Upper Lumbar Radiculopathy –
A Seldom Detected Cause Of Back Pain

C Chan Gunn, Deirdre Byrne, Hubert Chao, Lynn Chapman, Allan Lam, Man Kon Leung, Joanne McBrinn, Kathy Wong, and Kam Yan.

Institute for the Study and Treatment of Pain, Vancouver, BC

Editorial Note. This paper reintroduces the senior author’s definition of peripheral neuropathy and radiculopathy in relation to spondylosis and trauma which allows an interpretation of many features of neck and back pain which are often not recognized or described as non-specific. The signs which lead the authors to diagnose neuropathy and radiculopathy are local autonomic changes, trophedema, trophic changes and localized motor changes: these in turn are usually accompanied by secondary muscle shortening. Dry needle exploration is used as a further exploratory and confirmatory technique to identify this pattern.

The authors now present a series of cases of low back pain which failed to respond to conventional therapies: the features of these cases, using the specialized examination techniques, lead to the hypothesis that upper lumbar dysfunction, accompanied by features of neuropathy or radiculopathy, can be responsible for cases of persistent low back pain.

Abstract

The authors identify localized neuropathy in the back (radiculopathy) by clinical features of autonomic dysfunction, trophedema, and trophic and motor changes, followed by dry needling exploration.

They made a retrospective study 119 patients with low back pain who had tried physical therapies without success.

Every patient revealed neuropathic/radiculopathic signs. In 83 these were more prominent in the upper lumbar spine. Of these, 47 also had low back findings. All were treated by dry needling and were given an average of 9.3 treatments.

We propose that back pain remains a greater problem than it needs to be, because neuropathic features are not generally recognized. This series shows that signs of upper lumbar radiculopathy are likely to be present in cases who have failed standard therapy. Treatment to achieve desensitization by stimulation appears to give good results.

Key Words: back-pain, radiculopathy, spondylosis, muscle shortening

Introduction:

Chronic low-back pain continues to be a diagnostic and therapeutic challenge, because of the difficulty of arriving at an exact diagnosis. It is generally believed that only 10% - 15% of patients get a specific diagnosis.¹ Low back pain (LBP) can be a deceptive term, as the problem may be present at a higher level in the spine. The morbidity of back pain has been blamed on a wide spectrum of soft tissue damage, ranging from minor ligament strain, to major disruptions of stabilizing tissues of the spine. Although a majority of patients recover spontaneously, even without treatment, a proportion develop chronic symptoms.

We report the results of our specialized techniques of clinical observation. Whilst the L5 – S1 segmental level is generally regarded as the most troublesome one for pain felt in the low back, we have found that in this series of patients, signs of upper lumbar radiculopathy (L1-2, L2-3) were more frequent.

We describe below the rationale for our method of clinical examination and the dry needle ‘imaging’ technique we use to confirm the diagnosis.

Physical examination for signs of neuropathy

We diagnose radiculopathy almost entirely on specific clinical signs. The history typically gives little assistance, as reported pain so often appears to exceed that consistent with any reported injury. The basic back examination (comprising lumbaroscal movements, tenderness, tension tests and standard neurology of the lower limbs) has severe limitations as it often fails to find evidence of injury or inflammation. In contrast, our method of examination which searches for signs of peripheral neuropathy almost invariably uncovers sensory, motor, autonomic and trophic signs.²

Peripheral neuropathy may be defined as a disease that causes disordered function in the peripheral nerve. Although sometimes associated with structural changes in the nerve, a neuropathic nerve can appear deceptively normal: it still conducts nerve impulses, synthesizes and releases transmitted substances and evokes action potentials and muscle contraction.³
Peripheral neuropathy is not exceptional. Of the innumerable causes of nerve damage, such as trauma, metabolic, toxic and others, we believe that attrition from spondylosis is by far the most common. The spinal nerve root is prone to injury from pressure, stretch, angulation, and friction. Ordinarily, spondylosis follows a gradual, relapsing and remitting course that is silent until symptoms are precipitated by an incident often so minor that it may pass unnoticed. All gradations of spondylosis can exist, but early spondylotic changes can give rise to radiculopathy. 5,6

Significance of peripheral neuropathy
The proper function of innervated structures depends on the flow of impulses through the intact nerve to provide a regulatory or "trophic" effect. All structures (including skeletal muscle, smooth muscle, spinal neurons, sympathetic ganglia, adrenal glands, sweat glands, and brain cells), when deprived of excitatory input, develop supersensitivity. Supersensitive structures over-react to many forms of input, chemical and physical.

Damaged primary afferent fibers demonstrate three electrophysiologic features according to a fundamental physiologic law - Cannon and Rosenblueth's Law of Denervation.5: (a) spontaneous activity: supersensitive muscle cells can generate spontaneous electrical impulses that trigger false pain signals or provoke involuntary muscle activity; (b) exaggerated response to stimulus: denervated nerves are prone to accept contacts from other types of nerves including autonomic and sensory; and (c) increased sensitivity to chemical transmitters.

The features of neuropathic pain are well known and include:

• Pain in the absence of tissue-damage.
• Delay in onset after precipitating incident.
• Abnormal sensations such as "burning" pain (dysesthesia), or "deep, aching" pain.
• Paroxysmal brief "shooting or stabbing" pain.
• A mild stimulus can cause extreme pain (alldynia - pain from stimuli which are not normally painful), eg tenderness to digital pressure; multiple tender points are sometimes called 'fibromyalgia'.
• Marked summation and after-reaction following repetitive stimuli.
• Loss of joint range caused by muscle shortening.

Signs of neuropathy
(i) Autonomic dysfunction - vasomotor, sudomotor and pilomotor. Vasoconstriction differentiates neuropathic pain from inflammatory pain: in neuropathic pain, affected parts are perceptibly colder (as seen at times in complex regional pain syndrome). There may be increased sudomotor activity (hyperhidrosis), and the pilomotor reflex is frequently hyperactive in affected dermatomes as "goose-bumps" (Figure 1). 2

(ii) Trophedema – Subcutaneous tissue edema, also called neurogenic edema or trophedema is part of the tissue response to injury. It is likely to be a neuro-inflammatory reaction at the site of nerve injury, and to represent an early stage of host defense and tissue repair. 1,11

Figure 1: Pilomotor reflex on right buttock with muscle shortening in gluteus maximus

Figure 2: Upper Lumbar Trophedema with Peau d'orange appearance

Inflammation causes the permeability of small blood vessels around the injured area to increase; allowing large amounts of fluid and plasma protein to escape into the extravascular space. The exudate is not a static puddle, but a high-turnover pool of fluid passing from blood vessels to lymph drainage. While small molecules are able to rapidly diffuse across the vascular wall, larger plasma protein can
An important finding is the presence of knots in deep paraspinal muscles (semispinals, multifidus, and rotatores). These knots often become tender, with overlying trophedema, and identify an irritable segment that requires treatment.

Dry needle exploration as a powerful imaging and treatment tool
Having arrived at a clinical impression, we confirm our diagnosis with a localising technique that we have developed and refined over the past 25 years. We use a dry-needle to probe and explore muscles for its texture and any muscle shortening caused by contracture. We have found that our method of examination and diagnosis is particularly useful in the upper lumbar back, an area often inadequately scrutinized in a conventional back examination.

It is important to realize that chronic back pain is not simply nociception: the Deqi cramp-like sensations which are reproduced by the dry needling technique are associated with receptors that sense muscle shortening: proprioceptors. The needle stretches muscle spindles and produces a monosynaptic, myotatic stretch reflex.

Material and Methods
We reviewed retrospectively a series of patients who had presented to our outpatient clinic for low back pain over a period of 18 months. We included only those patients who had tried other forms of physical therapy without success. We did not exclude those with other musculoskeletal complaints.

Techniques of examination
The spine and adjacent tissues were examined for features of autonomic dysfunction, trophedema, trophic and motor changes as described in the introduction.

The Fabere Test. With the patient supine, the heel of the painful side is placed on the knee of the opposite leg. The examiner takes the thigh of the painful side and passively moves it at the hip joint into a combination of flexion, abduction and external rotation (Figure 4). If the knee on the affected side remains elevated compared to the other side, and cannot be depressed, either the hip joint is abnormal or there is a functional or other muscular cause of restriction to full flexion, abduction, external rotation and/or extension; this highlights the adductors and internal rotators (Table 1).

Dry needling localization. We employ a fine, flexible, acupuncture needle (6 centimeters in length, 0.35 mm diameter) in a plunger-type needle holder as a diagnostic tool.

Deep contractures can only be discovered by probing with a needle as they are beyond the finger's palpation reach. The fine, flexible needle transmits feedback information on the nature and consistency of the tissues that it is penetrating. When it penetrates a normal, relaxed muscle, the needle
meets with little resistance like piercing butter. The patient reports little or no pain. When the needle enters fibrotic tissue, there is a grating sensation like cutting through a pear. A muscle in contracture also resists penetration. Sometimes the resistance is so intense that it can be mistaken for bone. Frequent small pecking motions with the needle may then be required to release the contracture to allow penetration.

A neuropathic muscle is highly irritable and needle penetration can cause it to contract and grasp the needle. When the needle penetrates a contracture, the patient may report a cramplike sensation. The intensity of the cramp parallels the severity of muscle shortening: it can be excruciatingly painful, but gradually resolves as shortening eases. The peculiar, inimitable, grabbing sensation referred to by acupuncturists as the "Deqi response" is, we believe, unique to and diagnostic of neuropathy. The grab can be so intense that it resists withdrawal of the needle until muscle shortening is released.

An irritable spondylotic level can be located in minutes. The experienced examiner, guided by the needle-grasp and the Deqi response, is able to identify and pinpoint a 'distressed' segment quickly.

All patients were treated by dry needling.

Follow-up was for a minimum of 6 months.

### Results

During the 18 month period, there were 119 patients whom we included in this retrospective review. Ages ranged from 27 to 88 years. (see Table 2). 112 had back pain for more than 9 weeks, and 7 for less than 9 weeks.

<table>
<thead>
<tr>
<th>AGES</th>
<th>TOTAL PER AGE</th>
<th>FEMALE</th>
<th>MALE</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 30 years old</td>
<td>F + M = 4</td>
<td>F = 2</td>
<td>M = 2</td>
</tr>
<tr>
<td>30 - 39 years</td>
<td>F + M = 15</td>
<td>F = 8</td>
<td>M = 7</td>
</tr>
<tr>
<td>40 - 49 years</td>
<td>F + M = 23</td>
<td>F = 13</td>
<td>M = 10</td>
</tr>
<tr>
<td>50 - 59 years</td>
<td>F + M = 24</td>
<td>F = 11</td>
<td>M = 13</td>
</tr>
<tr>
<td>&gt; 60 years</td>
<td>F + M = 53</td>
<td>F = 39</td>
<td>M = 14</td>
</tr>
</tbody>
</table>

Table 2. Patients in the study comprised 73 women and 46 men

All had musculoskeletal complaints related to the spine, and many had symptoms and/or physical signs at more than one segmental level. These patients had, in addition to their low back pain, one or more of the following: neck pain, shoulder and arm pain, dorsal back pain, and pain extending to the leg.

We found signs of radiculopathy (that is, neuropathy at root level) in virtually every patient. We found that, in 83 out of 119 patients, the signs were most prominent at the upper lumbar spine; 47 of these also presented with L5 - S1 findings. In the deep paraspinal muscles, knots were most commonly found at the L1 - L2 levels.

The Fabere test was positive in 83 out of 119 patients.

The dry needling treatment was continued until an adequate response was obtained. All the patients responded, and the average number of treatments was 9.3. In our study,

### Discussion

We have found that back pain is not so much of a diagnostic challenge, using our specialized examination and treatment techniques; we have found that the recognition of neuropathic features has led to effective therapy. In this study, patients with radiculopathic pain were diagnosed by clinical examination and they responded to dry needling.

Importantly, we believe, we identified that 69% had abnormalities at upper lumbar levels. We used the Fabere
test to demonstrate shortening in L2 muscles especially the pectineus, adductor longus, adductor brevis and adductor magnus muscles. Muscle shortening occurs in myotomes. We therefore believe that upper lumbar radiculopathy is, in isolation or conjunction, a common but seldom detected cause of back pain.

Without a firm diagnosis, low back pain may be blamed on psycho-physiological factors, or even malingering: a patient with genuine discomfort may not be treated appropriately, simply because there are no “significant physical findings”.

Additional diagnostic investigations such as CT scans, MRI or electromyography are designed to find structural changes, but they often fail to uncover subtle functional irregularities. The dry needling technique identifies the functional changes which are part of the painful segment, whereas this is something which X-rays, scans, or MRIs cannot identify. Indeed, radiological findings can be misleading as they cannot distinguish an old, inactive structural lesion from an ongoing, irritable one.

Our emphasis on radiculopathy is not without reason: with an acute injury to a healthy nerve, there is no prolonged discharge of pain signals, whereas the same injury to a neuropathic nerve can cause a sustained discharge: for pain to become persistent, the fibers must be previously affected. That is why some people develop severe pain after an apparently minor injury, and why that pain can continue beyond a “reasonable” period.

Pre-spondylosis and the nascent radiculopathy model were first presented in Spine in 1978. The model enables many apparently dissimilar musculoskeletal pain syndromes to be grouped under one etiologic classification (radiculopathy). It also introduced the concept of “neuropathic pain”.

Our examination for neuropathic signs and dry-needling treatment (Intramuscular Stimulation, or IMS) have been reported in detail in Spine.

We are pleased with the results of our dry needling techniques and intramuscular stimulation. Because muscle pain eases concurrently with the release of the needle grasp, patients soon become aware of the importance of relieving muscle shortening during treatment.

There are benefits from needle therapy not obtained from other local modalities - stimulation from the current of injury produced by needle penetration can last for several days until the miniature wounds heal. Also, it delivers to the injured area the platelet-derived growth factor (PDGF), which promotes healing by inducing mitosis and stimulating collagen formation.

The study could not be double-blinded for technical reasons. We were, however, able to recognize the condition by its characteristic physical signs and then assess progress by their amelioration. Significantly, patients were able to return to gainful employment.

Since IMS needling primarily elicits segmental spinal reflexes, the placebo effect is not evoked. IMS has since been taught to thousands of physicians and therapists (See www.iSTOP.org).

Authors
C Chan Gunn, CM, OBC, MD,
President, Institute for the Study and Treatment of Pain (iSTOP), Vancouver, BC and Clinical Professor, Multidisciplinary Pain Center, University of Washington, Seattle, USA
Deirdre Byrne, BSc (Hons) Physiotherapy, CGIMS*
Hubert Chao, MD, CGIMS
Lynn Chapman, BScR, MCPA, FCAMT, GCIMS
Allan Lam, MD, CGIMS
Man Kon Leung, PhD, MD, BA, CGIMS
Joanne McBrinn, BSR, MCSP, CAFCI, CGIMS
Kathy Wong, BScPT, MCSP, CAFCI, CGIMS
Kam Yan, BA Physiotherapy, CGIMS
* Certified Gunn IMS Practitioner

Address for correspondence
Institute for the Study and Treatment of Pain (iSTOP)
5655 Cambie Street
Vancouver, BC
V5Z 3A4 Canada
Tel (604) 264-7867
istop@istop.org
www.istop.org

Note
This study was first mentioned in the form of a letter to the Editor of the Journal of Musculoskeletal Pain, 2007; 15(4):103-5

References
1. DeLeo JA, Yezienski, RP: The role of neuroinflammation and neuroimmune activation in persistent pain, Elsevier Science BV, 2000
8. Wooll CJI: The Pathophysiology of peripheral neuropathic pain - abnormal peripheral input and abnormal central processing.