PROLOTHERAPY FOR HEADACHE

Pain in the Head and Neck, and Neuritis

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INTRODUCTION . . .

Automobile accidents and other forms of violence and injury are resulting in an ever-increasing number of head and neck complaints, not only pain and headache, but vertigo, tinnitus, visual difficulties and many vasomotor symptoms. We should scrutinize every fact, theory or idea that might enlighten us as to the pathology, pathophysiology, management and treatment of these patients. This article crystalizes a lifetime of work in this field by Dr. Hackett, the pioneer in prolotherapy. This is now being published to provide a basic understanding of prolotherapy and its definitive approach to the treatment of head and neck complaints. This subject will be one of the principal topics presented at our Annual Meeting this June. Prolotherapy can best be learned by personal observation and instruction by one skilled in its use.

THE ASSOCIATION of headache and hemicranial pain in the head and face with homolateral suboccipital pain and cervical muscle spasm has long been recognized,[1-11] and nerve tracts connecting them have been identified.[12-15] Frequently the origin of noxious neural stimulation has not been identified and for a similar group of symptoms medical treatment has been directed to intracranial origin, while treatment in the cervical area has been directed toward interrupting the transmission of excessive neural impulses by somatic and sympathetic nerve block,[12-14] and by various manipulations and operations that either severed nerves or were aimed at releasing nerves from nocious stimulating infringements as interpreted from radiograms.

This article is confined to those cases in which the disability is due to overstimulation of cervical spinal nerves[16] within weak attachments of ligaments and tendons to the occipital bone and cervical vertebra.[7-11] The diagnosis is invariably confirmed, and skilled treatment by Prolotherapy is permanently successful.[7-11,17]

Recent scientific interpretation of the devastating effects of excessive antidromic impulses and their clinical application are described.

ANTIDROMIC IMPULSES

Excessive antidromic impulses from tension-overstimulation of afferent sensory nerve fibrils within weak attachments of ligaments and tendons to bone is a most important factor in post-traumatic neuritis

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and decalcification (dystrophy),[7, 9, 20-25] and, in association with sympathetic impulse overstimulation from the same source, there is an accompanying dysfunction of the neural, vascular and visceral systems and lowering of the pain threshold.[4,7,16,26-28] Investigation of antidromic impulses in afferent nerves from overstimulation has been under investigation since 1874 by Goltz, Stickler, Bayliss, Gaskell, Langley, Foerster, Hinsey, Gasser, de Takats, Lewis and many others as summarized and documented with noteworthy contributions by Wolff and Associates.[16,44] Wall[29] recently revealed that antidromic impulses within afferent nerves follow an orthodromic volley and "occur both in fibers carrying the orthodromic volley and in their passive neighbors" and that "repetitive firing is probably driven by bombardments from other neurous." The application of these scientific developments to our clinical observations[30] is supported by discussions with authorities in physiology[19] and neurology.[2]

LIGAMENT AND TENDON RELAXATION

The fibro-osseous attachments of ligaments and tendons to bone are weakened when the fibers do not become firmly attached following sprain,[7,12,17,31] continued strain and disruption, and by decalcification (osteoporosis) of disease, gonadotropic disturbances and the aging process.[1,24] It occurs frequently in the occipto-cervical area following the so-called "whiplash" injury, in occupations where the subject sits erect with head far forward for long periods as in writing or typing on a low table, reading in bed or slouched in a chair watching television, by continued cradling the telephone receiver between the ear and "cocked up" shoulder while using both hands at the desk, or in strenuous unaccustomed activities. It is often aggravated by one of these following a previous disability.

When the ligament/tendon attachments to bone become weakened the fibers yield under tension and permit tension - overstimulation[16-19] of the afferent sensory nerve fibers and tension receptors. To this noxious stimulation of afferent nerves the response is the origin (Fig. 1-0) of excessive afferent and antidromic impulses within the afferent nerve (A). Excessive afferent impulses (A) are transmitted to the spinal cord and to the brain as pain (P) and referred pain (RP).

Decalcification - excessive antidromic impulses (AN.AX) are transmitted to blood vessels in the bony attachment and stimulate a release of excess neurohumoral substance[16,32] which induces a neurovascular vasodilation edema that interferes with osteogenesis while osteoclysis continues. It is an interference with bone metabolism (dystrophy) in which decalcification (DD) further weakens the fibro-osseous attachment and increases the disability[20,21,23,25] in intensity and area such as occurs within the attachments of tendon fibers to the occipital bone.[7-11]

Inflamation Neuritis - other antidromic impulses are transmitted to blood vessels in nerves and surrounding tissues stimulating a release of excess neurohumoral mediator substance which causes a neurovascular vasodilation-edema-sterile inflammation-neuritis,[16,44] as in spinal nerves emerging through weak attachments of ligament/tendon to vertebra. Wolff assured me their interpretations of antidromic reactions in soft tissue are applicable to bone and that the antidromic impulses extend throughout the length of the neurones as in an extremety. This accounts for the pain and tenderness of inflamed nerves we find in the arm with neuritis originating at the cervical vertebra[7-9] and in the thigh and leg with sciatic neuritis originating in the 4, 5 lumbar vertebra and pelvis in sacroiliac-piriformis-sciatica.[7,25]

Sympathetic and autonomic imbalance - excessive reflex sympathetic impulses in response to afferent cervical nerve overstimulation cause a disturbance of the sympathetic and autonomic nervous and vascular systems which regulate cerebral and cervical organs and glands. In addition to disturbances of special sense organs, eye, ear and taste on a neurogenic basis, there are endocrine disturbances such as thyroid, parathyroid and the hy-

pothalmic - pituitary-adrenal pathway and all contributing to emotional stress.[32-36]

Muscle spasm results from barrages of reflex motor impulses (M) and adds additional afferent tension-stimulation at the weak tendon attachment during the sustained effort of muscle to relieve the nocious stimulation that actually causes it, thus defeating its own purpose and contributing additional stress[32-36] to the distraught patient.

OCCIPITO-CERVICAL LIGAMENT AND TENDON DISABILITY

The afferent sensory nerve supply within tendon attachments to bone in the occupital area is especially abundant and delicately sensitive because of the many involuntary and voluntary movements of the head in various directions[5] in response to stimuli from sight, sound, balance and volition. Afferent branches of 1, 2 cervical spinal nerves supply cervical muscle and tendon attachments to the occipital bone beneath the superior nuchial line.

Kimmels[37,38] excellent reports on the fetal development of cervical spinal nerves reveal dural branches of C1, 2, 3 give off branches to soft tissue (ligaments, tendons, muscle) before entering the foramen magnum to join blood vessels with sympathetic fibers which connect with cranial nerves. Branches of C1, 2 enter the cranial cavity through the hypoglossal canal and jugular foramen along with sympathetic nerves from the superior cervical ganglion. That would include branches from spinal nerves below C 3 which also innervate ligament/tendon attachments to their respective vertebra. Kerr[39,40] discussed the convergence of C 1, 2 roots and the cranial fifth nerve in post-traumatic suboccipital headache and atypical facial neuralgies. With C 1, 2 nerves exposed for rhizotomy[41]

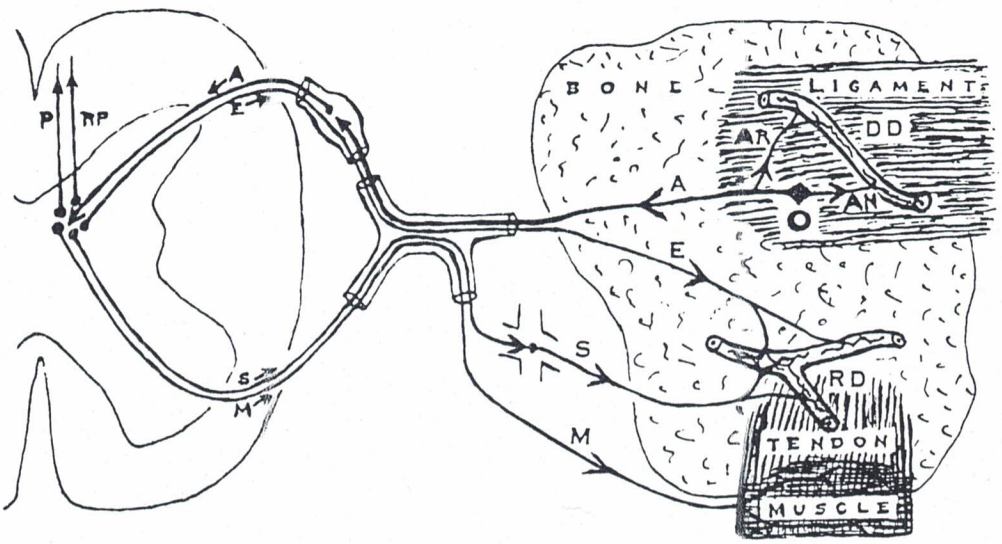

Fig. 1 Noxious stimulation of exaggerated afferent neural response in weak fibro-osseous attachments of ligament to bone.

Barrages of impulses **originate** (O) in afferent somatic sensory nerve (A) when weak ligament fibers yield under normal tension and permit **tension-overstimulation** of nerve fibrils within the weak fibro-osseious attachment to bone. Excess afferent impulses (A) are transmitted to the spinal cord and (P, RP) to the brain where they are interpreted as **pain** and **referred pain.**

Excess **antidromic impulses** within the afferent nerve are transmitted directly (AN) and by axon reflex (AR) to bone blood vessels and cause a neurovascular vasodilation - edema decalcification (DD) in the area of ligament attachment, while excess reflex **sympathetic** (S) impulses to bone blood vessels cause **decalcification** (RD) in larger areas of bone, and weaken the attachments of additional ligaments and tendons, (a vicious circle).

Excess antidromic and sympathetic impulses are transmitted to nerves and surrounding tissue and cause a **vasodilation-edema-sterile inflammation-neuritis.**

Excess **sympathetic** impulses cause an imbalance of the neural, vascular, antonomic nervous and endocrine systems.

Muscle spasm results from excess reflex motor (M) impulses as a protective measure, but stimulates additional afferent and antidromic impulses within its tendon attachment to bone.

C 1 was identified to have 3 rootlets with small dorsal-root ganglion. Stimulation of each rootlet separately by "a Grass Stimulator" or by "gentle pressure or traction with bayonet forceps was found to be equally effective." "When stimulated, the uppermost rootlet of C 1 evoked pain in the orbit; the mid rootlet, pain in the frontal area: and the lowest, pain in the vertex," with minor variations in the 2 subsequent patients. Kerr quotes Gray, Poinier and Charpy that in 8 per cent of cases C 1 dorsal root is absent.

Clinical observations - our clinical investigation of pain in head, face and headache associated with suboccipital pain and cervical muscle spasm have identified the disability to be weak fibro-osseous attachments of tendon fibers of posterior cervical muscles to the occipital bone beneath the superior nuchial line.[7-11] The reactions have been manifested by local suboccipital pain and trigger point tenderness, headache and homolateral pain in head and face, neuritis, nasal congestion, pain deep in the eye, in the teeth and ear, disturbances of taste and mild dizziness[7-11] An illustration (Fig. 2) of our observations designate the location and size of those head and face areas of pain, which when present are consistently related to specific trigger tender points on the occipital bone as the origin of noxious stimulation.

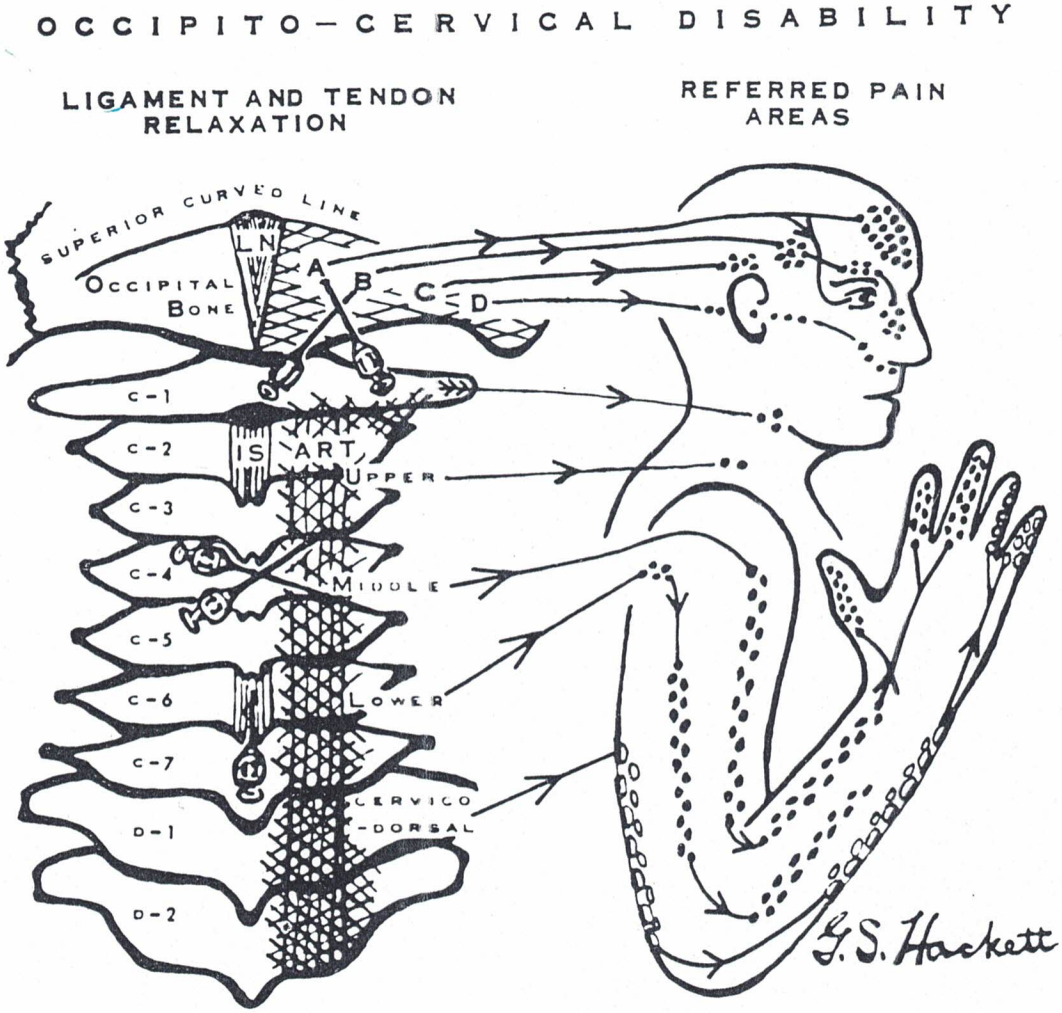

Fig. 2. Pain in the head, face and headache have their origin in weak fibro-osseous attachments of tendons to the occipital bone (A, B, C, D) when weak fibers yield under normal tension and permit tension-overstimulation of cervical spinal sensory nerves. Pain in the arm, hand and neuritis are from weak attachments of ligaments and tendons to the cervical and upper dorsal vertebrae.

Each pain area is almost always constant for a specific location of origin in all individuals, and is extremely valuable in diagnosis.

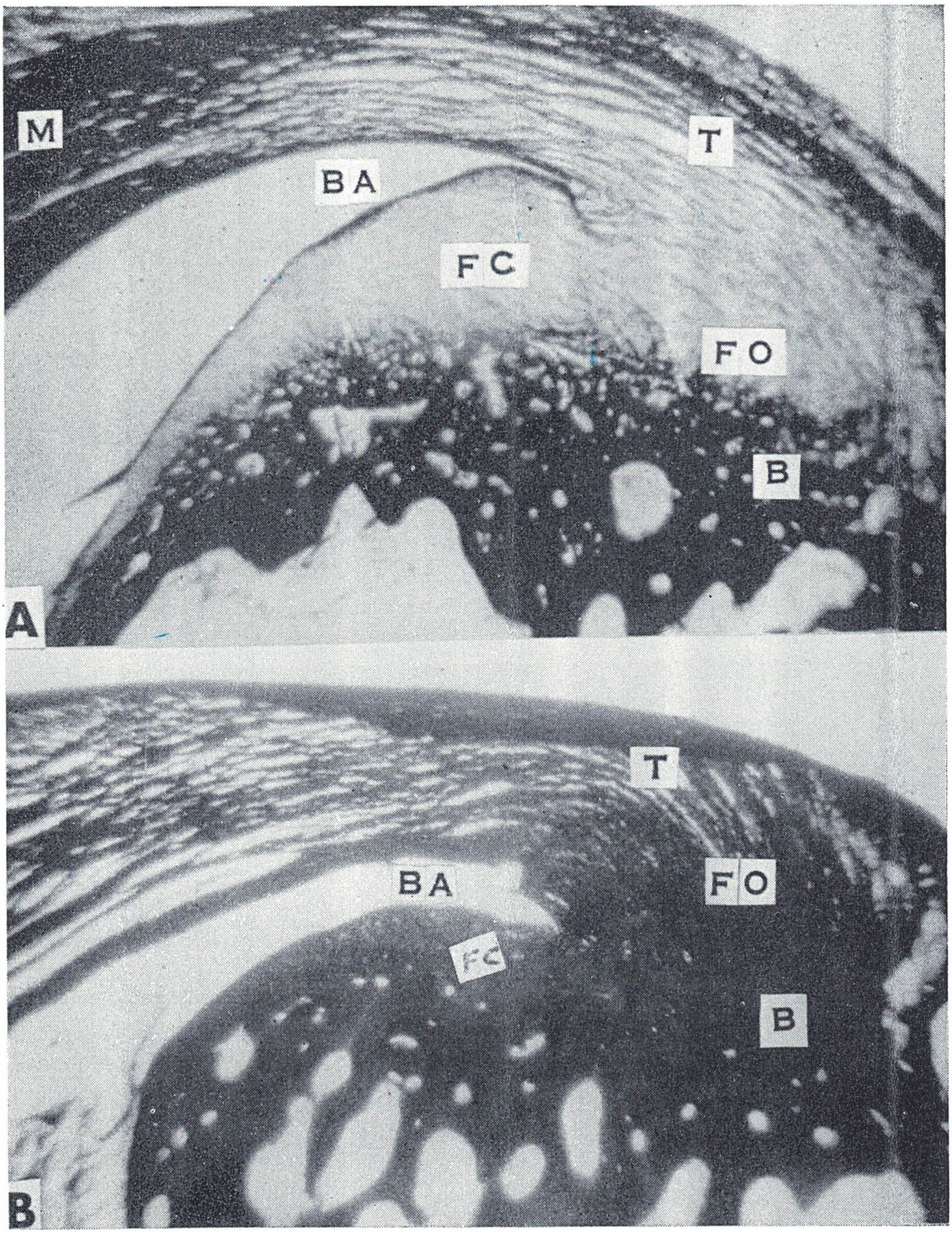

Fig. 3. Microphotographs of Induced Fibro-osseous Proliferation. Bone-B, Tendon-T, Muscle-M, Fibro-osseous junction-FO, Fibro-cartilage-FC, Bursal Area-BA.

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 fibro-osseous conjunction is strengthened.

Example - Case No. 37 in my monograph[7] is an excellent example of specific diagnosis and therapy. In this case severe suboccipital pain in the neck with trigger point tenderness was associated with homolateral pain in the eye and usually in the forehead, a phenomena reported by many observers.[5-14, 39-41] We have consistently identified the origin of this disability to occur by tension-overstimulation of the sensory nerve fibrils within weak fibro-osseous attachments of the semispinalis capitus tendon fibers to the occipital bone in the area limited to 1 inch in diameter, (A) the center being 1 inch below the superior nuchial line and one inch from the midline of the neck. Without exception when the neck and eye pain coexist, the patient affirms local tenderness obtained by deep palpation with the thumb, and pain by fibro-osseous contact with a **hypodermic needle** which is intensified by injecting any solution. Both eye and forehead pain are frequently reproduced by these examining tests. The neck, eye and forehead pain are invariably abolished by injecting a local anesthetic solution within the tendon attachment and all pain is permanently abolished by prolotherapy which strengthens the weak tendon attachment to bone.[9] Usually the eye pain does not return after the first treatment although it may require two or three treatments to abolish the local suboccipital pain and tenderness. Lancinating pain such as would accompany piercing the occipital nerve has not been observed. In severe cases the eye pain may be accentuated by reading and a bright light accompanied by lacrimation.[5,9,13] Severe relaxed tendon attachments of the outer portion of the semispinalis capitus (B) and rectus capitus muscles to the occipital bone have an associated homolateral circular area of pain in the temple two inches in diameter, a linear area just above the eyebrow hair line and on the upper half of the nose, and nasal congestion,[5] during severe exacerbations. Tendon relaxation lateral at (C) is associated with pain above the ear, while just medial to the mastoid (D) is associated with pain back of the ear, on the anterior wall of the external meatus,[5,9] in front of the ear, on the cheek lateral to the nares and on the upper half of the upper lip. It is also associated with pain in the **teeth** and **disorder of taste.**[5,9]

Exception - occasionally there have been very severe cases with marked tenderness on the occipital bone at (A) and (B) in which there was no associated pain in the eye, but pain in the temple, eyebrow and nose was severe. This probably occurs when no part of the semi-spinalis tendon is innervated by C 1 upper rootlet, which apparently is the only division of C 1, 2 nerves that has a sympathetic nerve connection with the fibers of the fifth cranial nerve that are considered to be involved in eye pain.[41]

CERVICO-DORSAL LIGAMENT AND TENDON RELAXATION

The strong interspinous ligament is important in stabilizing the vertebrae and when weakened permits increased movement and strain on the articular ligaments and pressure on the intervertebral disc.[7] Troup's[42] excellent contribution emphasizes the segmental nerve and blood supply to the intervertebral tissues of ligaments and disc. The features of any joint sprain are "loss of mechanical function, capillary dilitation and oedema, and a source of reflex nerve irritability," "limited to the tissues of one intervertebral segment." This is in accord with our observations over a period of twenty-three years. The interspinous ligament has no identifiable referred pain areas, but must be strengthened when weak because vasodilation edema[4,9,16,20-28,42] interferes with normal physiological rehabilitation.

Weak ligament/tendon attachments to the transverse, articular and spinous processes and lamina of vertebrae throughout the spine are the origin of excess afferent, antidromic and reflex sympathetic impulses from overstimulation of afferent fibers of spinal nerves. Sympathetic nerve fibers from the third cervical and each lower segmental level make up the sympathetic cord from which cervical and upper dorsal branches enter the cranium, have functional connections with cranial nerves and participate in control of the

neurovascular autonomic system. From weak attachments of C 1 vertebra there is an associated pain in the pharynx deep beneath the angle of the jaw (Fig. 2). From cervical and upper dorsal spinal nerve tension-overstimulation there are specific referred pain areas and neuritis in the arm and digits. Similar referred pain and neuritis in the lower extremety from 4, 5 lumbar and pelvic ligament/tendon relaxation have recently been clarified in an article on sciatica.[25]

We have clinically observed the development of lowered threshold in cervical neuritis extending into the arm as described experimentally by Wolff and Associates.[4,16,26-28] With the cervical area and shoulder girdle at rest, activity causing tense forearm muscles greatly increased the load of impulses in the inflamed nerve plexus and aggravated the pain. "Grasping" and maneuvering a small needle in sewing "lighted up" the pain, while knitting with a larger needle held "gently" without finger tenseness during simple manipulation did not.

DIAGNOSIS

A good history will: reveal local pain which the patient identifies with one finger; and referred pain areas which direct attention to specific ligament/tendon attachments to bone that the examiner identifies by trigger point tenderness with his thumb.[7-9] The physical examination is made with the patient seated astride a chair with hands on back of chair. To relax neck muscles the head is supported by the examiner's left hand. If reclining have two pillows lengthwise beneath chest while forehead rests on mattress, with neck flexed to straighten the vertebra and expose the suboccipital area. With the right thumb the examiner locates trigger tenderness on the occipital bone beneath the superior nuchial line (Fig. 2 - A, B, C, D) and on the certical veterbrae. In palpating, go from normal to painful areas and ask the patient "is that it," or "does that ring a bell." The diagnosis is invariably confirmed by intratendonous needling with a local anesthetic solution. The needle reproduces the trigger point pain with intensity and sometimes the referred pain, while both disappear within two minutes following the anesthetic injection.

TREATMENT

Treatment by Prolotherapy (L. Proles, new cells)[7] consists in injecting a proliferating solution combined with a local anesthetic solution within the weak fibro-osseous attachment of ligament/tendon to bone. It stimulates the proliferation of new bone and fibrous tissue cells which permanently strengthen the attachment (Fig. 3) Technic consits in using a 22 gage Luerlock 2 inch needle attached to a 10 cc. syringe containing the combined proliferating-anesthetic solution. After inserting the needle to contact bone within the fibro-osseous attachment, 5 drops are injected, the needle is withdrawn sufficiently to redirect without bending and recontact bone at one-half inch distance. Usually three to eight injections may be made during one insertion of the needle depending upon the size of the area. Treatment is given while patient reclines on the abdomen (see diagnosis). They are usually given in the office, except severe and nervous patients are treated in the hospital. New bone and fibrous tissue develop strength over a few weeks. The patient returns for report and re-examination in one month. Additional treatment is given if indicated.

A dextrose proliferating solution gives adequate fibro-osseous proliferation with a minimum of discomfort and can be prepared by any pharmacist. It is self-sterilizing.

DEXTROSE STOCK SOLUTION

Dextrose BP	25.0%
Phenol BP	2.5%
Glycerine BP	25.0%
Dist. Water to	100 cc.

It should be labeled - "Mix before injecting" and placed in 100 cc bottles (rubber stoppered). Prior to injection it should be mixed 1 part dextrose stock solution and 3 parts tetracaine (Pontocaine) 0.15% or lidocaine (Xylocaine) 1.0%. 50 cc. of this dilution has been given in multiple bone contact injections of 1/3 cc. each (4-

5 drops). Proliferating solutions of zinc sulfate and sylnasol should be diluted to half strength of previous recommended dosage[7,9] for comfort.

Medication - analgesics and sedatives are given before treatment to allay anxiety and add comfort. Following treatment analgesics are given for comfort as needed for several days. In severe cases, early administration of estrogen, androgen and vitamin C (such as Formatrix-Ayerst), and thyroid 1 to 2 grains daily, accelerate the formation of bone matrix and give a feeling of well being.

Prosthesis - a cervical collar relieves muscle spasm and pain in severe cases during activity following treatment. Traction is without value except to limit activity.

STATISTICS

During a 21-year period 1857 patients with ligament/tendon disability were treated by Prolotherapy in our clinic and hospital. The technic of diagnosis and treatment was improved and extended from the low back to the occiput and into the extremeties[7] in collaboration with our colleagues[30] to include several thousand patients in which a high degree of success continues. Good to excellent results were reported by 90% of eighty-two consecutive patients with occipito-cervical disability treated by Prolotherapy during the past four years. There were no unfavorable sequelae. Unsatisfactory results may be attributed to inadequate diagnosis, insufficient treatment in extent of area and follow up, post-treatment activity and co-existing disability.

COMMENT

Recent important contributions on excessive antidromci impulses[4,6,16,27-29,33] and in embryoanatomy[37,38,42] when applied clinically, clarify the origin and development of neuritis and decalification as they occur throughout the vertebral column from weak attachments of ligament and tendon to bone. The associated effect of antidromic and sympathetic impulse overstimulation clarify some visceral and endocrine disturbances with which we have been engaged for half a century.[43]

SUMMARY

Headache and pain in the head and face when associated with suboccipital pain in the neck very often results from weak attachments of ligaments and tendons to the occipital bone and cervical vertebrae, which permit tension-overstimulation of the sensory nerve fibrils. This results in excess afferent and antidromic impulses with pain, referred pain, neuritis and bone dystrophy. The associated excess sympathetic impulses cause disturbances of the neural, vascular and endocrine systems and stress.

The diagnosis is confirmed by needling, and conservative treatment by Prolotherapy is indicated.

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