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Differentially Diagnosing Mechanical Low Back Pain

A Four Case Report

End Range Loading

This is the fourth case report paper I've written for 'Massage Matters' (Summer 2010, Fall 2010, Summer 2011). I refer you to these previous editions for an in depth explanation of non-vascular edema.

My hypothesis is as follows: increased pressure at a nerve will curtail its arterial blood supply. The nerve is now compromised and will not function properly.

The pressure may be caused by a boney stenosis as seen in neurogenic claudication or it may be caused by soft tissue encroachment as seen in a bulging disc. The increased hydrostatic pressure most often seen is that due to an osmotic gradient caused by a high concentration of macromolecules in the extra cellular matrix. This increased hydrostatic pressure due to the diffusion of water will be discussed below

The ischemic nerve will have an effect on the tissues it innervates. Here are two examples. The efferent vasomotor function could be lost and therefore blood isn't flowing into the tissues. This can be seen in chronic tendinopathies. The afferent proprioceptive function is lost and therefore a particular joint will be prone to injury. This can be seen in repeated ankle inversion sprains.

As a quick review of increased hydrostatic pressure I will explain the common pathophysiology. According to Blaise Pascal's principle, all fluid flows from a higher pressure if pathways of egression exist. Obviously, arterial blood also flows from a higher pressure to a lower pressure. The contracting ventricle raises the pressure within the chamber and so, blood flows into the aorta and circulates. When nerves, connective tissue and smooth muscle cells become stressed, they secrete glycoproteins and proteoglycans into the extracellular matrix. These large molecules are hydrophilic and can absorb fifty times their own weight in water molecules. In some chronic conditions the concentration of proteoglycans can be forty times greater than the normal amount; water then diffuses into these areas of high concentration, otherwise known as osmosis. Space is limited therefore, the hydrostatic pressure increases. This new cellular pressure approaches that of the systolic pressure in the capillaries. Microcirculation now slows or stops.

Fortunately this process is reversible: nerves, and their associated connective tissue, are visco-elastic and can be stretched. As the therapist stretches the nerve, local pressure within increases resulting in a new therapeutic pressure that is even greater than that caused by proteoglycans. Again, by applying Pascal's principal, we understand that the fluid now moves out of the area. After the treatment, the resting pressure is more physiologically normal and regular blood flow returns.

The patient presentation will depend on what nerves are affected. Since nerves exist all through the body, the resulting conditions will vary. Consider for a moment the peripheral nervous system including ventral and dorsal primary rami, the autonomics, the cranial nerves, and those formed from neurogenesis; this system supplies tissues from the skin to the internal organs, and everything in between. Many conditions come to mind. Consider next the nerve roots and the cauda equina and finally, the central nervous system.

Most of the following procedures are held for five to ten seconds and repeated five or six times. This method serves two purposes: enough time is allotted for the fluid to shift locations giving the patient enough time to respond before the therapist moves forward.

To differentially assess and treat chronic lower back pain using this model, one must look at things from a fresh viewpoint: What we are concerned with is the lack of oxygen to the nerves and therefore determining firstly, which nerves are involved and secondly, how to load them. This viewpoint does not always coincide with more classical, or well known, diagnoses and treatments.

Here, I will include a short review of local neurology. Those nerves located proximal to the intervertebral foramen include the cauda equina, nerve roots, sinuvertebral nerves, nervi nervorum, and interstitial nerves formed from neurogenesis. After exiting the foramen, the nerve roots become the spinal nerve which will then divide. Both primary dorsal and ventral rami are found here, as well as the sympathetics, nervi nervorum and interstitial nerves. The ventral rami form the plexi and the rami communicantes; the dorsal branches then travel to the posterior aspects of the low back. Structures which are innervated include: discs, bones, facets, meninges, ligaments, blood vessels, joint capsuls, tendons, muscles, fascia, skin, and the nerves themselves.

Lack of circulation to any of these nerves can give rise to buttock and lumbar pain. This statement raises, again, certain key questions. Which nerves are involved in a particular case and, once found, how do we load them?

Epidemiology has given us more understanding regarding the generators of chronic low back pain. This is true particularly with the intervertebral disc. For example, if a patient is under fifty years of age, there is a fifty percent chance their pain is due to an internal disc disruption; lumbar facets are also pain generators that make up a significant percentage. Central and lateral stenosis may be the cause of disability found in the elderly population as well as in younger people who are genetically predisposed. Another area is within the intervertebral foramen. As a point of interest neurogenesis often has taken place at the site. The following case reports demonstrate these four locations and also the nerves involved.

Case I: A 49 year old male presented in January, 2010 was in good health, weighed 205 lbs, and stood 6' 2". He works as a registered massage therapist.

History: Onset was burning pain started in early 2008. A few months later the pain was radiating to the right posterior thigh and lateral leg (peroneal division). MRI was negative.
By 2009 the radiating pain was moderate to severe at times and at other times only mild pain was experienced. Pain indefinitely increased with sitting for long periods. The distribution of the pain became more distal and more intense in the peroneal division after any lifting activities. Excessive rowing on an exercise machine caused compression or friction irritation to the right pelvic and ischeal areas. Some numbness or parasthesias were in the peroneal division of the right lower leg.
Pre-disposing factors include a frank labral detachment of the right hip in 2006 and a T12 compression fracture in 1980.
He was seen by me on five occasions in 2010.

Outcomes Measures - **Pre-treatment**:
-Observation: Limping gait
-Palpation: Lumbar muscle hypertonicities
-Symptoms: Burning pain radiating down the right posterior thigh and leg. Pain intensity 4-5 out of 10.
-Strength: Weak hip flexion, no other weakness noted
-Orthopedic tests: SLR slightly positive on right
-ADL'S: -Driving for periods longer than 1 hour became very painful, flying in an aircraft for any period of time, during his clinical day performing treatments became painful by the 2nd or 3rd hour of a 6 hour day

Post Treatment:
-Observation: Normal gait
-Symptoms: 90% reduction in pain
-ADL's: Only mild pain when sitting for long periods, no pain during his work day
-Strength: No change in hip flexor strength

Treatment:

End range loading tissues within the intervertebral foramina of the lumbar spine.

Note the belt is over the ASIS's. Flexion and lateral flexion is applied. See in the picture on the right how long axis traction is applied

Diagnosis: Non-vascular edema within the right lateral canal and intervertebral foramen of the lower lumbar spine affecting blood supply to the nerves located here.

Reference: Yeung A.T., Yeung C.A. In-vivo endoscopic visualization of patho-anatomy in painful degenerative conditions of the lumbar spine, Surg Technol Int. 2006; 15:243-56.

Case II: A 35 year old male presented in May 2010. He is in good health and works as a stock broker.

History: He was in a motor vehicle accident in June, 2007. His vehicle rear-ended a car that had cut in front. He was travelling at 50 mph. Shortly after he suffered from low back pain. After the MVA and prior to his first visit with me, he experienced no significant improvement in his lower back symptoms. After the various treatments he had undergone and prescribed home exercises he performed, every time he attempted to get back into his pre accident routines he would become injured again and go back to 'square one'.
He had no predisposing factors.
He was seen in my office on thirteen occasions in 2010.

Outcomes Measures- Pre-Treatment:
-Symptoms: LBP pain 4-9 on VAS scale, Rt. leg paresthesias
-Imaging: MRI -L4-5 left sided disc bulge
-Orthopedic tests: SLR 80 Deg. bilaterally, ROM restriction in trunk flexion
-Palpation: L5 tenderness to deep palpation
-Unable to perform any vigorous exercise
-Psychological: depression, anxiety, poor temper, degraded social/personal life, mood swings

Post Treatment:
-Symptoms: Greater than 95% abatement of symptoms
-Orthopedic tests: SLR over 90 deg. bilaterally, full range of motion in lumbar flexion
-Palpation: No tenderness at l5 to deep palpation
-Can perform 99% of all vigorous exercises
-Psychological: much improved

Treatment:
End range loading tissues within the lumbar discs.

Some flexion in the lumbar spine to prevent facet imbrication.
Long axis traction can be applied. The amount of force and the direction of force is all determined by patient response.

For convenience sake, this procedure his held for a longer period of time.

Diagnosis: Lack of blood to nerves within the disc due to local increased hydrostatic pressure.

Reference: Koike Y., Uzuki M., Kokubun S., Sawai T. Angiogenesis and inflammatory cell infiltration in lumbar disc herniation, Spine, 2003 Sep 1; 28(17):1928-33.

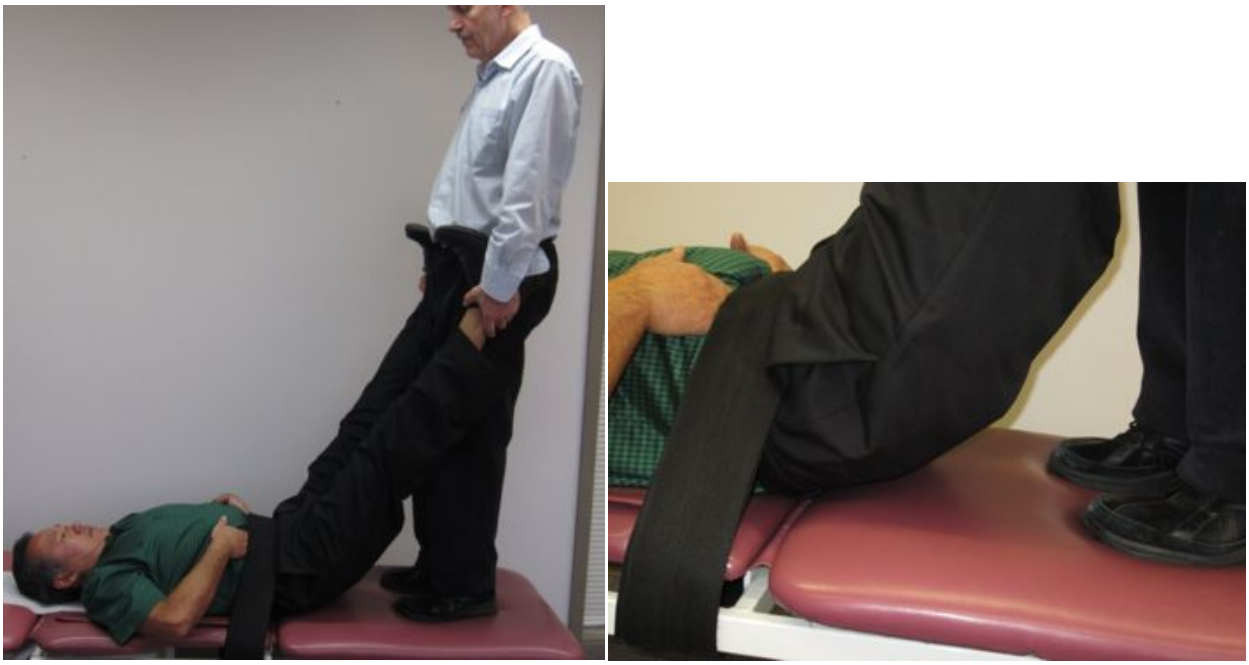
Case III: This 73 year old male retired lawyer presented in October, 2011.

History: He was involved in a motor vehicle accident in 2005 and has been experiencing right sided lower back and buttock pain ever since. His symptoms increase when walking. Imaging studies has revealed a pronounced L4-L5 disc degeneration and moderate L1-L3 disc degeneration. He was receiving chiropractic care for the first three years after the MVA and be doing exercise therapy for the last five years. His symptoms had plateaued over the last several years. He had no pre-existing factors. He was treated on eight occasions from early October to early November 2011.

Outcomes Measures – Pre-Treatment: Right sided lower back and buttock pain 7-8 on the pain scale, difficulty sleeping, social and personal life significantly curtailed, always moving with caution, experiencing constant exhaustion, symptoms exacerbate with walking, hypertonic lumbar musculature, moderate restriction in lumbar extension
Post-Treatment: 60% percent abatement of symptoms, increased range of motion in lumbar extension, far less hypertonicity of the lumbar muscles, no difficulty sleeping, social and

personal life almost back to normal, no exacerbation of symptoms with walking, doesn't move with caution anymore

Treatment: Long axis loading of the lumbar spine with some lumbar flexion.



I always try and look into the patients' eyes. Notice the buttocks are actually raised off the table in the picture on the right.

Diagnosis: Lack of blood to some nerves in the cauda equina.

Reference: Spine (Phila Pa 1976). 2006 Apr 15;31(8):869-72.Effects on improvement of blood flow in the chronically compressed cauda equina Sekiguchi M, Konno S, Kikuchi S.

Case IV: A 66 year old male Caucasian manual therapist was first seen in October, 2006.

History: Low back pain since March 2006 when he hurt himself lifting a heavy object and again reinjured the area when falling awkwardly from a ladder. He had to quit work because of the disability this caused. Since that time he experienced symptoms varying from 1-9 on the pain scale. He had constantly to be vigilant about his ADL's so as not to cause a flare-up resulting in him being almost incapacitated for days on end. These setbacks are often heralded by flashing pains running down the left lower limb. Until now his disability has been characterized by these periods of disability.

This particular case is interesting because of the patient's communication with me and his knowledge of the spine. Not finding relief has led him to seek various forms of care and several consults with experts. Imaging studies revealed some annular tears in the upper lumbar spine, facet degeneration in the lower lumbars and S1, and sclerosing of the sacroiliac joint. He underwent facet blocks on two occasions. Curiously, the doctor couldn't penetrate the left lumbosacral apophyseal joint. Surgery was not advised. He tried active therapy including core stabilizing exercises, manipulation, medications, bed rest and modalities such as IMS, heat, etc.

Outcomes: He now, in November 2011, feels a general since 2006. He still has flare-ups but not as bad and not lasting as long. He is still vigilant in his ADL's but is freer to do more activities. He is even starting back to work as a manual therapist. I see him usually on a monthly basis. This kind of degenerative condition tends to worsen and therefore his prognosis is only fair.

Treatment: End range loading the primary dorsal rami of the left lower lumbar spine.

Again, the amount of force and the direction is determined by the patients' response.

Diagnosis: Instability of the left lumbosacral apophyseal joint, perhaps a form of degenerative spondylolisthesis, resulting in non-vascular edema affecting blood supply to nerves in this joint.

Reference: Medial branch blocks versus pericapsular blocks in selecting patients for percutaneous cryodenervation of lumbar facet joints. Birkenmaier C, Veihelmann A, Trouillier HH, Hausdorf J, von Schulze Pellengahr C. Reg Anesth Pain Med. 2007 Jan-Feb;32(1):27-33.

Discussion and Summary: Notice in the above cases, the diagnosis was entered after the treatment. The logic behind this represents a paradigm shift in clinical reasoning. Remember, we are concerned with chronic mechanical back pain in this paper. I can only be comfortable with a diagnosis after the patient responds to a treatment. The treatment procedure is also an orthopedic and neurological test and therefore the therapist is always looking for patient responses as the techniques are being performed. This communication elicits information that leads to the correct loading strategy and as a result, the diagnosis.

In these four studies the actual sites of the pathologies are less than one inch from each other. I can be sure that any one procedure is going to affect more than one structure. Take for example the cauda equina technique: both the discs and joint capsuls are going to be affected during the procedure.

In case one, we loaded nerves in the lateral canal namely, the nerve roots, the sinuvertebrals, the sympathetics and the interstitials formed from neurogenesis. In case two, the hydrostatic pressure was decreased within the discs allowing blood to flow into the sympathetic interstitial fibres formed from neurogenesis. In case three, by flexion tractioning the lumbar spine, both the central and lateral canals were at a greater volume during the procedure; this increased volume decreased the pressure within the canal and allowed blood to flow in. Nerves affected here could be any of the parasympathetics, sympathetics and the spinal nerves. In my opinion, too much emphasis is placed on neurogenic claudication with spinal stenosis. You will remember nerves to the legs are formed from the plexi which are only from the primary ventral roots of the spinal nerves; any of the nerves within the cauda could be affected. Case four demonstrates nerves from the primary dorsal ramus.

On the initial presentation, therapists, most often only get a general idea of the patient's condition. In the cases above, it took several visits before a diagnosis was formed; the fourth subject took over a year to assess properly. The patients' response to the treatment is the guiding light.