%	Back
Spinous process	193	21 (11%)	0.0%	Back
Interspinous ligament	157	10 (6%)	0.5%	Back
Lamina	192	57 (30%)	0.0%	Back, buttock (rare)
Facet capsule	186	0 (0%)	0%	
Facet synovium	167	0 (0%)	0%	
Ligamentum flavum	193	1 (0.5%)	0%	Back
Epidural fat	92	21 (23%)	6%	Buttock, leg
Posterior dura	64	15 (23%)	5%	Back, buttock
Anterior dura	167	166 (99%)	90%	Buttock, leg, and foot
Compressed nerve root	55	6 (11%)	9%	Buttock, leg
Normal nerve root	183	135 (74%)	15%	Back
Central annulus	144	102 (71%)	30%	Back
Central lateral annulus	176	0 (0%)	0%	
Nucleus	109	67 (61%)	9%	Back
Vertebral end plate				

perienced preoperatively. In spite of all that has been written about other tissues in the spine causing leg pain, we were never able to reproduce the patient's sciatica except by finding and stimulating a stretched, compressed, or swollen nerve root. Sciatica could be produced by either pressure or stretch on the caudal dura, on the nerve root sleeve, on the ganglion, or on the nerve distal to the ganglion, depending on the site of compression. The ganglion was somewhat more tender than other parts of the nerve root, but the difference was not dramatic. In general, the closer one stimulated to the site of compression or tension, the greater the pain response. This pain could always be eliminated by the injection of 0.5 cm³ of 1% xylocaine via a 30-gauge needle beneath the nerve sleeve proximal to the site of compression.

Another interesting finding involved operations on patients who had undergone prior laminectomies. In those cases, there was always some degree of perineurial fibrosis. The scar tissue itself was never tender. The nerve root, however, was frequently very sensitive. In addition, we concluded that the presence of scar tissue compounded the nerve pain by fixing the nerve in one position and thus increasing the susceptibility of the nerve root to tension or compression.

Table 3. Findings by Order of Pain Production

Always	Skin
	Compressed nerve root
Often	Outer annulus fibrosus
	Vertebral end plate
	Tissues in anterior epidural space, i.e., anterior dura and posterior longitudinal ligament
Rare	Supra- and interspinous ligament
	Facet capsule
	Muscle attachment at bone or neurovascular bundle
Never	Ligamentum flavum
	Lumbar fascia (except at site of vessels)
	Lamina bone
	Spinous process bone
	Facet synovium
	Uncompressed nerve root (unless great stress is applied)
	Uninflamed dura
	Facet cartilage

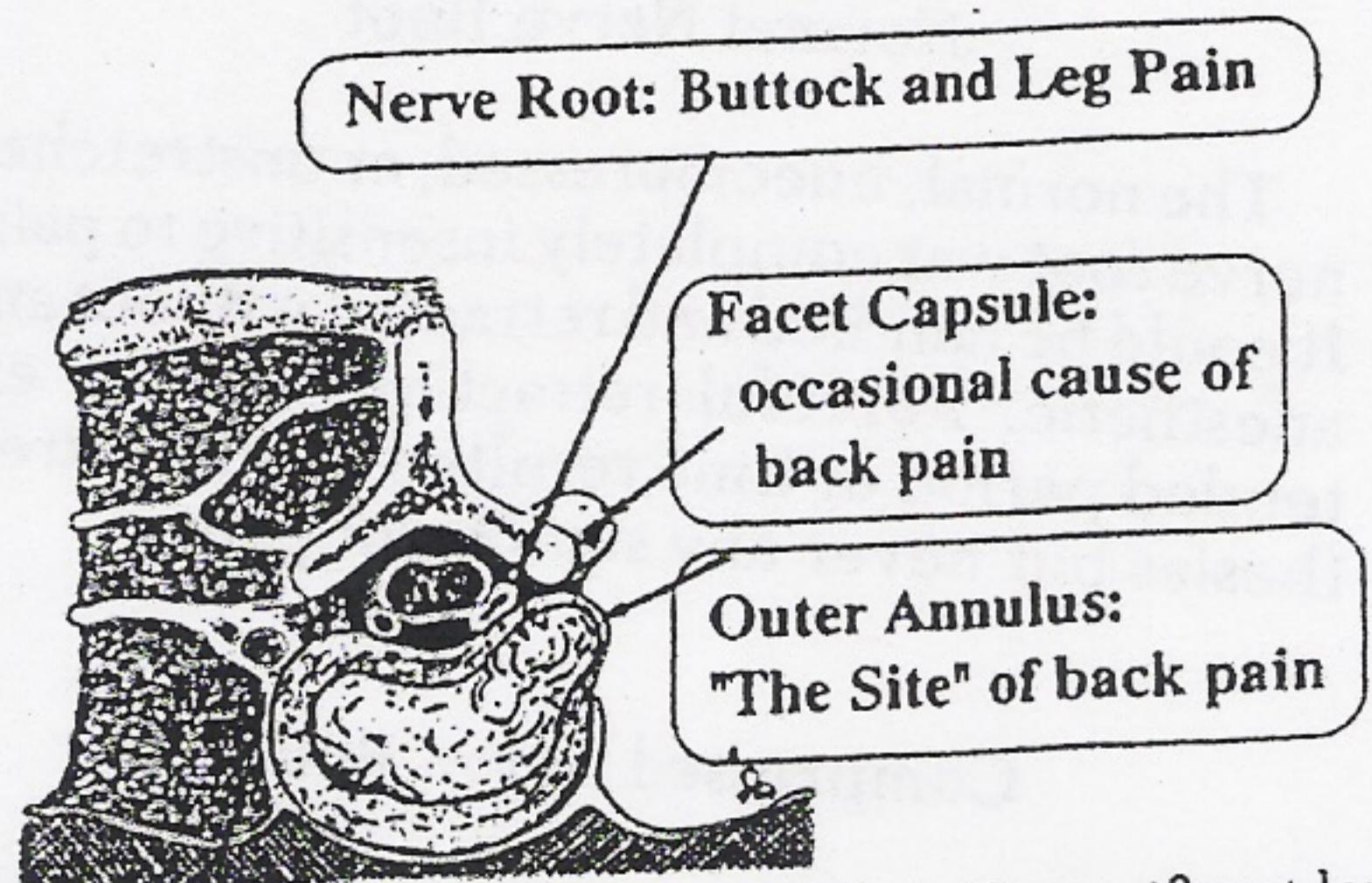


Figure 2. The tissue origin of clinically significant low back and leg pain.

Annulus Fibrosus

About two thirds of patients responded with pain at this site. In those patients, stimulation always produced back pain that was similar to the low back pain suffered preoperatively. Likewise, the application of local anesthesia obliterated the pain. We had more difficulty producing buttock pain, but sometimes we were able to produce it by the application of pressure on the nerve root and outer annulus simultaneously. Otherwise, the reproduction of true, clinically significant buttock pain was rarely possible by the stimulation of any other tissue. Stimulation of a disc hernia lateral to the foramen sometimes resulted in the sensation of buttock pain.

The annulus was exquisitely tender in about one third of cases, moderately tender in one third, and insensitive in the remaining one third operated for herniated disc or stenosis. Perhaps certain individuals are more richly innervated than others. Or, alternatively, perhaps there exists some chemical or mechanical irritant that sensitizes certain discs to become painful. Our observations did not clarify this point. They did, however, provide an explanation for the observations made by other authors that some individuals with disc protrusion are asymptomatic, while other patients are acutely tender.

Referral of pain depended upon the exact site of the annulus being stimulated. The central annulus and posterior longitudinal ligament produced central back pain. Stimulation to the right or left of center of the posterior longitudinal ligament directed pain to the side of the back being stimulated. This observation correlates with the finding of back pain on one side or the other when a "bulge" is noted on CT scan on that side of the midline.

Posterior Longitudinal Ligament

We observed that the posterior longitudinal ligament was intimately connected with the posterior, central portion of the annulus. It was frequently tender and produced central low back pain. Because of its close proximity to the annulus, we were not able to differentiate its specific role as well as we would have preferred. In general, when the posterior annulus was tender, the posterior longitudinal ligament was also sensitive.

Vertebral End Plate

Pressure or curettage of the vertebral end plate frequently resulted in a deep, rather severe low back pain. It was usually more severe and sharper in quality than the preoperative discomfort.

Facet Joint

The tissues around the facet capsule were sometimes sensitive to the forceful stimulation by means of a needle or a Cobb elevator during efforts to mobilize the paravertebral muscle. The pain, however, was described as sharp and localized to that region. Its quality did not match the preoperatively perceived deep, dull pain of clinical low back pain syndrome. The capsule was sometimes tender, but when it was, it referred pain to the back, or very rarely the buttock—never the leg. It could always be blocked by a few cubic centimeters of local anesthetic injected around the facet. It was never necessary to inject into the joint itself. The facet synovium was never sensitive. The facet articular cartilage was never tender.

One further observation regarding the facet joint may be of interest to the reader. In those cases where a trefoil-shaped central canal produced a tight lateral recess, we observed that the undersurface of the superior articular facet and its joint capsule frequently came into intimate contact with the posterior surface of the disc. We were able to produce low back pain by applying pressure to the disc at this site in many cases. Could it be that repeated contact between the superior facet and the disc causes an irritation that the patient interprets as low back pain and the physician interprets as facet syndrome? We offer this as another possible cause of low back pain and a reason for the effectiveness of facet injection in certain cases of low back pain.

Other Tissues

The ligamentum flavum, epidural fat, posterior dura, nucleus, lamina, and spinous processes were insensitive to local mechanical stimulation. Forceful stretch of the interspinous ligament occasionally produced localized central low back pain. The surface of bone, even at the level of the periosteum, was insensitive. The spinous processes, laminae,

and facet bone could be removed with a rongeur without anesthetic. We did not test for deep bone pain by means of increasing the marrow hydrostatic pressure.

CONCLUSIONS

Operative exploration of the lumbar spine using progressive local anesthetic provides a unique opportunity for the surgeon to learn about the tissues that are responsible for the pain syndromes that are seen in clinical practice. The spinal surgeon has the opportunity to define the true origin of pain *in vivo*. Sciatica can only be produced by direct pressure or stretch on the inflamed, stretched, or compressed nerve root. No other tissues in the spine are capable of producing leg pain.

Proximal infiltration of the nerve using a 30-gauge needle and 0.5 cm³ of 1% xylocaine completely relieved the sciatica and allowed for painless retraction of the nerve root.

The outer annulus is the tissue of origin in most cases of low back pain; the facet synovium is never the site of back or leg pain. The facet capsule is sometimes tender, but its true significance in the area of low back pain and sciatica probably involves its ability to compress or irritate other sensitive local tissues, i.e., the nerve root or the outer annulus. The nucleus is never tender.

In spite of all that has been written about muscles, fascia, and bone as a source of pain, these tissues are really quite insensitive. These observations cast doubt on the effectiveness of several commonly used forms of therapy for spinal pain, e.g., massage, ultrasound, electrical stimulators, exercises, magnets, toe-tickling, manipulation, muscle relaxants, anti-inflammatory medicines, psychotherapy, and even some surgical procedures. Perhaps we should be spending more time learning how to effectively treat the true sources of spinal pain, and less time massaging, manipulating, heating, and cooling tissues that have little to do with the production of low back pain and sciatica.

Based on the evidence above, we may find a really effective treatment for low back pain and sciatica when we learn how to decompress a nerve atraumatically and, at the same sitting, treat the painful disc by stabilizing the motion segment atraumatically. Such a procedure does not currently exist, although we

and others are in the process of developing the technology.

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Address reprint requests to
Stephen D. Kuslich, MD
825 South 8th Street, Suite 550
Minneapolis, MN 55404