

The Missing Link in Low Back Pain Syndrome: the Iliopsoas Connection?

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A common controversy exists over whether structure governs function or function governs structure. From a comparative anatomist's point of view of anatomical changes over thousands of years, "structure governs function." However, from a chiropractic point of view, "function governs structure" is valid when evaluating and treating our patients.

Barring acquired or congenital structural abnormalities which may limit or cause an increase in motion with regard to the biomechanics of posture and movement, it is the long-term use of the musculature which will ultimately determine the nature of the structure. Vladimir Janda, Czech neurologist, uses postural observation to identify possible areas of faulty motion patterns.¹

A case in point could be made for the iliopsoas muscle. The iliopsoas plays a primary role in determining postural faults and may have a profound effect on the stresses placed on the lumbar spine, ultimately resulting in facet imbrication and discopathy.

Although many muscles are responsible for determining equilibrium of the lumbar spine and body, the iliopsoas has multiple functions: it is bilaterally responsible for flexion movements of the spine and hip joint; and maintains the lumbar lordosis and the correct angle of anteversion of the pelvis, and acts as the lateral stabilization of the spine. If one were limited to directing treatment to a single muscle, probably the most profound effects could result by treating the iliopsoas muscle, a key link in many faulty gait patterns.

An illuminating description of the role of the iliopsoas in the evolutionary development in man is the subject of the second chapter in A. Michele's text, Iliopsoas.² In the other "great apes," gorilla, orangutan and chimpanzee, the lumbar spine is in a flexed or neutral posture with the shorter lower limbs in a slightly flexed position. Whereas in man, the iliopsoas-induced lumbar lordosis and full extension of the proportionally longer legs are a specific and necessary adaptation to bipedal locomotion.

Vladimir Janda and others categorize iliopsoas as a "postural" muscle, which makes it prone to contracture and shortening. Clearly, this may add to the "loading" of the facet and discs and may be implicated in the long term source of back pain from a disc bulging/protrusion. The effects of the iliopsoas shortening may be seen in an altered gait from a failure of hip extension, ultimately causing facet over-strain.

Stodolny and Mazur³ published their findings of a study of low back pain patients and found contracture of the iliopsoas muscle in patients who exhibited the symptoms of low back pain along with the signs of lumbar discopathy. Utilizing post-isometric relaxation exercises, similar to therapeutic muscle stretching techniques, they found an appreciable reduction of shortening within these patient populations. Unfortunately they did not correlate a relationship to a decrease in symptoms of pain with range of motion. In practice, however, most patients experience a sense

of a greater freedom of movement, and often describe a "feeling of being taller," following a therapeutic muscle stretching treatment.

To argue from a different perspective, Godges et al.⁴ conducted a study comparing the effects of end-range static stretching and mid-range proprioceptive neuromuscular facilitation (PNF) stretching techniques for improvements in hip range of motion and gait economy. Although only a preliminary study involving seven subjects, it suggests in cases where end-range stretching is contraindicated (joint pathology or other limited factors), improvement in range of motion can be obtained by utilizing a mid-range PNF-style stretching.

Tight iliopsoas, lower erector spinae and hamstrings are associated with inhibited lower abdominals and gluteals in Janda's crossed pelvis syndrome. The hypertonic hip flexor and iliopsoas shut down the hip extensor and the gluteus maximus. The hamstrings, which have dual functions as a knee flexor and hip extensor, become hypertonic attempting to compensate for the weakened gluts.⁵ This is another possible source of low back joint dysfunction.

Does joint dysfunction precede muscle imbalance, or is muscle imbalance the precipitator of joint dysfunction? This is a case of "which came first, the chicken or the egg?" They both result in establishing faulty motor patterns that can perpetuate chronic low back pain.

It is a moot point to the field doctor. The doctor should assess and specifically treat the soft tissue dysfunction by stretching the shortened muscles and prescribing rehabilitative exercises for their weaker inhibited antagonists, and to manipulate the spinal joint dysfunction.

References

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