

### Relation of Foraminal (Lateral) Stenosis to Radicular Pain

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In their article in this issue of *AJNR*, Nowicki and coauthors (1) take as their point of departure the embarrassing fact that we are quite poor at relating the sensory and motor symptoms of spinal stenosis to the radiologic picture of the spinal canal and its foramina. They quote figures of 30% false negatives (ie, pain with no obvious disease), and 20% false positives (ie, asymptomatic disease). We suspect that these numbers are overly optimistic for most clinical settings. The situation is no better for pain associated with intervertebral disk herniation. Why are we so inaccurate at diagnosing back pain in the radiology suite? Are there more informative ways to image the spine?

Clinical-radiopathologic correlation is generally carried out with the patient recumbent and at rest. In daily life, however, people are constantly in motion. It is well known that in patients with lumbar spinal stenosis, postural factors and load bearing can elicit and increase pain. With this in mind, the authors carried out a detailed combined radiologic and histologic study of spinal roots and their foramina under conditions of precisely controlled spinal loads. Observations were made of isolated cadaveric "motion segments," tissue units made up of two adjacent spinal segments and the intervening disk, joint capsule, and nerves. Calibrated, physiologically relevant loading forces were applied, and effects on the spatial relation between the nerve roots and the disk/ligament were monitored.

The authors refer to nerve root compression seen only under load deformation as "dynamic stenosis," and point out that such compression is hidden (occult) under normal imaging conditions. The bottom line of their study is that in terms of radiologic diagnosis, things might be even worse than we thought! If we were to mon-

itor dynamic stenosis routinely, the percentage of cases calling for a diagnosis of functionally significant lateral stenosis would be far higher than at present, based on classic recumbent resting images. For example, in spines with "normal" aging signs, such occult stenosis occurs in about half of all individual foramina sampled from T-12 to S-1. Moreover, it is universal (100%) in persons with advanced disk degeneration.

Had these data been collected from a population of patients with severe back pain, we might have expected this sky-high incidence of radiologically defined disease. But they weren't. Spines of patients with diagnosed or suspected spinal disease, or with significant spinal radicular symptoms, were specifically excluded from the investigation. These were mostly healthy folks. A major weakness of the study is that premonitory symptoms were not specifically provided. Nonetheless, the message is clear that *the observed high incidence of stenosis with movement is characteristic of healthy, largely asymptomatic persons*. Imagine that we could routinely image the spines of our patients under conditions of physiologic loading comparable to those used by Nowicki et al in their cadavers. Factoring in occult stenosis along with classically evident stenosis, there would no doubt be fewer instances of false-negative diagnoses. The cost, however, would be a greatly inflated number of false positives. More than ever before we are left with twin dilemmas: (a) Why does spinal nerve compression cause radicular symptoms? and (b) Why does it so often *not* cause symptoms?

To gain some insight into these questions it is important to consider a few fundamental and, on the face of it, trivial, facts. First, stenosis causes pain only to the extent that it causes

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**Index terms:** Commentaries; Foramina, vertebral; Nerves, spinal; Spine, anatomy; Spine, stenosis

nerve fibers to fire electrical impulses. There is a widespread belief that disk herniation floods the local tissue with inflammatory exudates that sensitize and activate the endings of nociceptors in the local muscle, tendons, and fascia. We doubt that this is the major pain mechanism in disk disease (2), and consider it even less likely to be the key factor in pain of lateral stenosis. What is the putative source of inflammatory mediators in lateral stenosis? And why does the pain usually have a radicular distribution, and often feel like an electric shock, rather than being restricted to the region of the stenosis, and feeling like inflammation?

The obvious conclusion is that stenosis causes the activation of nerve fibers that are in transit through the spinal nerve on their way to the leg. That is, radicular pain is referred pain. But this raises a second problem. The graded pressure that lateral (foraminal) stenosis can apply to the nerve, particularly in the case of dynamic stenosis, does not normally evoke nerve impulses. Try the following experiment on yourself: Press with your right index finger over your median nerve in the left forearm. You undoubtedly felt the pressure of your fingertip locally. There are cutaneous nerve ending here. However, unless you have a median nerve neuroma, you probably did not feel any radiation of pain into your medial hand, the sensory distribution of the median nerve. The point is that spinal stenosis, and particularly the sort of dynamic spinal stenosis studied by Nowicki et al, is not, in itself, expected to cause referred pain symptoms. By this line of reasoning the huge incidence of asymptomatic stenosis is no surprise. What we need to know is why stenosis sometimes *is* painful.

There are two circumstances in which graded mechanical pressure on midnerve axons can trigger impulses. One is when there has been chronic local trauma to the nerve resulting in either severance of some or all of its axons (neuroma-in-continuity or nerve-end neuroma) or segmental demyelination. Under these conditions a change occurs in the local electrical properties of the axon membrane, rendering it hyperexcitable, with electrogenic behavior more like a sensory ending than a midnerve axon (3). A likely example is trigeminal neuralgia, where microvascular compression appears to be the culprit in producing a local region of paroxysmal neuronal hyperexcitability (4). In principle, stenosis can also cause this change,

although it need not always do so. There appear to be a number of potential sources of variability, including the genetic predisposition of the individual patient (5, 6).

The second circumstance that can render foraminal stenosis symptomatic is impaction of the dorsal root ganglion. The dorsal root ganglion is the region at the junction of the nerve root and the spinal nerve, within the spinal foramen, where the cell bodies of the primary sensory neurons reside. Because of the presence of the cell bodies in the dorsal root ganglion, this region has different electrical properties than the rest of the nerve. Specifically, graded pressure here frequently evokes action potentials, and hence is able to trigger radicular referred sensations (7, 8). Dynamic stenosis may impact the dorsal root ganglion directly. It may also facilitate the transmission of compressive, tensile, and torsion forces to the dorsal root ganglion by altering local hydrodynamic properties of the tissue (9), or by affecting the relation between the dorsal root ganglion and the local connective tissue that anchors it within the foramen. Radiating radicular pain on straight-leg raising (the Lassegue sign) is a likely example of this.

Unfortunately, available imaging techniques still do not permit us to map the degree of local electrical excitability along the length of a nerve. Our prediction is that if we could, the symptomatic lateral stenoses would be the ones in which various factors have conspired to render the spinal root axons and/or their dorsal root ganglia electrically hyperexcitable. By the same token, we suspect that by focusing on the neural processes responsible for hyperexcitability (10), and by more thoughtful use of membrane stabilizing drugs (11, 12), it might be possible to develop more effective avenues for relief of symptoms in the future.

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