Prolotherapy for Sciatica from Weak Pelvic Ligaments and Bone Dystrophy

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by finding trigger-point tenderness and is confirmed by intraligamentous needling with a local anesthetic solution. By promoting the growth of new bone and fibrous-tissue cells, prolotherapy strengthens the ligament attachments and eliminates pain, including sciatica.

Sciatic neuritis occurred frequently as a dystrophic complication of weak sacroiliac ligaments in 1857 patients seen during 21 years. Diagnosis by trigger point tenderness of specific ligaments was confirmed by needling. The fibro-osseous attachments of ligament to bone were strengthened by prolotherapy, i.e., the intraligamentous injection of a proliferating solution.

When ligament fibers do not regain their normal tensile strength following strain, the

fibers stretch under normal tension and permit excessive tension-stimulation of the non-stretchable sensory nerve fibrils,² which are abundant within the fibro-osseous attachments.^{3,4} This is the origin of noxious barrages of sensory afferent and antidromic impulses⁵⁻⁹ which cause pain and bone dystrophy (decalcification¹⁰). Decalcification¹¹ in disease, menopause and old age also causes ligament and tendon re-

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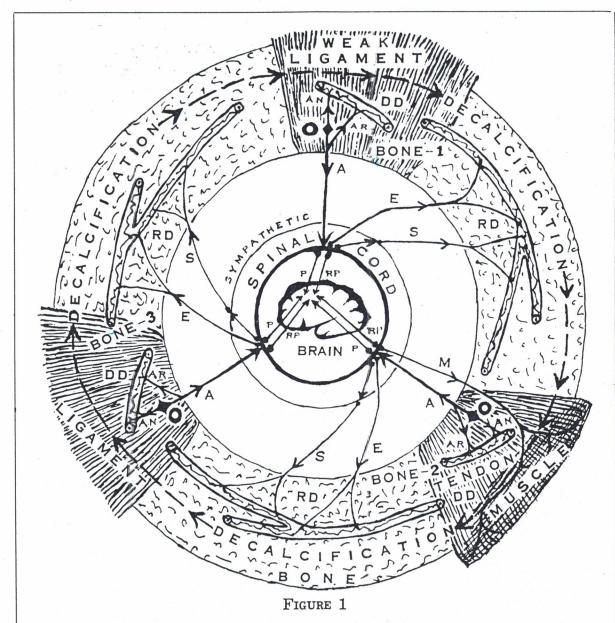
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^{1.} Hackett, G. S., Ligament and Tendon Relaxation Treated by Prolotherapy, Third Edition, Charles C Thomas, Springfield, Illinois, 1958.



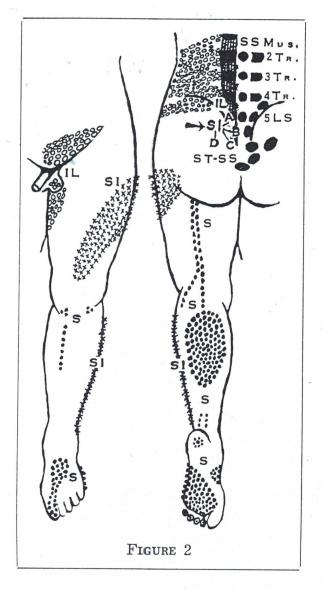
- O—Origin of neural impulse by traction-stimulation of nerve fibrils within weakened fibro-osseous attachment of ligament to bone
- A-Afferent sensory somatic noxious impulse to spinal cord
- AR—Axon reflex neurovascular impulse in antidromic direction through afferent fibril
- AN—Antidromic neurovascular impulse
- DD—Direct decalcification (metabolic)
 - E-Efferent neurovascular impulse
 - S—Sympathetic neurovascular impulse
- RD—Reflex decalcification (metabolic)
 - M-Reflex motor impulse to muscle
 - P—Pain impulse to brain
- RP—Referred pain impulse to brain

Note: The etiology may be trauma or disease. All ligament-tendon attachments become weakened when invaded by decalcification and are the origin of most pain in all types of osteoporosis.

laxation^{12,13} at their bony attachments.

Tension stimulation on afferent sensory nerves within the weak fibro-osseous attachments of ligament to bone is the origin of barrages of afferent impulses transmitted to the spinal cord and to the brain where they are interpreted as pain and referred pain, while from the same origin barrages of antidromic impulses pass directly and by axon reflex to bone blood vessels and cause a neurovascular disturbance of bone metabolism that results in direct decalcification, which further weakens the ligamentous attachment to bone. From the afferent stimulation in the spinal cord, there are noxious barrages of efferent impulses that cause muscle spasm, while other efferent and sympathetic impulses14 pass from the same and adjacent cord segments and cause a reflex neurovascular decalcification 10 of larger areas of bone. This weakens the attachment of all ligaments and tendons in the decalcified areas^{10,11,15} and completes a vicious circle^{2,16} (Figure 1) of ligament relaxation and decalcification.

Relaxation of the posterior



sacroiliac ligament is accompanied by local trigger-point pain and tenderness (Figure 2, SI-A, B,C,D) and referred pain areas to the outer anterior thigh (SI) and outer side of the leg. 17,18 Severe relaxation of the sacroiliac ligament results in decalcification of the sacrum, ilium, and ischium, which weakens the attachments of all ligaments and tendons to the decalcified areas

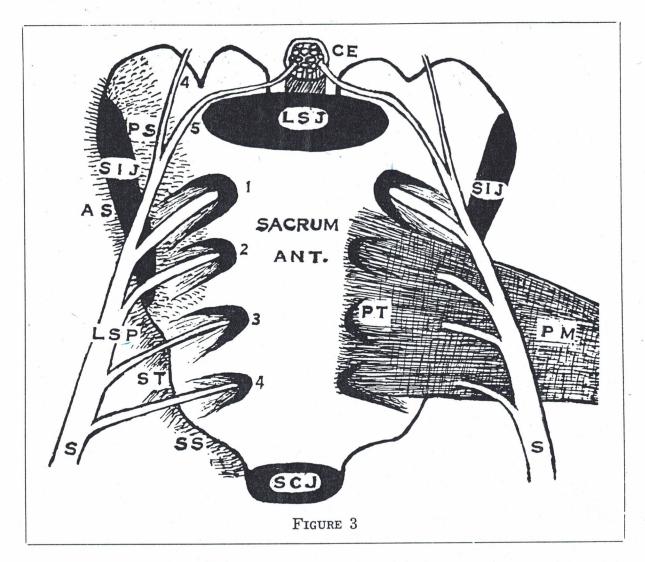
^{12.} Meisenbach, R. O., Surg. Gynec. & Obst., 12:411-434,1911.

^{13.} Livingston, W. K., Pain Mechanism, Macmillan, New York, 1943.
14. Drucker, W. R., et al., Am. J. Surg., 97:

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^{15.} Watson-Jones, R., & Roberts, R. E., Brit. J. Surg., 21:461-499,1934.
16. De Takats, G., Arch. Surg., 34:939,1937.

^{17.} Baer, W. S., Bull. Johns Hopkins Hosp., 28:159-163,1917. 18. Hackett, G. S., Ohio M.J., 49:877,1953.



of bone. 10,11 Those relaxed ligaments and tendons which are of special importance in contributing to sciatica are the sacrotuberous (ST), sacrospinous (SS) and anterior sacroiliac ligaments, and the tendons of the piriformis muscle,1,5,19-24 for they become additional sources of noxious bombardments of afferent and antidromic impulses which are transmitted into the sciatic nerve components.

Sciatica

The piriformis tendons are attached to the bodies of the 1,2,3,4 segments of the sacrum (Figure 3), to the grooves of the 2,3,4 sacral foramina, and to the anterior sacroiliac (capsular) ligament (AS), the lower portion of the posterior sacroiliac ligament (PS), and the sacrotuberous and sacrospinous (SS) (ST)

^{19.} Yoeman, W., Lancet, 11:119,1928. 20. Freiberg, A. H., & Vinke, T. H., J. Bone & Joint Surg., 16:126-136,1934. 21. Freiberg, A. H., Ohio M.J., 1934. 22. Ross, J. P., St. Bart. Hosp. Rept., 65:103-1031

^{108,1932.}

^{23.} Hackett, G. S., Arch. Surg., 73:878,1956. 24. Hackett, G. S., J.A.M.A., 163:183-185,1957.

ligaments. The 4,5 lumbar and 1 sacral nerves (lumbosacral plexus (LSP)) cross the anterior sacroiliac ligament (AS) 25,26 and join the 2,3,4 sacral nerves as they emerge with congenital variations through the piriformis tendons (PT) and muscle $(PM)^{20,23,27-30}$ to form the sciatic nerve (S). A continuous fibrous sheath envelops and is attached to the piriformis muscle and tendons; the 2,3,4 sacral nerves, the 4.5 lumbar and 1 sacral nerves of the lumbosacral plexus, and sciatic nerve; and the anterior sacroiliac, sacrotuberous, sacrospinous, and lower margin of the posterior sacroiliac ligaments.

Noxious bombardments of sensory nerve impulses from within the relaxed ligament/tendon attachments to bone cause pain and inflammation^{19,31} within the encapsulated tissues (nerve, muscle, tendon, ligament, blood and lymph vessels, fibrous tissue). Inflammation increases their sensitivity and extends into the sciatic nerve trunk to account for its tenderness. 11,24,31 Spasm of the piriformis muscle is a contributing factor.20

Noxious bombardments of antidromic impulses not only disturb local neurovascular metabolism, but may extend throughout sciatic nerve distribution^{2,31} and cause early decalcification as revealed by x-ray. This weakens the ligaments and tendons in the lower extremity, causing instability and additional pain and decalcification.10 Impaired function of the sciatic nerve inhibits reflexes and causes muscle dystrophy.

Sciatic pain (Figure 2, S) that has been identified as a complication of relaxed sacroiliac (SI-A.B.C.D) and sacroischial (ST, SS) ligaments that normally stabilize the sacroiliac joint has been described by the patient as deep in the posterior thigh and popliteal space, gripping pain deep in the calf, at one or both sides of the Achilles tendon, and like a hot coal or a sharp tack beneath the heel. It may feel like a pressure beneath the instep, and from the outer side of the foot, it may cross underneath the metatarsal arch and into the 2,3, 4.5 toes. Anteriorly it may burn on either side of the upper tibia or feel like a piercing nail. It may extend medial to the upper tibia. It often involves the top of the arch of the foot and extends into the great toe. When severe, any

^{25.} Hilton, J., Rest and Pain, Second Edition, Wm. Wood & Co., New York, 1879.
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29. Jackson, C. M. (Editor), Morris' Human Anatomy, Fifth Edition, Blakiston's Son & Co. Philadelphia, 1914. Co., Philadelphia, 1914.

^{30.} Lewin, P., Backache and Sciatic Neuritis, Lea & Febiger, Philadelphia, 1943.
31. Chapman, L. F., et al., Arch. Neurol., 1: 557-572,1959.

of the pain areas in the thigh, leg or beneath the heel may be tender, but not tender distal to the ankle.

Tendon relaxation of the three gluteii by decalcification of the ilium and sacrum is a frequent painful complication of severe sacroiliac ligament relaxation.¹⁰ A referred pain from the gluteus minimus has been identified on the outer side of the leg and foot just posterior to the ankle joint.

Lumbosacral Joint Ligament Instability

This condition is found in the proportion of two to three in sacroiliac joint instability. 1,32 It consists chiefly of relaxation of the interspinous ligament (Figure 2, 5LS) 1,33 and the iliolumbar ligament (IL). When severe, there is accompanying relaxation of the articular capsular ligaments, the upper part of the posterior sacroiliac ligament (SI-A,B) and the tendons of the sacrospinalis muscle (SS Mus) at their attachments to the 1,2,3 sacral segments and the transverse processes of the lumbar vertebrae (TR). The ligament/tendon relaxations may extend to the lower dorsal vertebrae with trigger-point tenderness, and with referred pain laterally.

Relaxation of the iliolumbar

ligament (IL) is an excellent example of the result of noxious stimulation originating within the attachments of one relaxed ligament. A referred somatome appears in the upper gluteal muscles from the attachment to the transverse process of the fifth lumbar vertebra. From the ilial attachment there is a dermatome in the groin and upper medial thigh (IL), pain and tenderness in the testicle or vagina, a dysfunction of the large intestine manifested by gas pains, constipation, or diarrhea, urgency to void, and there may be decalcification of the vertebrae and ilium.

Referred pain from the interspinous ligament has not been identified. It must be relaxed to permit abnormal strain on the articular capsular ligaments, abnormal movement of one vertebra upon another, or abnormal pressure on an intervertebral disc.

Since relaxed articular capsular ligaments have been identified with noxious stimulation of cervical, dorsal, and lumbar spinal nerves^{1,10} (radiculitis), it would be a logical hypothesis that bombarding impulses from relaxed 4,5 lumbar articular capsular ligaments and attached relaxed tendons would cause pain, inflammation, antidromic dystrophy, and dysfunction in the 4,5

 ^{32.} Steindler, A., & Luck J. V., J.A.M.A., 110: 106-113,1938.
 33. Kohler, R., Acta Radiologica, 52:21-27,1959.

lumbar spinal nerves which adjoin them and are enclosed in the same sheath of fibrous tissue. This would account for some sciatic pain accompanying lumbosacral disability.

Severe lumbosacral ligament relaxation is frequently accompanied by relaxation of the upper portion of the posterior sacroiliac ligament (SI-A,B) because of the synchronizing forward rotation of the ilium on the sacrum in the sacroiliac joint, along with flexion of the lumbar spine.^{1,34}

Symptoms and Diagnosis

The chief symptoms of sacroiliac ligament relaxation complicated with sciatica are local pain, referred pain, and sciatic pain. The sciatic pain has usually been preceded by recurrent attacks of sacroiliac ligament pain. The pains are aggravated by activity and by traction when the disability is severe. The perception and intensity of pain varies with the pain threshold of the patient. In severe cases the ankle reflex may be diminished or absent, and the circumference of the leg and thigh may be reduced as much as one inch.

There is trigger point tenderness of the attachments of the posterior sacroiliac ligament (Figure 2, SI-A.B.C.D) and the

sacrotuberous and sacrospinous (ST,SS) ligaments, and there may be referred pain (SI) to the outer anterior thigh and outside of the leg. Referred pain areas are constant for specific ligaments.

Trigger point tenderness of the upper portion of the posterior sacroiliac (SI-A,B), the lumbosacral interspinous (5-LS), and the iliolumbar (IL) ligaments are best elicited with the patient standing erect and relaxed, while the lower portion of the sacroiliac (SI-C,D), sacrotuberous and sacrospinous (ST-SS) ligaments are best elicited while the patient reclines relaxed on the abdomen.¹

Trigger-point tenderness beneath the buttock in the sciatic notch at the lower margin of the sacroiliac joint (SI-D) is due to relaxation of the posterior sacroiliac, sacrotuberous, and sacrospinous ligaments. It is present before sciatica develops and becomes accentuated with inflammation involving the piriformis muscle^{10,21,24} and sciatic nerve.

Trigger-point tenderness of the sciatic nerve is best obtained while the patient lies prone with the knee flexed to a right angle and the foot supported by the examiner's left hand. The right thumb is rolled over the median and outer branches of the sciatic nerve in the popliteal space and

^{34.} Francis, C. C., The Human Pelvis, Mosby, St. Louis, 1952.

compared with the opposite side. Pain on straight leg raising in sciatica, in severe sacroiliac relaxation, and other conditions reduces its value in differential diagnosis.

X-rays do not reveal ligament relaxation per se, but decalcification in the area of clinical pain and tenderness is evidence of ligament and tendon relaxation in the area.

The earliest x-ray evidence of decalcification from sacroiliac ligament relaxation may appear in three or four weeks7,10,35,36 and is observed in the ischial tuberosity by fading of the major trabeculae and pitting or mottling of the bone margin. It is verified by trigger-point tenderness of the medial aspect of the tuberosity by pressure with the thumb or with one finger in the vagina or rectum, and the pain is reproduced by needling.

It has been found³³ that in pathologic cases a contrast medium injected bilaterally adjacent the lumbar interspinous ligaments penetrates into the ligament through defects.

The diagnosis of ligament and tendon relaxation is confirmed by intraligamentous needling with a local anesthetic solution. This will produce intense trigger point pain, which disappears within two minutes as anesthesia takes place.

Treatment

When sciatica is a complication of posterior sacroiliac ligament relaxation, the treatment consists of strengthening the fibro-osseous attachments of the posterior sacroiliac, sacrospinous, and sacrotuberous ligaments to bone by prolotherapy^{1,23,36,37} (a method of rehabilitation effected by inducing proliferation of new bone and fibrous tissue cells). It permanently stabilizes the sacroiliac joint and eliminates all pain, including sciatica. Other complications, such as ligament/tendon relaxation of the lumbosacral, hip, and coccygeal joints, the sacrospinalis and gluteal muscles, and sometimes the sacrotuberous attachment to the medial aspect of the ischium, are also treated.

Technique consists in using a 22-gauge Luerlock needle of sufficient length to contact bone. This needle is attached to a 10 cc. syringe containing the proliferant combined with a local anesthetic solution. After inserting the needle to contact bone within the fibro-osseous attachment, five drops are injected, the needle then partly withdrawn and redirected against bone. Usually three to eight injections are made during one insertion

^{35.} De Lorimier, A. A., Bull. Hosp. Joint Dis., 12:22-37,1951.
36. Compere, E. L., & Kernahan, W. T., Med. Clin. North America, 42:299-307,1958.
37. Hvid, N., Ugesk. laeger, 121:619-622,1959.

of the needle, depending on the size of the area. Treatments may be given in office, but severe cases are treated in hospital. New bone and fibrous tissue develop over some four weeks, and the patient returns for report and reexamination in one month. Additional treatments are given when indicated.

A dextrose proliferating solution which gives adequate fibroosseous proliferation with a minimum of discomfort can be made up by any pharmacist.

DEXTROSE STOCK SOLUTION

Dextrose BP	25.0%
Phenol BP	2.5%
Glycerine BP	25.0%
Dist. water to	100%

This can be made up and placed in 100 cc. rubber-stoppered bottles. The solution is self-sterilizing. Prior to injection, the solution should be mixed as follows: one part dextrose stock solution, and three parts tetracaine (Pontocaine) 0.1 per cent or lidocaine (Xylocaine) 1.0 per cent. During one treatment, 50 cc. of this solution has been given in multiple bone contact injections of $\frac{1}{3}$ cc. each (4-5 drops).

Proliferating solutions (Sylnasol, zinc sulfate) should be diluted to half strength of previous recommended dosage. The usual analgesics and sedatives for highly nervous, low threshold, and sciatica patients can be withdrawn early during treatment.

Early administration of estrogen, androgen, and vitamin C, together with thyroid, 1 to 2 grains daily, appears to accelerate the formation of bone matrix^{38,39} and to give the patient a feeling of well-being.

Any support that gives relief is beneficial in preventing bombardments of neural impulses. The Brooks-Hackett sacroiliac belt is diagnostic by immediately relieving strain (pain) sacroiliac ligaments, but is ineffective in other lowback disability, and in sciatica from other causes. It is used following acute strain and following treatment to support the ligaments while they are gaining strength. A raised heel prevents full knee extension and traction on the weakened attachment of biceps tendon fibers to the decalcified ischial tuberosity.21

Activities that induce pain are prohibited; pain is the alarm signal of the noxious stimulation of impulses that inhibit normal metabolic rehabilitation and induce decalcification. Traction which induces pain and increases ligament relaxation should be avoided.

Comment

It is impossible to dissociate ligament relaxation and decalci-

^{38.} Albright, F., Rec. Prog. Horm., 1:293-353, 1957.

^{39.} Ibarra, J. D., Texas M.J., 52:20,1956.

fication, for either one may precede and induce the other. Decalcification weakens the attachments of ligament and tendon to bone a few weeks before it can be recognized by x-ray. Weak ligament and tendon attachments probably account for much of the pain associated with osteoporosis of the spine and pelvis.

Animal experiments over six-year period revealed new bone proliferation.40 Intraligamentous injections of whole blood stimulated bone and fibrous tissue one-fourth as effectively as a sclerosing agent.* It probably has the same effect clinically in the repair of torn ligament/tendon fibers when not disturbed by too much activity. Blood serum, saline, anesthetic solutions, and iron tonics were ineffective. The injection of steroids combined with proliferants inhibited the proliferation of any new bone and fibrous tissue for three weeks, followed by a diminished proliferative effect. The intraligamentous injection of steroids is inadvisable either in acute or prolonged disability when it interferes with the inflammatory process of healing.

The number of patients with ligament relaxation treated in the past 21 years is 1857; of these, 1583 had sacroiliac liga-

ment relaxation. Ages ranged from 15 to 88 years. Duration of disability was three months to 65 years. Patients had consulted as many as 30 physicians over the country and abroad.

With experience and with the encountering of more serious cases, lumbosacral disability and sacroiliac complications (decalcification, sciatica), these become easily recognized. Of 412 consecutive cases of sacroiliac ligament relaxation during the past three years, 312 (78 per cent) were complicated by sacroischial (tuberous, spinous) ligament relaxation. These cases were classified as cases of sciatica or potential sciatica, the diagnosis depending on pain distribution (popliteal, calf, foot). Among them were some 80 patients with obvious sciatica. Many had undergone spine operations, and ligament disability continued; 82 per cent were treated by prolotherapy to their satisfaction. There were no unfavorable sequelae. Failures may be attributed to improper diagnosis, activity, insufficient treatment, and co-existing disabilities.

Summary

Sciatica occurs frequently as a complication of sacroiliac ligament relaxation (incompetency). Barrages of sensory and antidromic impulses induced by trac-

^{40.} Hackett, G. S., *Mil. Med.*, 126:517-525,1961. *Sylnasol,® G. D. Searle & Co., Chicago, Illinois.

tion-stimulation on non-stretchable sensory nerve fibrils within the fibro-osseous attachment of weak sacroiliac ligament fibers are the cause of pain and neurovascular dystrophy (decalcification) of the sacrum and ilium. The decalcification weakens the attachments of the sacrotuberous and sacrospinous ligaments and the tendons of the piriformis muscle, so that they become the origin of additional barrages of impulses that overwhelm the sciatic nerve components resulting in the production of pain, inflammation, dystrophy, and dysfunction. It is a vicious circle of ligament relaxation and decalcification in which either may induce the other.

The diagnosis of ligament relaxation is by trigger-point tenderness. It is invariably confirmed by intraligamentous needling with a local anesthetic solution.

Treatment by prolotherapy permanently strengthens the ligament attachments with new bone and fibrous-tissue cells, stabilizes the sacroiliac joint, and eliminates the pain, including sciatica.

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