Indications for Low Back Prolotherapy

Assessment is not simple, but prolotherapy usually provides satisfactory and permanent results if patients are selected carefully.

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Prolotherapy is an injection therapy used to treat ligament, joint capsule, fascial, and tendinous injuries. It is used to stimulate proliferation of new connective tissue at the sight of injury, thereby restoring strength to injured and weakened connective tissue. Solutions such as dextrose 12.5% are commonly injected creating a controlled inflammatory response. The proliferating substance provokes an inflammatory cascade, the later stages of which include the deposition of collagen. This new collagen is identical in every way to the preexisting collagen.

Prolotherapy is also known as sclerotherapy, a term still used in Britain and by many osteopathic physicians in the United States. Prolotherapy is preferred by some because it avoids Suggesting the hardening of tissues implied in the Greek root scleros. Other terms sometimes used in lay publications include ligament or joint reconstructive therapy.

From ancient times, prolotherapy has been practiced sporadically in different forms. Hippocrates recommended the use of hot cautery to treat recurring shoulder dislocation; however, the modern era of prolotherapy began with the injection of Sylnasol into the temporomandibular joint in 1937 by Schultz, a dentist.[1] In the same year, an osteopathic physician, Gedney, reported injecting the sacroiliac ligaments with neoplas-moid and McDonald's solution, neither of which are currently used.[2] Gedney taught at the Philadelphia College of Osteopathic Medicine, where he sowed the seeds of scle-rotherapy. Teaching and research have continued to this day through the American Osteopathic Academy of Sclerotherapy.

In the early 1940s, George Hackett, a trauma surgeon in Canton, Ohio, began to study ligament injury in his patients. Drawing upon the observations of Head, Baer, and perhaps Kellgren, he mapped pain referral patterns that occurred in his patients with post-traumatic chronic pain.[3-5] If pain were reproduced by the irritation of injection and relieved by local anesthetic, he deduced that the injected sight was the source of the patient's pain. Since many of these pain sources occurred in liga-ments, he also concluded they were actual sights of injury and that the ligaments had been made lax by trauma.

Prolotherapy treatment requires inducing a controlled inflammatory response.

Hackett coined the term *prolotherapy* and wrote a monograph describing these pain patterns and methods of diagnosing and treating ligament laxity.[7] Updated in recent years by Hemwall and Montgomery, it remains the most widely known text on prolotherapy.

After two widely publicized medical disasters involving prolotherapy in the 1960s, prolotherapy fell into disuse. The ideas and techniques were kept alive during this period by a handful of British physicians and the Prolotherapy and Sclerotherapy Societies.[7] The latter two societies were made up largely of American medical doctors and doctors of osteopathy. During the 1980s, a modest revival occurred, resulting in the publication of several key studies on prolotherapy. Among these was an animal study that demonstrated that collagen was indeed formed by injections of sodium morrhuate, which significantly strengthened the fibro-osseous junction.[8] Another controlled clinical trial involving a large group of patients showed that low back prolotherapy was of lasting benefit.[9]

Published studies have continued to demonstrate the value of prolotherapy in low back and knee pain.[10-12] In the United States, the Hackett Foundation, the American Association of Orthopaedic Medicine, and the American Osteopathic Academy of Sclerotherapy teach prolotherapy regularly, offering 2- and 3-day courses. Approximately 600 physicians now practice prolotherapy in North America.

LIGAMENT AND MUSCLE ANATOMY

Ligament, fascia, joint capsules, and muscles are all of mesodermal origin. Evolutionary history shows that muscle is the most primitive of these tissues. This is a counterintuitive observation, since it might seem that tissues necessary for active motion are more highly evolved than passive tissues. Nevertheless, ligaments only appear in mechanically more evolved animals where strength and speed are important. A phylogenetic example is the differentiation of the lower part of the quadratus lumborum into the iliolumbar ligament in early adulthood in man.

The evolutionary origin of muscle and ligaments has both mechanical and clinical implications and is intertwined with function. Tension on ligaments is modulated by muscle attachments directly onto ligaments (eg, gluteus maximus onto the sacro-tuberous ligament) or by muscles running parallel to ligaments (paraspinal, multifidus, and intertransversii muscles and intervertebral ligarnents). Muscle may even modulate the tension of a joint capsule. This should not be surprising if the tensegrity model of mechanics is accepted.[13] Ligaments and muscles are tension elements essential to the strength and stability of the whole.

Ligaments protect muscles from injury and allow them to rest under certain circumstances, eg, locking of the knee and the hip when standing easy. Ligaments not only transmit forces generated by muscles, but also may, through their elastic properties, have an energy storing effect. Dorman has hypothesized that this is a key function of the pelvic ligaments in gait.[14]

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Because ligaments are stronger than muscles and cannot give way when overloaded, they probably bear the brunt of external trauma, especial-ly when large forces are involved. Commonly a cascade of ligament injury occurs in which the number of injured ligaments in a region is proportional to the severity of the trauma. This phenomenon is well recognized in the shoulder and forms the basis of the classification system of acromioclavicular injuries; the coracoclavicular ligaments become involved only in the more severe (class 3 and 4) injuries. Hackett believed that the posterior sacroiliac ligaments were the most commonly injured. The interosseous, sacrospinous, and sacrotuberous ligaments appeared to be involved in more serious injuries. With single ligament injuries, however, the iliolumbar ligament is most often implicated. This may explain why pain from the iliolumbar ligament has been described as a distinct syndrome, whereas pain from the other low back ligaments has not. [15]

Although in vitro studies had shown that peripheral ligaments most commonly fail in the midsubstance when stressed,[16] this is not necessarily true in vivo particularly for the more central ligaments of the low back. Clinical experience seems to suggest that the fibro-osseous junction is the more common site of injury, at least in those instances that become chronic. This is the working hypothesis from the point of view of prolotherapy, where permanent laxness is believed to occur at this spot. Trigger points then develop because the sensory nerve endings are more vulnerable to the otherwise innocuous strains of everyday life.

MECHANICS

Low back mechanics are exceedingly complex. Despite many years of research on the topic, clinicians cling tenaciously to different schools of thought. This reflects not only poor standards of examination, but also a lack of communication between clinicians and researchers.

Probably no group of clinicians has carried clinical assessment of low back mechanics as far as the osteopathic profession. From the early 1900s, Lovett and later Fryette described the mechanics of the spine in great detail.[17,18] Mitchell, Greenman, and Kuchera have demonstrated clinical methods of examining the pelvis and have outlined the mechanical assumptions that accompany them.[19-21] These assumptions include recognition of multiple axes of motion, both physiologic and nonphysiologic somatic dysfunctions and a close interaction with surrounding muscles and total body mechanics.

Although these examination methods are widely accepted by the osteopathic profession and many physiotherapists, interexaminer reliability remains a problem. Also, the observations are unintelligible to those not trained in manipulation. Technology does not yet exist that can demonstrate to the non-initiated the mechanics postulated by the osteopathic profession.

The allopathic understanding of low back mechanics has for the most part remained primitive. In the early part of the last century, sacroiliac strain was a common diagnosis and some research implicated the pelvic ring.[4,22] Mixter and Bar's paper on disc herniation in 1934 changed all this, however, and most low back mechanical research has since centered on the disc and the surrounding structures.

A small but steady stream of papers on sacroiliac anatomy and mechanics continued in parallel through the 1950s, 1960s, and 1970s, mostly by nonclinicians.[24-26] This research added little to the knowledge of physicians already familiar with sacroiliac joint examination; however, the prevailing view that the sacroiliac joints are immobile proved irrational.

In the early 1980s, a number of new developments began to challenge this stagnant Situation. Osteopathic and medical physicians began to interact, first in the North American Academy of Manipulative Medicine and soon after in the American Association of Orthopaedic Medicine. Manual medicine flowered in Europe and transatlantic communication increased. Physiotherapy and chiropractic became increasingly sophisticated and cooperation between these and the medical professions improved. Consequently, the need for

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better research, especially in mechanics, became apparent.

In the 1990s, two international conferences on the sacroiliac joint were held bringing together physicians, physiotherapists, anatomists, engineers, and other researchers from North America and Europe. First class research on all aspects of sacroiliac joint function and dysfunction was presented. Of particular interest to those interested in prolotherapy was the relative importance of ligaments and muscles in joint stability.

The engineering terms "form closure" and "force closure" were introduced to differentiate the passive (ligament and joint) mechanisms versus the active (myofascial) mechanisms providing joint stability.[27] Although no consensus yet exists as to their relative importance, both appear to be important in sacroiliac joint stability.

Although the understanding of pelvic ring mechanics has advanced greatly in the last decade, the central challenge of demonstrating the relationship between abnormal mechanics and pain remains unmet.[28-30]

LOW BACK STABILITY

Instability can be defined as a loss of the functional integrity of a system that provides stability. It is a broad term that can be applied to any dynamic system. When applied to the low back, it can mean anything that consistently interferes with normal function.

Hypermobility and hypomobility are mechanical terms that directly apply to orthopaedic medicine. Hypomobility refers to the restriction of motion of any body part. Hypermobility refers to excessive range of motion and is of special interest to physicians practicing prolotherapy.

Joint hypermobility may be categorized into two types: *primary* and *secondary hypermobility*. Primary hypermobility results from a weakening of the joint capsule and ligaments. Peripheral joint hypermobility occurs commonly following injury, but central axial joint hypermobility may occur as well. The most obvious examples are sacroiliac joint hypermobility and intervertebral hypermobility after motor vehicle accidents. Hypermobility of this kind may be difficult to detect clinically as muscles will compensate in many different ways. Piriformis syndrome, quadratus lumborum syndrome, and psoas syndrome are just a few of the muscular patterns that may develop in response to hypermobility in the pelvic or lumbar intervertebral joints.

Secondary hypermobility may develop as a result of abnormal neuromuscular control of a joint or group of joints. Weakness of passive supporting structures may not exist, but the mechanical effect may be identical to that of a primary joint hypermobility.[31] An example of secondary joint hypermobility would be sacroiliac joint hypermobility caused by a change in muscular tension of the gluteus maximus muscle acting upon the sacrotuberous ligament. The sacrotuberous ligament is a major support of the sacroiliac joint.

The origin of secondary hypermobility may be quite complex, since the neuromuscular abnormality may

be part of a much larger postural pattern. Abnormalities emanating from mechanical disturbances in the lower extremity, trunk, neck, and cranium are common and make treatment of the local problem inefficient or impossible if not addressed.

The autonomic nervous system may affect muscular balance by facilitating muscles or activating muscle trigger points. Sources of autonomic nervous system destabilization include visceral disturbances (viscerosomatic reflexes) and foci of electrophysiologic instability ("interference fields") such as scars, teeth, and nerve entrapments.[32]

CLINICAL ASSESSMENT OF LOW BACK INSTABILITY

Both a medical history and physical examination are important. Radiologic studies of the pelvic ring may demonstrate mechanical abnormalities when stressed in different ways,[33] but their clinical significance is not known. Lumbar spine dynamics have been studied radiologically with little success. The Spinoscope, a device that tracks intervertebral motion using llght-emitting diodes taped to the skin, shows promise for the lumbar spine, but is ineffective for the pelvic area.

In an unstable back, pain may be induced or exacerbated by prolonged standing or sitting, and relieved by activity – the so-called "Theatre-Cocktail Party syndrome," coined by Barbor.[7] Clinical experience has shown that prolotherapy benefits these patients. Some have concluded that patients with unstable backs have ligament laxity with or without secondary muscular trigger points and pain.

Another symptom of low back instability is recurrent episodes of acute back pain associated with unguarded movements of the trunk possibly resulting from lack of ligamentous mechanical support. Patients with this problem benefit from prolotherapy, but less so than those with "Theatre-Cocktail Party syndrome."

There is no consensus about which physical signs indicate low back instability. The osteopathic concepts of physiologic and non-physiologic somatic dysfunction imply that the soft tissue supports of the sacroiliac joints may, under certain circumstances, decompensate and cause a true subluxation of the sacroiliac joint. Others consider recurrent pelvic asymmetry or "asymlocation" to be significant. Whether this apparent asymmetry results from displacement of bones or from changes in the overlying soft tissues has been called into question.[34]

The sitting and standing flexion sign (or variations of these tests) are widely used to assess pelvic ring mechanics, but their reliability and significance have not been established. Presumably they demonstrate abnormal motion of the sacroiliac joint, but this does not necessarily indicate joint instability.

Various techniques may be used to isolate large low back ligaments and test their sensitivity to stretch. Stretching the ligament to reproduce the patient's pain is thought to be a sign of ligament pain and laxity. For persons with above-average manual skills, passive motion palpation of the pelvic ring joints and the vertebral segments is direct evidence of relative joint hyper- or hypomobility.[31,35,36] This probably reveals more about the stability of the joints than does assessment of symmetry and active

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motion testing. These techniques, however, suffer from three major limitations: (1) The required skills are highly subjective, difficult, and time-consuming to acquire; (2) Interexaminer reliability is often too poor to be valuable in research; and (3) Although changes in motion characteristics can be detected at the time of the examination, they may not represent the stability of the joint in everyday life. This is where the modulating effect of muscle activity on ligament tension comes into play (secondary hypermobility). Changes in posture and recruitment of different supporting muscles may completely alter the patterns of joint stability.

There is another method for diagnosing low back instability that rests on the assumption first proposed by Hackett about chronic pain emanating from a ligament trigger point (always at the fibro-osseous junction) that results from ligament laxity. The best evidence to support this assumption is that repeat injections of a proliferating solution frequently do abolish the pain and the trigger points.

A number of difficulties arise, however, when the theory of ligament laxity is examined more carefully. The first is the question of whether pain at a ligament trigger point is always, usually, or only sometimes due to unresolved injury. In ligaments supporting central joints, this is particularly difficult to determine, but examination of peripheral joints may provide some theoretical answers. It

should first be noted that joint instability by itself does not necessarily mean that lax ligaments supporting a joint produce pain even when stretched to a certain degree. A weakened anterior cruciate ligament can be demonstrated to be lax, without provoking pain when it is stretched. Clinical experience certainly suggests that lax ligaments may render the joint and its supporting ligaments more vulnerable to injury, but with everyday usage they are generally painless.

Not only are lax ligaments not necessarily painful, but also healthy ligaments may produce pain under certain circumstances. This may be easily demonstrated in a finger interphalangeal joint. If a proximal interphalangeal joint is gently abducted and tension put on the opposite collateral ligament for a minute or so, pain will gradually develop. Even when the tension is removed, some pain will remain and a repetition of the strain will induce the same pain more quickly than before. In other words, the ligament becomes sensitized by prolonged tension and readily produces pain.

In most peripheral joints, active muscle contraction does not produce abnormal tension on ligaments. In central axial joints, however, this almost certainly does occur. How often it occurs is open to conjecture, but abnormal postural patterns resulting from injury, illness, degenerative processes, or emotional stress cannot help but place prolonged abnormal tension on central ligaments. This might be the cause of at least some of the ligamentous pain blamed on ligamentous laxity.

Another cause of ligament pain is heightened sympathetic nervous

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system tone. Ligaments in the lumbar and pelvic regions are richly supplied with sympathetic nervous system efferent fibers. Sympathetic efferent fibers may activate primary afferent fibers or potentiate inflammatory processes by releasing neuropeptides and catecholamines.[37] This may explain the sudden, lasting, response to low back prolotherapy in some patients. Almost certainly this is a neural therapy effect from the local anesthetic that is always mixed into proliferant solutions.

Ligament pain does not necessarily indicate ligament laxity, and ligament laxity does not necessarily cause pain. Evidence of pain coming from a ligament alone is, therefore, an inadequate indication for prolotherapy.

OVERVIEW OF LOW BACK PROLOTHERAPY INDICATIONS

Gedney's original paper in 1937 described prolotherapy of both the sacroiliac joint and the knee. His indication was "joint instability due to elongated ligament structure following trauma from whatever cause." He also used the term "hypermobile joint" and referred to "lacerated" ligaments, but did not explain how he made these assessments. In 1951, he described a technique of testing vertebral segments for passive motion (Dandy's sign).[38]

Hackett described in great detail methods of detecting lax ligaments.[39-43] History was very important, as was palpating potential ligament trigger points. He felt it important not just to find trigger points, but also to have the patient agree upon the particular point.

Perhaps Hackett's greatest contribution was the mapping of pain referral patterns from ligamentous trigger points. His textbook is rich in clinical pearls such as the observation that pain down the posterior leg is ligamentous in origin if it skips the back of the knee, and is true sciatica if it does not.

The detection of ligamentous trigger points was essential to Hackett's method. He believed that trigger points could only develop from ligamentous laxity. Naturally prolotherapy was the treatment. For many years he paid scant attention to muscles, believing that muscle spasm was mostly a secondary phenomenon. In acute low back pain, he recommended waiting for the muscle spasm to settle before examining for the underlying ligamentous laxity.

By 1955, Hackett realized, however, that laxity at the osseotendinous junction could also occur, and he began treating these trigger points with prolotherapy. From our current vantage point, it seems surprising that nowhere does he consider that pain at a fibro-osseous junction could be caused by chronic excess tension. Perhaps this is because his knowledge of mechanics was limited, and there was no technique allowing him to assess joint mobility.

The British tradition of prolotherapy was probably best represented by Barbor who examined the causes of lumbar instability and divided them into four categories: (1) disc protrusion; (2) sacroiliac strain or subluxation; (3) ligamentous insufficiency; and (4) spondylolisthesis.[7]

Prolotherapy was the treatment or part of the treatment for all of these conditions with the exception of disc protrusion. The term disc protrusion as described by Cyriax is a syndrome characterized by the restriction of gross lumbar motion in one or more, but not all directions. No x-ray or other imaging evidence or neurologic deficit is required to confirm a diagnosis of disc protrusion. As a clinical term, disc protrusion is similar to, but not identical to the osteopathic term somatic dysfunction. Consequently, Barbor believed that prolotherapy should not be performed unless there is normal range of motion in the lumbar spine.

Barbor's diagnosis of ligamentous lesions depended almost entirely on symptornatology. Barbor recommended taking a meticulous history, noting the quality of pain, its location at the time of examination and at its onset, and the effect of posture and activity on the pain. Physical examination required normal range of motion and pain on stretching ligaments. He did not describe his techniques for stretching ligaments in his paper, but emphasized the importance of maintaining stretch for an adequate length of time. In some cases, he used Hackett's technique of injecting suspect ligaments with local anesthetic and observing the response.

Dorman extended the ideas of his predecessors in the British school by incorporating ideas of mechanics derived from osteopathy.[44] He concurred with Cyriax in citing intervertebral disc fragments as a source of pain in the lumbar spine; however, he believed the disc to be more vulnerable to injury when the intervertebral ligaments were insufficient. He was able to describe many situations implicating ligaments as a source of pain in the lumbar spine and in the pelvic ring.

Perhaps Hackett's greatest contribution was the mapping of pain referral patterns from ligamentous trigger points.

Central to his thinking is the concept of *asymlocation* of the sacrum, a term coined to describe the static expression of disturbed sacroiliac mechanics. Sacral asymlocation not only puts strain on its supporting ligaments and muscles, but also distorts the mechanics of the lumbar spine. In his view, most disturbed mechanics have underlying ligamentous insufficiency as its basis.

Because this paradigm is ligament-centered, the possibility of painful ligaments (either lax or tense) from abnormal muscle balance is not considered. His treatment protocol (Ongley's method), named after his mentor Milne Ongley, combines manipulation and injections to relieve pain and relax muscles.[12]

SUMMARY

The main purpose of prolotherapy is to strengthen and tighten ligaments around hypermobile joints. The challenge remains to: (1) determine when joint hypermobility is a cause of pain; (2) identify the affected joint or joints; and (3) decide if ligament laxity is the cause of the hypermobility.

For most prolotherapy practitioners, ligament tenderness is synonymous with ligamentous laxity. It is assumed that when pain demonstrably emanates from other sources, eg, muscle trigger points, the underlying cause is ligament laxity; however, these assumptions rest on minimal evidence.

Deciding if ligament pain results from laxity, excessive tension, a combination of the two, or some other cause requires a reliable method of assessing primary joint hypermobility, ie, the impact of form and force. Such a method does not yet exist.

Until primary hypermobility can be accurately diagnosed, the indications for rational low back prolotherapy should include (1) a history of injury; (2) physical findings consistent with joint hypermobility; and (3) a failure to maintain joint stability after skilled treatment for all potential causes of secondary hypermobility.

REFERENCES

1. Schultz LW. A treatment for subluxation of the temporomandibular joint. JAMA. 1937; 109:1032-1035.
2. Gedney EH. Hypermobile joint: a preliminary report. *Osteopath Prof.* June 1937:30-31.
3. Head H. On disturbances of sensation with special reference to pain of visceral disease. *Brain.* 1893;16:1-13.
4. Baer WS. Sacro-iliac strain. *Bull Johns Hopkins.* 1917;28:159-163.
5. Kellgren JH. Observations of referred pain arising from muscle. *Clin Sci.* 1938;3:175-190.
6. Hackett GH, Hemwall GA, Montgomery GA. *Ligament and Tendon Relaxation Treated by Prolotherapy.* 5th ed. Oak Park, III: Gustav A. Hemwall; 1991.
7. Barbor R. Sclerosant therapy, the theory of treatment of ligamentous disturbance by a dextrose sclerosant. *Reunion Sobre Patologia tie la Columina Vertebral.* Murcia, Spain. March 30, 1977.
8. Liu YK, Tipton CM, Matthes RD, Bedford TG, Maynard JA, Walmer H. An in situ study of the influence of sclerosing solution in rabbit medial collateral ligaments and its junction strength. *Connect Tissue Res.* 1983;11:95-102.
9. Ongley MJ, Klein RG, Dorman TA, Eek BC, Hubert IJ. A new approach to the treatment of chronic low back pain. *Lancet.* 1987; 2:143-146.
10. Klein RG, Eek BC, DeLong B, Mooney V. A randomized double-blind trial of dextrose-glycerine-phenol injections for chronic, low back pain. *J Spinal Dis.* 1993;6:1:23-33.
11. Klein RG, Dorman TA, Johnson CE. Proliferant injections for low back pain: histological changes of injected ligaments and

objective measurements of lumbar spine mobility before and after treatment. *J Neurol Orthop Med Surg.* 1989;10:123-126.
12. Ongley MJ. Dorman TA, Eek BC, Lundgren D, Klein RG. Ligament instability of knees: a new approach to treatment. *Man Med.* 1988;3: 151-154.
13. Levin SM. The icosahedron as the three-dimensional finite element in biomechanical support. Proceedings of the Society of General Systems Research Symposium on Mental Images, Values and Reality. Philadelphia, Pa, May 1986. St. Louis, MO: Society of General Systems Research, 1986:G 14-G26.
14. Dorman T. Storage and release of elastic energy in the pelvis: dysfunction, diagnosis and treatment. *J Orthop Med.* 1992;14:54-62. 15. Hirschberg GG, Froetscher L, Naeim F. Iliolumbar syndrome as a common cause of low back pain: diagnosis and prognosis. *Arch Phys Med Rehab.* 1979;60:415-419.
16. Butler DL, Grood ES, Noyes FR, Zernicke RF. Biomechanics of ligaments and tendons. *Exer Short Sci Rev.* 1978;6:125-181.
17. Lovett RW. *Lateral Spinal Curvature of the Spine and Round Shoulders*. Philadelphia, Pa: P. Blakistons Son & Co; 1907.
18. Fryette H H. Physiologic movements of the spine. *J Am Osteopath Assoc.* 1918; 18:1-2.
19. Mitchell Fl Jr, Moran PS, Pruzzo NA. *An Evaluation and Treatment Manual of Osteopathic Muscle Energy Procedures.* Valley Park, Mo: Mitchell, Moran and Pruzzo Associates; 1979.
20. Greenman PE. *Principles of Manual Medicine.* Baltimore, Md: Williams & Wilkins; 1993.
21. Kuchera WA, Kuchera ML. Osteopathic principles in practice. 2nd ed. Kirksville, Mo: KCOM Press; 1992.
22. Chamberlain WE. The symphysis pubis in the roentgen examination of the sacroiliac joint. *Am J Roentgenol Radium Ther.* 1930;24:621-665.
23. Mixter WJ, Bar JS. Rupture of intervertebral disc with involvement of spinal canal. N Engl J Med. 1934:211:210-214.
24. Weisl H. The articular surfaces of the sacroiliac joint and their relations to the movements of the sacrum. *Acta Anat.* 1954;22:1-14. 25. Solonen KA. The sacroiliac joint in the light of anatomical roentgenological and clinical studies. *Acta Orthop Scand Suppl.* 1957; 27 (suppl):1-127.
26. Colachis SC. Worden RE, Bechtol CO, et al. Movement of the sacroiliac joint in the adult male: a preliminary report. *Arch Phys Med Rehabil.* 1963;44:490-498.
27. Snijders CJ, Vleeming A, Stoeckart R, Mens JMA, Kleinrensink GJ. Biomechanics of the interface between spine and pelvis in different postures. In: Vleeming A, Mooney V, Dorman T, Snijders CJ, Stoeckart R, eds. *Movement, Stability, and Low Back Pain.* New York, NY: Churchill Livingstone; 1997:103-114.
28. Kidd RF. Pain localisation with the innominate upslip dysfunction. *Man Med.* 1988;3:54-96.
29. Kidd RF. Does the innominate upslip cause musculoskeletal pain? *Man Med.* 1992;6:169-17().
30. Laslett M. Pain provocation sacroiliac joint tests: reliability and prevalence. In: Vleeming A, Mooney V Dorman T, Snijders CJ, Stoeckart R, eds. *Movement, Stability, and Low Back Pain.* New York, NY: Churchill Livingstone; 1997:287-294.
31. Kidd RF. The innominate upslide sign and assessment of pelvic ring stability. *J Orthop Med.* 1997;19:43-46.
32. Dosch P. *Manual of Neural Therapy According to Huneke.* 1st English ed. Heidelberg, Germany: Haug Publishers; 1984.
33. Dihlman W. Diagnostic radiology of the sacroiliac joints. New York, NY: Georg Thieme Verlag; 1980.
34. Lewit K, Liebenson C. Palpation-problems and implications. *J Manipulative Physiol Ther.* 1993;16:586-590.
35. Lee DG. Treatment of pelvic ring instability. In: Vleeming A, Mooney V, Dorman T, Snijders CJ, Stoeckart R, eds. *Movement, Stability and Low Back Pain.* New York, NY: Churchill-Livingstone; 1997:448-451.
36. Hesch J, Aisenbrey J, Guarino J. Manual therapy evaluation of the pelvic joints using palpatory and articular spring tests. First Inter-disciplinary World Congress on Low Back Pain and its Relation to the Sacroiliac Joint. San Diego, Calif. November 5-6, 1992:435-459.
37. Willard FH. The muscular, ligamentous and neural structure of the low back and its relation to back pain. In: Vleeming A, Mooney V, Dorman T, Snijders CJ, Stoeckart R, eds. *Movement, Stability and Low Back Pain.* New York, NY: Churchill-Livingstone; 1997:30.
38. Gedney EH. Disk syndrome, new approach in the treatment of symptomatic intervertebral disk. *Osteopath Prof* 1951;18:12:11-46.
39. Hackett GS. Joint stabilization through induced ligaments sclerosis. *Ohio State Med J.* 1953;49:877-884.
40. Hackett GS. Shearing injury to the sacroiliac joint. *J Int Coll Surg.* 1954; 22:631-642.
41. Hackett GS. Referred pain from low back ligament disability. AMA *Arch Surg.* 1956;73:878-883.
42. Hackett GS. Low back pain. *Br J Phys Med.* 1956;19:25.
43. Hackett GS. Referred pain and sciatica in low back diagnosis. JAMA. 1957;163:183-185.
44. Dorman TA, Ravin T. *Diagnosis and Injection Techniques in Orthopaedic Medicine.* Philadelphia, Pa: Williams & Wilkins; 1995.