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Back Pain Following Trauma and Disease—* Prolotherapy

By

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Induced Fibro-Osteogenesis strengthens weak ligament/tendon attachments to bone.
Clinical therapy-21 years; Animal experiments-8 years.

(With five illustrations)

MUSCLE coordination, balance, sensation, sense of position, and neurovascular metabolism of skeletal tissue are in response to nerve impulses that have their origin by tension-stimulation[1] of the abundant afferent sensory nerve fibrils and tension receptors which are located within the muscle-tendon and ligament attachments to bone.[2]

The ligament/tendon fibro-osseous attachments are weakened when they do not regain their normal tensile strength following sprains and tearing of the fibers, and when the fibers are weakened by decalcification such as occurs in disease, menopause and old age.[3]

This weakness of the ligament attachment to bone has been found to-be the cause of much pain, referred pain, sciatica, and decalcification during the past 21 years in the management of 1,857 patients with ligament relaxation in which the diagnosis was invariably confirmed by needling, and the fibro-osseous attachments were strengthened by induced recalcification.[4]

Weak ligament fibers stretch under normal tension and permit an abnormal tension-stimulation on the nonstretchable sensory nerve fibrils and tension receptors[5] (Fig. 1). This becomes the origin (0) of : Barrages of afferent sensory impulses (A) which cause pain and referred pain (P,RP), muscle spasm (M) and reflex efferent (E) and sympathetic (S) neurovascular disturbances of bone metabolism (reflex decalcification) (RD), and inflammation of the nerves; also barrages of antidromic impulses[5-8](AN, AX) which cause direct neurovascular decalcification (DD), and inflammation of nerves.

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Decalcification weakens the bony attachments of all ligaments and tendons in the area, and they become additional sources of afferant and antidromic impulses of pain, inflammation and decalcification. It is a vicious circle.

DIAGNOSIS

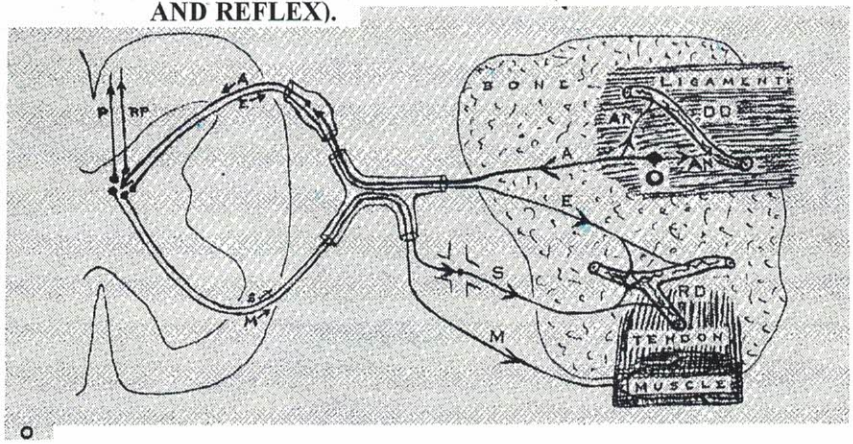
Diagnosis of ligament relaxation by trigger point tenderness of specific ligament attachments to bone is confirmed by intraligamentous needling with a local anesthetic solution[3] (Fig. 2). Referred pain areas are constant and direct attention to specific ligaments. Decalcification (Fig. 3) in severe cases may be observed on x-rays in 3-4 week S[7-9] by mottling of the bone margin and fading of the major trabecula.[4]

Kohler[10] reported degeneration of 4,5 lumbar interspinous ligaments accompanied by increased mobility in cadavers, and clinically at operation after x-rays had revealed that contrast media invaded the ligaments when injected laterally.

TREATMENT

Treatment of weak ligaments/tendons is by prolotherapy[3] *(Gr-Poles,* new cells). It is accomplished by the induced proliferation of new bone and fibrosis tissue cells which permanently strengthen the fibro-osseous attachments to bone (Fig. 3). The technic consists in injecting a combined proliferating and local anesthetic solution within the weak fibro-osseous attachment. A few drops are distributed in proximate positions while the point of the needle contacts bone.

POST-TRAUMATIC BONE DYSTROPHY. ORIGIN OF IMPULSE-WEAK LIGAMENT. A VICIOUS CYCLE OF PAIN, REFERRED PAIN AND DECALCIFICATION (DIRECT AND REFLEX).



O - ORIGIN OF NEURAL IMPULSE BY TRACTION-STIMULATION OF NERVE FIBRILS, WITHIN WEAKENED FIBRO•OSSEOUS ATTACHMENT OF LIGAMENT TO BONE
A - AFFERENT SENSORY SOMATIC NOXOUS IMPULSE TO SPINAL CORD
AR - AXON REFLEX NEUROVASCULAR IMPULSE IN ANTIDROMIC DIRECTION THROUGH AFFERENT FIBRIL
AN - ANTIDROMIC NEUROVASCULAR IMPULSE
DO - DIRECT DECALCIFICATION (METABOLIC)
E - EFFERENT NEUROVASCULAR IMPULSE
S - SYMPATHCTIC NEUROVASCULAR IMPULSE
RD – REFLEX DECALCIFICATION (METABOLIC)
M - REFLEX MOTOR IMPULSE TO MUSCLE
M - PAIN IMPULSE TO BRAIN
RP - REFERRED PAIN IMPULSE TO BRAIN

FIG. 1. Noxious Stimulation of Exaggerated Neural Response in Weak Fibro-osseous Attachment of Ligament to Bone.

Barrages of impulses *originate (0)* in an afferent somatic sensory nerve (A) when weak ligament fibers stretch under normal tension and permit *tension-stimulation* of the non-stretchable nerve fibrils within the fibro-osseous junction. Barrages of afferent impulses are transmitted (A) to the spinal cord and (P, RP) to the brain where they are interpreted as *pain* and *referred pain.*

Antidromic impulses are transmitted directly (AN) and by axon reflex (AR) to periosteal and bone blood vessels, and cause *direct decalcification* (DD) in the area of ligament attachment to bone by an imbalance of neurovascular bone metabolism.

Reflex decalcification (RD) takes place over *a* larger area of bone from impulses that are transmitted reflexly from the spinal cord through efferent (E) and sympathetic (S) nerves to bone blood vessels.

Muscle spasm results from reflex motor impulses (M) as a protective measure.

The new bone and fibrosis tissue cells become strong in 4-6 weeks. The patient returns for re-evaluation in 8 weeks and additional injections are given when indicated. Two proliferating solutions have been used clinically: (A)-Sylnasol (G.D. Searle & Co.) 1 part combined with 4 parts of Pontocaine 0.15% (Winthrop Lab.) ; (B)-Zinc Sulfate stock solution is combined with pontocaine and saline and can be prepared by any chemist.

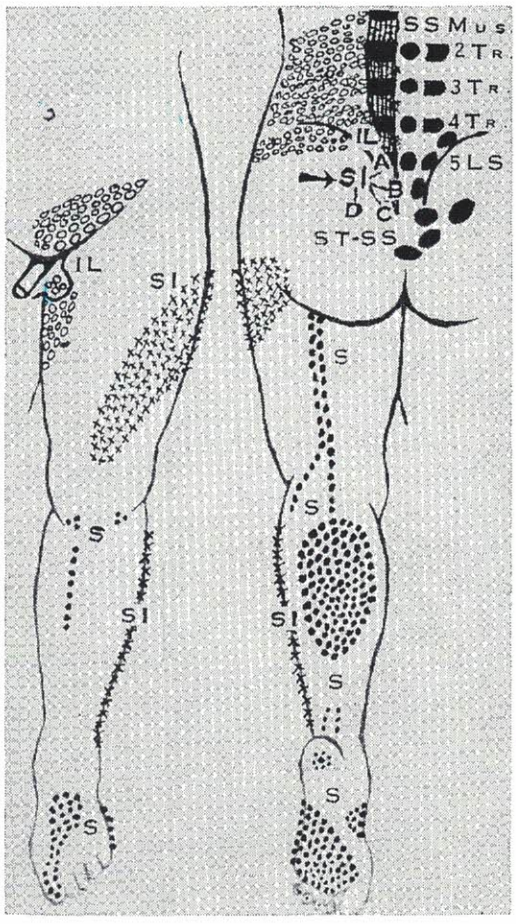
Zinc Sulfate-Phenol Stock Solution

Zinc Sulfate	8 gms.
Liquid Phenol	12 cc.
Glycerine	24 cc.
Dist. water add to	100 cc.

Mix solution for injection as follows:

Zinc Sulfate—Phenol Stock	2 cc.
Pontocaine	42 cc.
Normal saline	42 cc.

When patients are hospitalized and comfort is assured by analgesics and sedatives,



Fir- 2. Low Back Ligament and Tendon Disability.

Lumbosacral: Trigger point tenderness of the lumbar interspinous ligament (5-LS) is medial, and of the iliolumbar (IL) ligaments laterally, with referred pain into the buttock, groin, thigh, testicle, vagina, and large intestine (IL). When severe, there is accompanying trigger point tenderness of the upper portion of the relaxed posterior sacroiliac ligament (SI-A, B) with referred pain in the thigh and leg (SI). Also trigger point tenderness of the relaxed tendon attachments of the sacrospinal muscle (SS MUS) to the sacrum and transverse processes of the lumbar vertebrae, with referred pain laterally into the loin.

Sacroiliac: There is trigger point tenderness at the attachments of the relaxed posterior sacroiliac ligament (SI-A, B, C, D) and referred pain into the thigh and leg (SI). When severe, there is accompanying tenderness of relaxed sacrotuberous (ST) and sacrospinous (SS) ligaments and sciatic nerve, with pain in the area of sciatic nerve distribution (S).

Sciatica is a complication of severe relaxation of the posterior sacroiliac ligament (SIA, B, C, D), when it is accompanied by relaxation of the sacrotuberous (ST) and sacrospinous (SS) ligaments, and the tendons of the piriformis muscle, and an associated decalcification of the sacrum and ilium, and with inflammation and pain in the area of sciatic nerve distribution.

Sylnasol 1 part is diluted with saline 3 or 4 parts, thus eliminating the toxicity of the anesthetic solution so that a greater amount may be injected over larger areas. 50 cc. of Sylnasol combined with 100 cc. of saline has been used on a hospitalized patient during one treatment.

Sylnasol and Zinc Sulfate solutions have been well tolerated when adequate pre and post-treatment analgesics are given. They are devoid of systemic or allergic reactions to most patients. When such reactions occur they are usually due to the local anesthetic solution or analgesic to which the patient is susceptible.

Pontocaine provides prolonged anesthesia with a minimum of discomfort, systemic and allergic reaction, and combines readily with the proliferating agents. Some doctors prefer Xylocaine (Astra Phar.).

Male and female hormones and Vitamin C. such as Formatrix (Ayerst Lab.) and Thyroid gr., 1 or 2 daily, have appeared to materially assist in recalcification.[11, 20]

ANIMAL EXPERIMENTS

Animal experiments were started in 1952 to explain the scientific rationale for the favorable clinical results that had been obtained by strengthening weak ligaments in painful low back disability, and have been continued along with clinical observations to determine the most satisfactory combinations that would have the desirable features of : a minimum of inflammatory edema and discomfort ; no impairment or destruction of any tissue ; and a maximum of new bone and fibrous tissue that would become permanent.[12]

Our earlier report on animal experiments

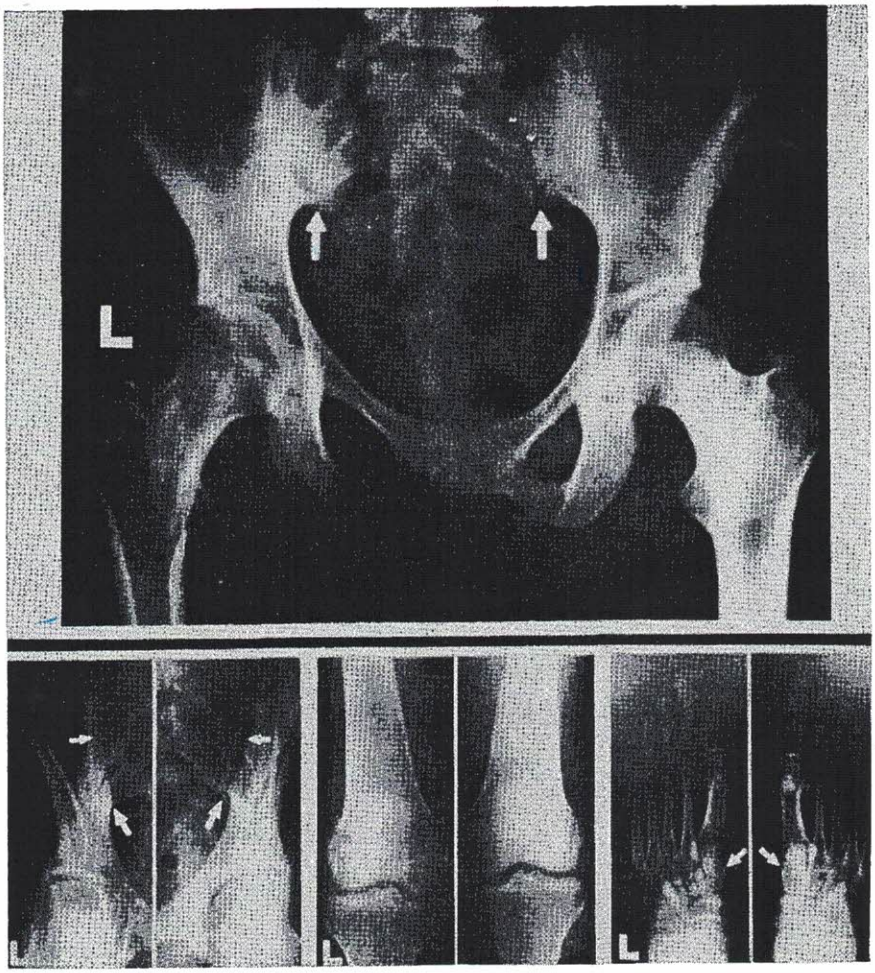


FIG. 3. Sacroiliac Ligament Relaxation, Pelvic Decalcification, Sciatica, and Recalcification

A 23-year-old nurse severely sprained the left posterior sacroiliac ligament, from which noxious bombardments of impulses caused pain and neurovascular *decalcification* of the sacrum and ilium that relaxed the fibro-osseous attachments of the sacrotuberous and sacrospinous ligaments and the tendons of the piriformis muscle. This induced *sciatica* one month later while hospitalized in traction. Decalcification progressed throughout the left side of the pelvis and lower extremity (arrow).

Prolotherapy stimulated Recalcification (arrow) of the attachments of the left sacroiliac, sacrotuberous and sacrospinous ligaments, eliminated the disabling pain, and stabilized the sacroiliac joint, so that gradual increasing activity facilitated Recalcification throughout, and complete recovery.

in 1955[13] presented a history of induced fibrous tissue proliferation clinically and experimentally since 1835 when it was introduced for the treatment of hernia and later extended to include other disabilities.[14] Our report included illustrations of the induced proliferative stages from inflammation to adult bone and fibrous tissue, and tendons that had increased 40% in diameter and bone 30% in diameter, which were estimated to double tensile strength of the ligament and its bony attachment.

Schultz in 1937[15] reported 30 patients with subluxation of the temporomandibular joint that were successfully treated by injecting Sylnasol within the joint cavity. Included in the report were animal experiments in which there were no local or systemic disturbances or sloughing, following

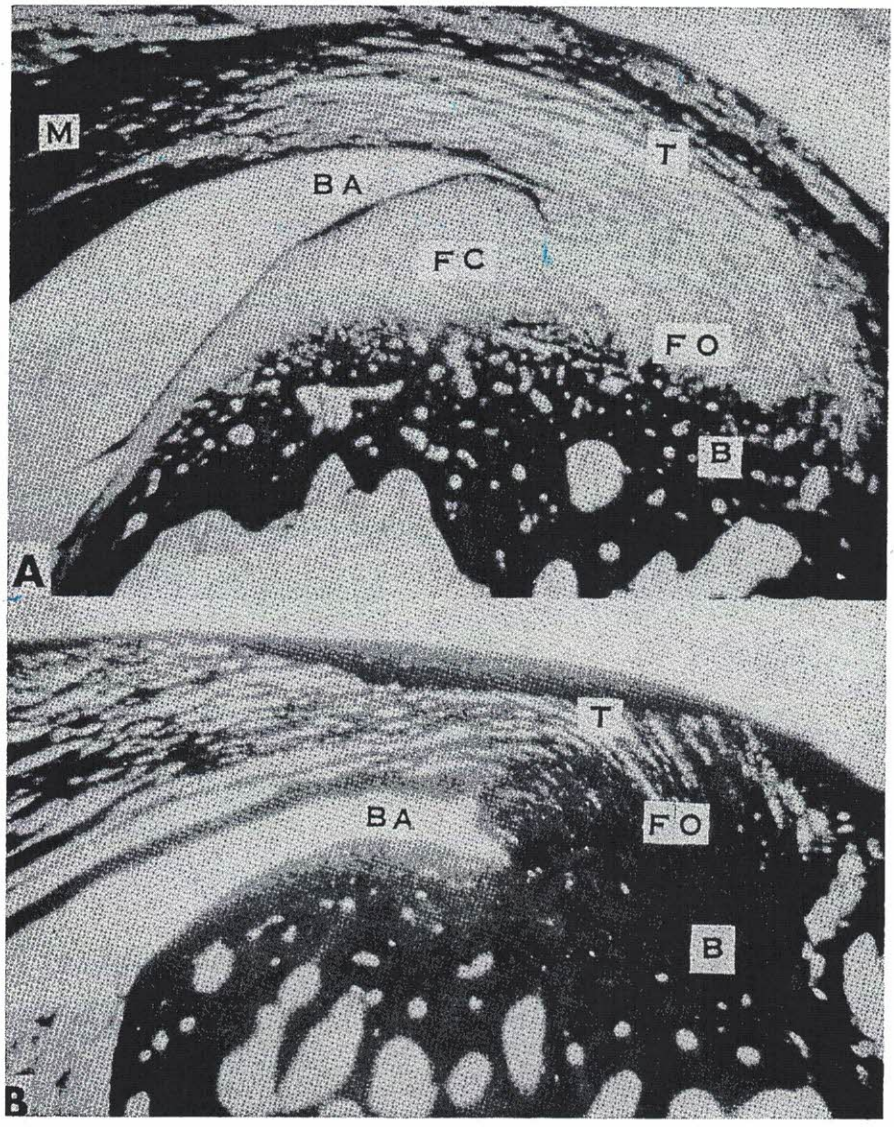


FIG. 4. Microphotographs of Induced Fibro-osseous Proliferation Bone-B, Tendon-T, Muscle-M, Fibro-osseous junction-FO, Fibro-cartilage-FC, Bursal Area-BA.

Microphotographs of decalcified Achilles tendon attachments to the tibio-tarsal bones of a rabbit, two months after 1 injection of 0.5 cc. of a proliferating solution (Sylnasol 25% in Pontocaine) was made into the right leg [B]. The injection was made against bone within the fibro-osseous attachment of the tendon. The control left leg [A] was not injected.

A. Control leg (above)

The tendon fibers (T) blend with the periosteum and continue into bone (B). They are firmly attached by calcification which extends outward into the fibro-osseous junction (FO).

B. Injected leg (below)

Proliferated new bone cells increase bone density (B), extend outward and increase the area and density of the fibro-osseous junction (FO), and encroach on the fibro-cartilage (FC) and bursal area (BA), without penetrating the tendon capsular sheath. The weld of tendon to bone is strengthened.

TABLE 1

Solutions injected to determine their effect on fibro-osseous proliferation in 8 weeks following injection as compared with controls	Fibro-osseous proliferation	Early acute inflammation
1. Controls	0	0
2. Sylnasol 33% in saline (G. D. Searle & Co.)	5	4
Sylnasol 25% in Pontocaine (Winthrop Lab.)	4	4
Sylnasol 25[1]76 + Cortisone in Pontocaine	1	1 -
3. Zinc sulfate (stock sol.) 4cc. in Pontocaine-saline 84cc.	5	4
Zinc sulfate (stock sol.) 3cc. in Pontocaine-saline 84cc.	4	4 -
4. Sotradegol 25% in Pontocaine (Wallace-Tiernan)	2	*2*
5. Varisol 25[1]7o in Pontocaine (Abbott Lab.)	2	2
6. Q.U. (Plasma) 25% in Aquacaine (Farnsworth Lab.)	*2* -	*2* -
7. Salicylic acid sol. in Pontocaine (Montrose Chem.)	1	1
8. Benzyl Salicylate in oil (Bensal)	1	3
9. Whole blood (Citrated from blood bank)	1+	1 -
10. Blood plasma (Citrated from blood bank)	0	0
11. Imferon (Iron Dextran, Lakeside Lab.)	0	0
12. Calcium Glucinate (Jansen-Salsbery Lab.)	1 -	0
13. Estrogen, Androgen, Vit. C. (Formatrix, Ayerst Lab.)	0	0
14. Cortisone	0	0
15. Butazoladine Solution (Geigy Pharm.)	0	0
16. Silica Crystals S102, in Pontocaine (Dequin Phys. Lab.)	5+	3
Silica Fluoride SIOz, CaFt Pontocaine (Dequin Phys. Lab)	4+	51
Silica Oxide suspension (Abbotts)	3	2
17. Effect of daily exercise on proliferation (1 year)	1+	0
18. Fractured tibia-fibro-osseous proliferation in	*Callous*	Hematoma
3 weeks as compared to controls		*Inflammation*
Control fracture	2	2
Sylnasol 25% (Injected at fracture site)	4	3
Zinc sulfate 4cc. (Injected at fracture site)	4+	3
Cortisone (Cortone acetate) (Injected at fracture site)	1	1
Sylnasol + Cortisone (Injected at fracture site)	2 -	1+

1. One or more controls were used in each litter.

2-3. Sylnasol 1 part with Pontocaine 3 parts (25%), and Zinc sulfate stock solution-3 cc. with Pontocaine-saline 84 cc. (as used clinically) are equivalent in inducing fibro-osseous proliferation. Injections repeated at 2 month intervals induced greater proliferation than single injections. The reactions of one year old slightly discolored solutions showed no remarkable difference than fresh solutions. Sylnasol has been recognized as a proliferating agent of choice for 25 years", and Zinc sulfate has been used for 60 years".

4-6. Limited fibro-osteogenesis.

7-8. Inadequate fibro-osteogenesis. (Injected peri-articularly for arthritis in England.)

9-13. Whole blood stimulates fibro-osteogenesis moderately. Blood plasma, iron and calcium solutions and steroids are ineffective in stimulating fibro-osseous proliferation.

14-15. Cortisone Acetate and Hydrocortone (Upjohn) injected alone and when combined with proliferants, inhibited proliferation for 3-4 weeks and materially affected the end results following injection within tendons and at the site of fractures.

16. Various silica preparations were used. Silica Oxide crystals (SIO2) is an excellent proliferant with only moderate early inflammation. Silica Fluoride (SIF) is effective but is accompanied by excessive early inflammation. Both precipitate rapidly causing the syringe plunger to stick. Silica Oxide suspension (Abbott Lab.) does not cause the syringe plunger to stick. (Perhaps a higher percentage of silica would be a more effective proliferant.)

17. Exercises of strain applied daily for 12 months revealed a gradual increase of fibrous proliferation and only a slight proliferation of bone as compared with the control.

18. Solutions of Sylnasol, Zinc Sulfate and Silica injected at the site of fractures (Tibia) markedly increased callous formation, over a period of 3 weeks as compared with controls, but callous was markedly inhibited when they were combined with Cortisone.

0.5 cc. of Sylnasol and Zinc Sulfate solutions injected within the tibio-talus joint induced a peri-articular inflammation. There was no sloughing but moderate limitation of motion.

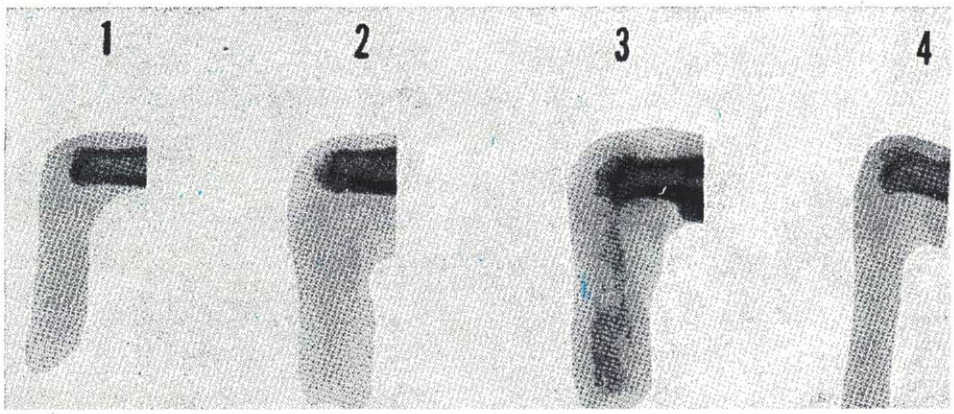


FIG. S. X-rays of Induced Fibro-osseous Proliferation

1. Control, 2. Sylnasol, 3. Zinc sulfate, 4. Zinc sulfate + Cortisone

X-rays of the Achilles Tendon attachment to the tibio-tarsal bone of a rabbit, 1 month after 1 intraligamentous injection of 0.5 cc. of fibro-osseous proliferants.

1. Control. A faint shadow reveals bone extending into the tendons at the fibro-osseous junction where the tendon fibers enter the end of the bone and are firmly attached by ossification.

2-3. Injections of Sylnasol and Zinc sulfate solutions have stimulated the proliferation of new bone as revealed by bone enlargement and increased density, which also extends further within the tendon where ossification of the fibers strengthens the weld of tendon to bone.

4. Injection of Zinc sulfate and Cortisone solutions combined (3 to 1), reveal that Cortisone inhibited the proliferative action of the Zinc sulfate solution.

repeated injections of Sylnasol subcutaneously, and within joint, pleural, peritoneal and heart cavities. In 1956 he further reported the "soundness and efficacy" of the same treatment in several thousand patients with "lax" joint ligaments.[15]

The injection technic within tendon attachments to bone is the same as that used clinically. Experimental injections in animals were also made within joints, at the site of fractures, and intrathecally (Table 1

One hundred ninety-two (192) New Zealand white rabbit Achilles tendons were used to determine the amount of new bone and fibrous tissue that was induced over variable periods from a few days to one year, following one or more injections of various solutions into the fibro-osseous junction of tendon to bone. The table reveals the comparative fibro-osseous proliferation that resulted over a period of 8 weeks following a single injection of 0.5 cc. of various solutions. The acute inflammatory edematous reaction following the injections are also recorded.

Intrathecal injections in rabbits of clinical intraligamentous dosage (4-5 drops) had no noticeable effect. (It is further diluted by the spinal fluid.) When the dose was increased to produce spinal anesthesia, complete recovery occurred. It was necessary to use much greater then clinical dosage to induce paraplegia for a few weeks duration, which also cleared up.

The strength of the proliferating agents are so greatly diluted that 5 drops (each bone contact dose) contains only 0.01 grains of phenol crystals, and 0.017 grains of Zinc Sulfate. Further dilution would take place by mixing the 5 drops with 120 cc. of spinal fluid, should an inadvertent injection be made clinically into the spinal canal, which is impossible with our short needles used medially, and the dural sleeve of spinal nerves lying anterior to the vertebral transverse process. Withdrawal of the plunger before the injections are made against bone add additional safety.

STATISTICS

A number of patients (1,857) with ligament relaxation were treated by prolotherapy in 21 years. Ages were from 15 to 88 years. Duration of disability was 3 months to 65 years. They had consulted as many as 30 doctors and had undergone various unsuccessful pelvis and back operations to as many as 6 on the spine. Some had been referred to the psychiatrist. Eighty-two percent (82%) were cured to their satisfaction. There were no unfavorable sequelae. Other physiscians[16-18] have reported similar success on over 11,000 patients.

COMMENTS

Consultations with authorities on physiology, neural and vascular disorders,[5,7-8] were valuable in evaluating our clinical and animal observations. Pain is a warning signal of noxious sensory nerve stimulation in weak ligaments/tendons, with an associated disruption of normal physiological and metabolic response. Consequently continued activities and rehabilitation exercises that cause pain are contraindicated.

Induced recalcification eliminates the noxious stimulation of neural impulses and permits gradual resumption of normal activities and reestablishment of neurovascular metabolism.

It seems apparent that extravasated blood in torn ligament/tendon attachments to bone and in fractures, mildly stimulates the proliferation of new bone and fibrous tissue cells in physiological repair. Also exercises stimulate the proliferation of fibrous tissue slowly and bone to a less degree. When disability continues, only prolotherapy will provide adequate fibro-osteogenesis. This is especially true in the loose-jointed type individual with an inadequate osteogenetic proclivity.[3,19] The search for a silica crystal suspension that would have a minimum of acute inflammation and discomfort, and a more prolonged proliferation because of its electrolytic reaction, has not been abandoned.

SUMMARY

Back pain occurs frequently from ligament/tendon attachments to bone that have been weakened by strain, and by decalcification of disease, menopause and old age. Weak ligament fibers stretch on normal tension and permit abnormal tension-stimulation of the abundant non-stretchable sensory nerve fibrils. This causes exaggerated afferent and antidromic neural responses that result in pain, referred pain, inflammation, sciatica, dysfunction and neurovascular dystrophy (decalcification). A vicious circle.

The diagnosis by trigger point tenderness of specific ligaments is confirmed by needling.

Treatment by induced recalcification of weak ligament attachments to bone is successful.

Experiments on rabbit tendons in conjunction with clinical observations were useful in determining the proliferating solutions and dosage to be used in prolotherapy.

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