CASE REPORT

Treating Whiplash-Associated Disorders with Intramuscular Stimulation: A Retrospective Review of 43 Patients with Long-Term Follow-Up

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ABSTRACT. Background: Trauma to the neck can cause acute pain. Chronic pain and dysfunction from such injury is called "whiplash-associated disorder" [WAD]. The Quebec Task Force [QTF] classified WAD severity. This case study summarizes the courses of 50 WAD patients [class 2 or 3].

Findings: Forty-three accepted intramuscular stimulation therapy. They had pain and abnormal physical signs [allodynia, trophedema, muscle knots, limited range of motion]. With treatment, twenty-nine achieved long-term improvement.

Conclusions: The symptoms were due to abnormal function of the central nervous system. Most improved subjetively and their abnormal physical signs resolved. Such direct clinical evidence of benefit is clearly meaningful.

KEYWORDS. Whiplash, litigation, intramuscular stimulation, spine, neuropathic pain, radiculopathic pain.

INTRODUCTION

The most common cause of whiplash-associated disorders [WADs] is the rear-end automobile collision, but sporting injuries are also common. A whiplash injury can cause a wide spectrum of soft tissue damage, ranging from minor ligamentous strain, to major disruptions of stabilizing tissues in the neck and entire spine. The injury is often associated with high costs and a prognosis that is variable and difficult to predict. Patients not infrequently develop chronic symptoms, with their initiating incident typically having occurred months or even years ago (1-5). A Quebec study in 1987 with a seven year follow-up showed the median recovery time was 32 days, and 12% of subjects still had not recovered after six months (6). The conclusion was that longer recovery was associated with several specific musculoskeletal and neurological signs and symptoms. They proposed a working clinical classification:

Grade 0 No complaints about the neck. No physical signs
1 Neck complaints of pain, stiffness or tenderness only. No physical sign[s]

2 Neck complaints and musculoskeletal sign[s]
3 Neck complaints and neurological sign[s]
4 Neck complaints and fracture or dislocation

According to the Quebec Task Force [QTF], in a mild injury [Grade 1], symptoms are relatively insignificant and subside quickly. In WADs of moderate severity [Grade 2], symptoms generally include pain in the neck, often in the upper trapezius, on one or both sides, and there may be pain in the arms. Muscle spasm was identified by palpation and/or by limited range of motion. X-rays may show a loss of cervical lordosis. In Grade 3, there are also neurological signs. In extremely severe injuries [Grade 4], there may even be rupture of tissue in front of the vertebral column–such as the esophagus and trachea–and tear of supporting ligaments in the spine.

REVIEW OBJECTIVES

In the year September 1, 1997 to September 1, 1998, 1,021 patients were accepted for treatment at the Institute for the Study and Treatment of Pain [ISTOP] in Vancouver, Canada. Of these, 50 were suffering from WADs. They were reviewed to determine if there was a common cause for pain to persist. Following examination and assessment, 43 accepted intramuscular stimulation [IMS] treatment [Table 1]. All our patients belonged to Grades 2-3, except for one Grade 4 who sustained damage to a vertebra and disc.

HOW PAIN CAN BECOME CHRONIC

The International Association for the Study of Pain underscores tissue damage in its definition of pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described by the patient in terms of such damage," but pain need not be linked causally to injury. Injury does not always generate pain, nor does pain always signal injury.

Wall saw pain as a reaction pattern of three sequential behavioral phases: immediate, acute, and chronic (7). Each phase may exist independently, or in any combination and proportion with the others. The immediate nociceptive phase is usually transient, unless there is tissue

TABLE 1. Whiplash Review–July 15, 2000

Fifty WAD patients were examined. Seven were assessed but not treated

	Patient	Sex	Age	Clinical Findings	Treatment [Tx]
1.	BA	F	53	Neuropathic Signs	No
2.	PM	M	42	Neuropathic Signs	No
3.	HM	F	39	Neuropathic Signs	No
4.	TB	F	44	Neuropathic Signs	No
5.	SC	F	44	Neuropathic Signs	No
6.	SD	F	47	Neuropathic Signs	No
7.	AR	F	24	No neuropathic Signs	No

Of the Remaining forty-three, six had less than five treatments and were discounted

	Patient	Sex	Age	Response	# of Treatments
1.	FF	F	44	No change	2
2.	WC	F	30	No change	4
3.	HF	F	67	No change	3
4.	BP	M	46	Worse	3
5.	AB	F	63	Other problems	5
6.	DR	M	44	No change	5

Patients with more than five treatments but no improvement

	Patient	Sex	Age	Response	# of Tx	Date of Last Treatment	Follow Up [weeks] Since Last Tx	Total Disability Period [weeks]	Notes
1.	BT	M	62	No Improvement	31	10/Jul/98	112	168	
2.	AW	F	38	No Improvement	18	25/Mar/98	129	155	Severe depression
3.	SS	M	41	Minimal Improvement	18	10/Sep/97	160	237	Improvement not maintained

Patients improved at time of discharge–No long-term follow up

	Patient	Sex	Age	Response	# of Tx	Date of last Tx	Follow Up [weeks] Since Last Tx	Total Disability Period [weeks]	Notes
1.	SL	M	39	Improved	2	27/Aug/97	145	391	Unable to contact
2.	VH	F	54	Improved	13	3/Oct/98	127	205	Unable to contact
3.	MW	M	41	Improved	7	31/Oct/97	133	233	Unable to contact
4.	WG	M	68	Improved	8	28/Jul/97	158	184	Unable to contact
5.	PM	M	29	Improving	16	16/Mar/98	129	231	Lost contact

Twenty-nine patients with Improvement and long-term follow up

	Patient	Sex	Age	Response	# of Tx	Date of last Tx	Follow Up [weeks] Since Last Tx	Total Disability Period [weeks]	Notes
1.	KG	F	43	Good	13	21/Aug/97	142	200	Back to original activities
2.	HR	F	53	Good	10	14/Jan/98	149	208	Back to work
3.	FR	M	51	Good	38	8/Apr/99	61	131	Much better
4.	AP	F	62	Good	8	14/Aug/97	130	230	Much better, improved mobility
5.	BR	F	52	Good	31	7/Sep/99	159	285	Less pain, increased flexibility
6.	SD	M	66	Good	42	18/Jan/99	118	170	Multiple complaints resolved
7.	CC	F	39	Good	5	29/Jun/98	104	282	Back to work
8.	ES	F	48	Good	11	4/Dec/97	140	276	Better
9.	WC	M	50	Good	4	28/Aug/97	148	252	Less pain
10.	CG	F	52	Good	4	9/Nov/98	84	152	Pain free
11.	PG	M	42	Good	14	1/Dec/98	95	231	Much better, headache is gone
12.	AY	F	35	Good	14	9/Sep/97	164	220	Feels "perfect"
13.	SG	F	34	Good	17	21/May/99	93	283	Back to work
14.	KA	F	67	Good	30	28/Jun/99	102	105	Better, on monthly maintenance
15.	RJ	F	41	Good	27	25/Jun/98	129	177	Good
16.	MS	F	29	Good	27	23/Sep/99	113	223	Needs maintenance
17.	SS	F	35	Good	8	16/Jan/98	128	232	Feels better
18.	SR	F	43	Good	12	18/Jun/98	156	162	Feels perfect
19.	BC	F	50	Good	2	28/Aug/98	92	196	Back to work
21.	CG	M	69	Good	16	29/Jun/99	93	98	Less pain
22.	CC	M	50	Good	2	23/Jun/97	135	265	Better
23.	AV	F	28	Good	4	12/May/97	156	278	Much better
24.	DS	F	54	Good	34	18/Nov/99	132	182	Still needs maintenance
25.	WE	M	53	Good	9	20/May/98	115	163	Better
26.	S	M	30	Some Improvement	7	29/Oct/98	97	196	Better than before
27.	WR	F	50	Good	19	9/Apr/98	134	248	Some improvement
28.	LV	F	35	Some Improvement	14	7/Apr/98	127	399	Discharged
29.	ES	M	49	Some Improvement	5	17/Jan/99	78	216	Discharged

WAD = Whiplash associated disorder

injury and damaged cells release algogenic substances [such as histamine, bradykinin, serotonin, prostaglandins] to produce inflammatory pain. Anti-inflammatory medications are commonly used in this phase, but it can be counter-productive to completely abolish inflammation, as inflammation is the necessary prelude to healing. No medications are prescribed at ISTOP.

After injury, most people heal rapidly and become pain-free, but in some, pain persists beyond the usual time for the healing process and becomes intractable. Chronic pain can occur if there is:

1. Ongoing nociception or inflammation,
2. A psychosomatic disturbance such as a somatization disorder or depression, or
3. A functional disorder in the nervous system.

Categories one and two are usually recognized, but three is often missed. Our hypothesis is that peripheral neuropathy following WADs is a common functional disorder and it can often cause pain. Because the neuropathic condition is not always understood, the pain it causes is frequently undiagnosed.

While it has been accepted that pain can follow gross nerve injury, the effects of peripheral neuropathy are of recent concern. The term "neuropathic pain," which has been introduced to describe pain associated with peripheral neuropathy (8), has now been expanded to include any acute or chronic pain syndrome in which the mechanism that sustains it is inferred to involve aberrant somatosensory processing in the peripheral nervous system or central nervous system. "Radiculopathic pain" is probably more accurate, as peripheral neuropathy commonly stems from spondylosis where the spinal nerve root is subjected to irritation from pressure, stretch, angulation, and friction (9).

Ordinarily, spondylosis is degenerative. It follows a gradual, relapsing, and remitting course that is silent, unless symptoms are precipitated by an incident, often so minor that it passes unnoticed. All gradations of spondylosis exist, including incipient spondylosis, which is prone to injury. Peripheral neuropathy is not exceptional in adults: in a previous study, early and subtle signs of peripheral neuropathy were found in a significant number of young [under 30 years], apparently normal, and symptom-free individuals (10).

In this study, our emphasis has been on radiculopathic pain because nociception or inflammation pain usually resolves within days or weeks and none of our patients showed signs of these. Psychologic factors

were present only in one [a recent immigrant who was desperately homesick, severely depressed, and whom we were unable to help]. Specifically, we wished to determine if whiplash associated disorders are radiculopathic manifestations of traumatic spondylosis, and if so, do they respond to dry-needling (11,12).

SIGNS OF RADICULOPATHY– A SUMMARY OF WHAT WE LOOKED FOR

Whiplash associated disorder is puzzling because symptoms often persist in the absence of detectable injury or inflammation. However, there are specific features of radiculopathy that a specially trained examiner can detect. A skilled examiner can also clinically identify any irritated segment[s].

The history gives little assistance and the degree of reported pain often far exceeds that consistent with the injury. Laboratory and radiologic investigations are generally not helpful; consequently, we do not routinely order x-rays. Thermography reveals decreased skin temperature in affected dermatomes and this can be an indication of neuropathy, but does not necessarily signify pain. Electromyography is not specific either. Diagnosis of pain and dysfunction caused by radiculopathy therefore depends almost entirely on the examiner's clinical experience and acumen and a knowledge of the segmental nerve supply to muscles is necessary.

Every patient was given a standard examination designed to detect neuropathic signs (13). The manifestations of radiculopathic dysfunction, particularly muscle tenderness and shortening, palpable muscle knots, restricted joint range, and autonomic epiphenomena were carefully recorded [Figures 1, 2 and 3]:

Motor–

- The laws of physics determine that large diameter nerve fibers are the earliest to be affected–axons of motoneurons and myelinated primary afferents [muscle proprioceptors] at the nerve root. Motor manifestations are therefore the first to appear, and *early radiculopathy is without pain.* Neuropathic changes are most prominent in muscle; even when symptoms appear in joints or tendons, signs in muscles are the most consistent and relevant. Each muscle must

be palpated, including paraspinal muscles that extend through most of the length of the spine.

- Transient, tight, but painless muscle knots can be felt in most individuals, even in toddlers. The knots, caused by muscle shortening, as normal muscle contraction does not usually produce pain [Classic contracture refers to the evoked shortening of a muscle fiber in the absence of action potentials.] (14). Muscle knots become excessively tender in neuropathy when they respond painfully to a stimulus that is not normally noxious-allodynia. Allodynia is described as "deep and aching," and the sensation.
- The shortened muscle can also generate pain by physically pulling upon sensitive structures, e.g., on tendons, to produce "tendonitis" (15).
- Muscle shortening is inherent in neuropathic allodynia and any physical stimulus that restores muscle to its normal length concurrently relieves neuropathic "pain."

Sensory–

- Neuropathic allodynia can occur without tissue-damage. Nociceptive pain is not a feature of radiculopathy unless A-delta or C-fiber pathways are involved, for example: when there is a wound or inflammation at the same time. Many neuropathies are painfree, such as sudomotor hyperactivity and muscle weakness in ventral root disease.
- There is often a delay in onset of pain following the precipitating injury as it may take several days for hypersensitivity to develop.
- There can be severe pain in response to a noxious stimulus–hyperalgesia.
- The pain can be neuralgic–paroxysmal brief "shooting or stabbing" pain.
- There is pronounced summation and after-reaction with repetitive stimuli.

Autonomic–

- Heightened pilomotor response ["goosebumps"],
- Increased sudomotor response–hyperhidrosis,
- Increased vasomotor response. Vasoconstriction differentiates neuropathic pain from inflammation pain: in neuropathic pain, affected parts are perceptibly colder.

- Trophedema: Increased permeability in blood vessels can lead to local subcutaneous tissue edema–"neurogenic edema" or "trophedema." This can be seen as "peau d'orange" skin [Figure 4] and confirmed by the "matchstick" test [Figure 5]. Trophedema is nonpitting to digital pressure, but when a blunt instrument such as the end of a matchstick is used, the indentation produced is clear-cut and persists for many minutes. This quick and simple test can demonstrate neuropathy earlier than electromyography.
- Dermatomal hair loss,
- Collagen degradation: Neuropathy degrades the quality of collagen, causing it to be frailer than normal collagen (16). The amount of collagen in soft and skeletal tissues is also reduced. Because collagen gives strength to ligament, tendon, cartilage, and bone, neuropathy expedites degeneration in weight-bearing and activity-stressed parts of the body–these can become a source of pain. Collagen weakness in tendons can lead to thickening–enthesopathy.

BREAKDOWN OF PATIENTS REVIEWED

Age: 29-69 years. The average age was 46.2 years.

Sex: 26 Female, 17 Male.

Weeks disabled: Two were admitted in the first month following the initiating accident. One was admitted after six weeks. The rest were admitted after 26 to 272 weeks.

Previous accidents: It was the first accident for 36 patients, 11 had one previous accident, and 3 had two accidents.

Previous treatments: [Most patients received more than one modality]

Physiotherapy–28 patients
Remedial exercises–4 patients
Massage–20 patients
Chiropractic adjustments–17 patients
Acupuncture–10 patients
Surgery–1 [laminectomy]
Cortisone injections–1 patient
Naturopathy–1 patient
Medications only–3 patients

FIGURE 1. Sample physical examination chart part 1.

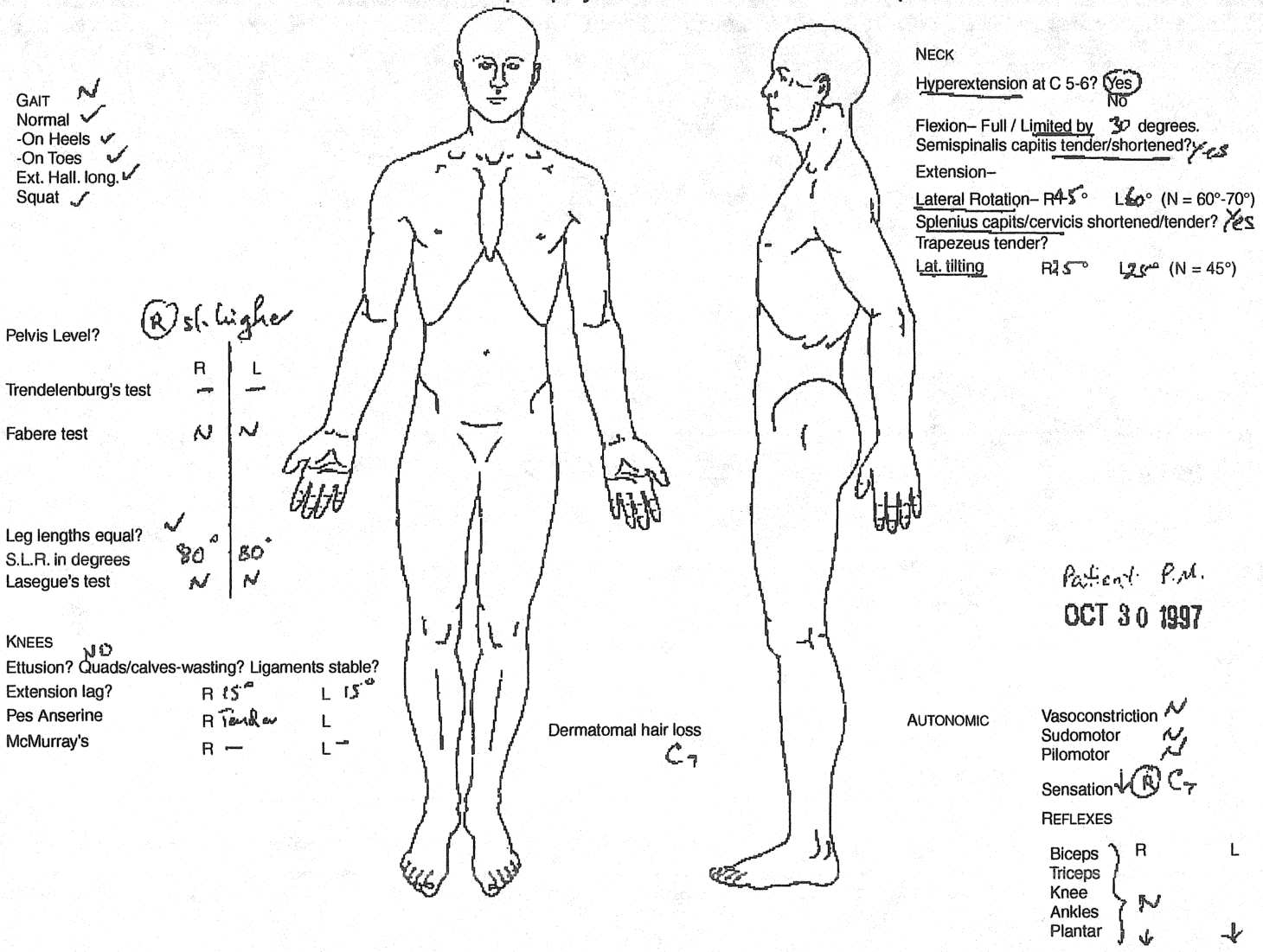


FIGURE 2. Sample physical examination chart part 2.

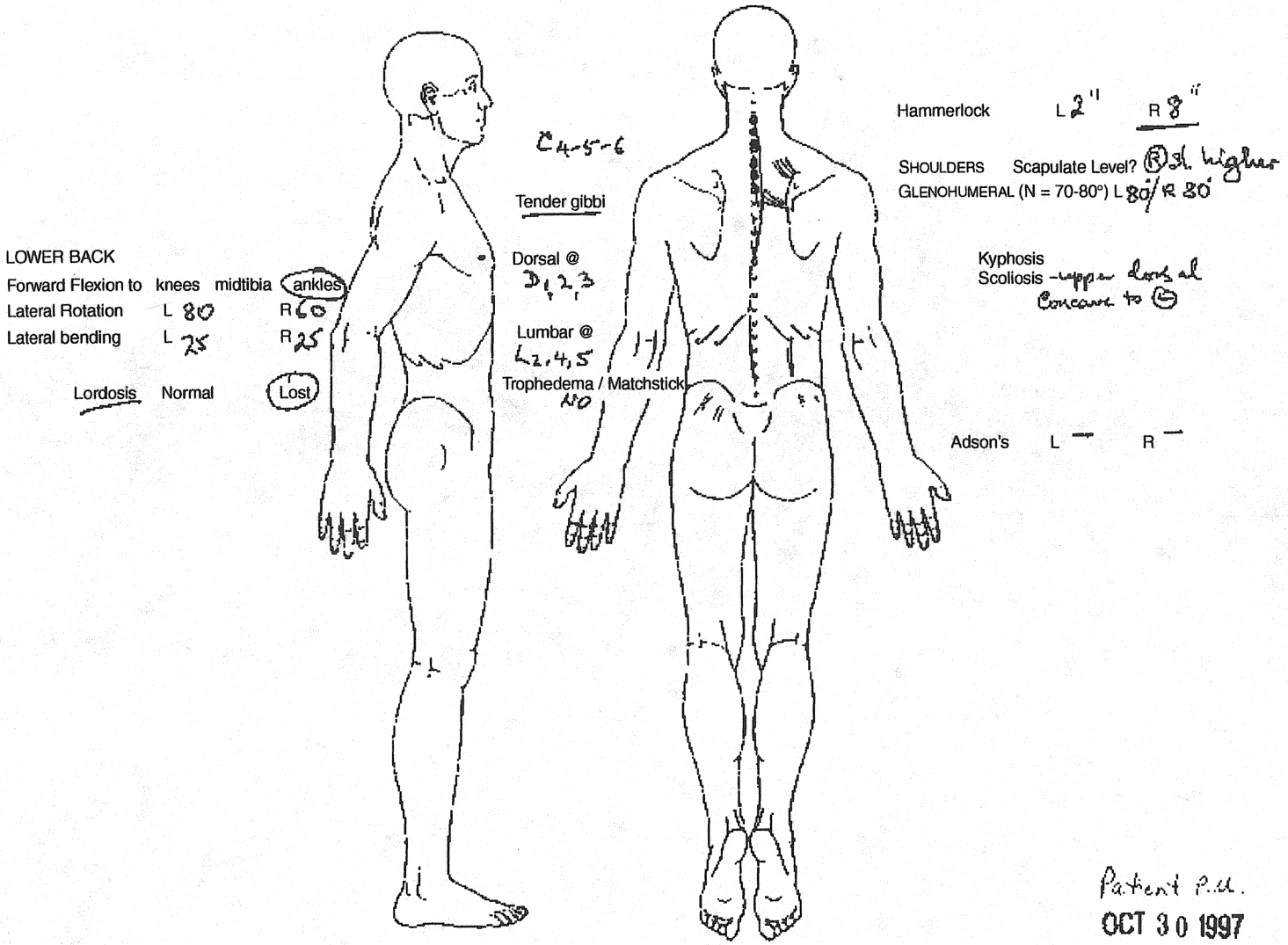


FIGURE 3. Tender motor points at examination and first treatment.

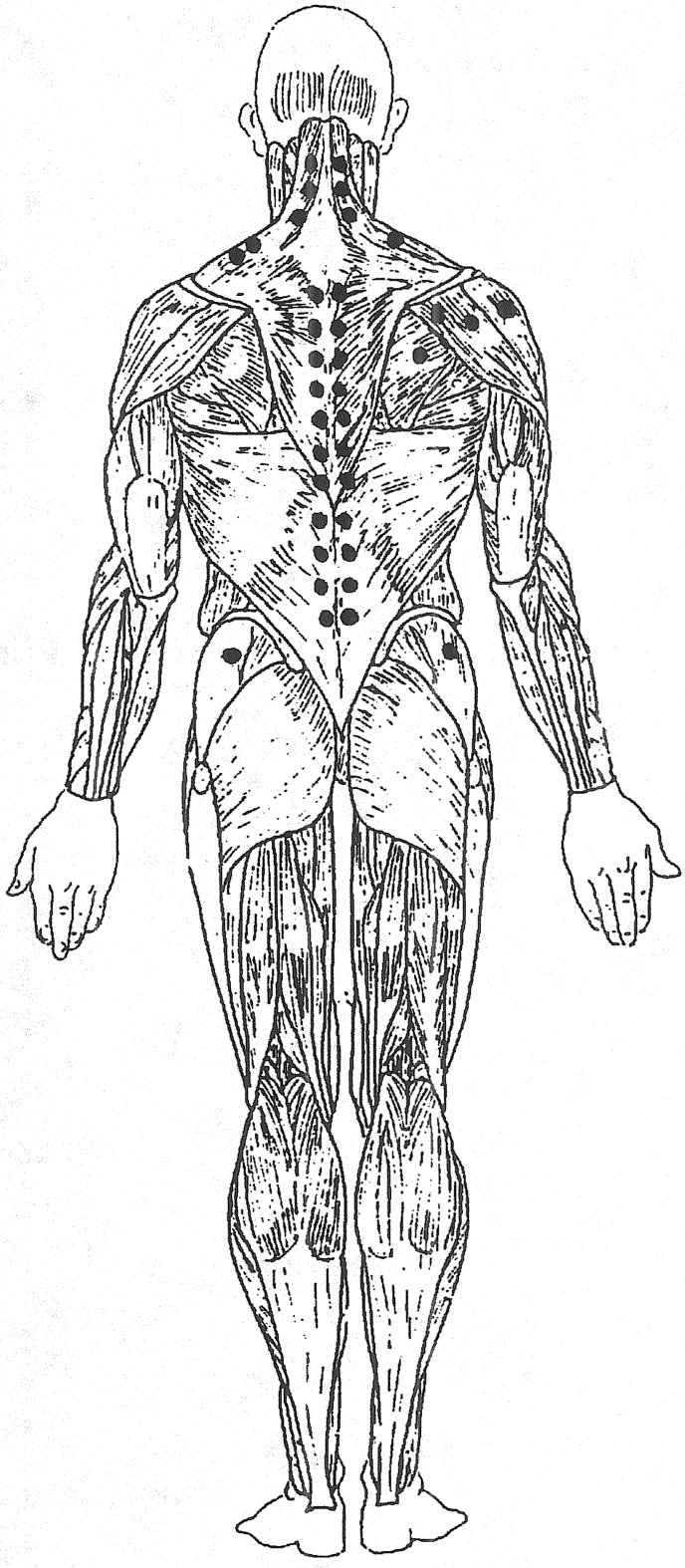


FIGURE 4. Example of trophedema as "peau d' orange" skin.

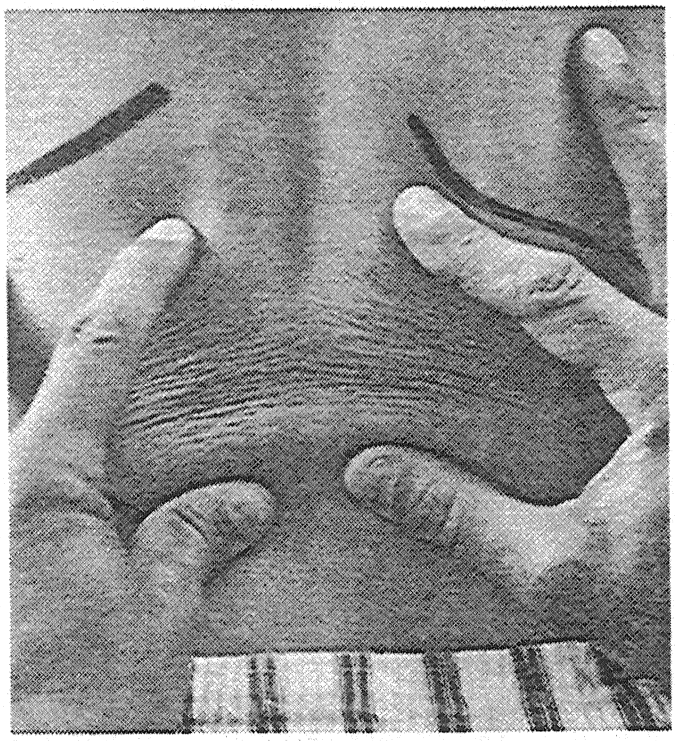
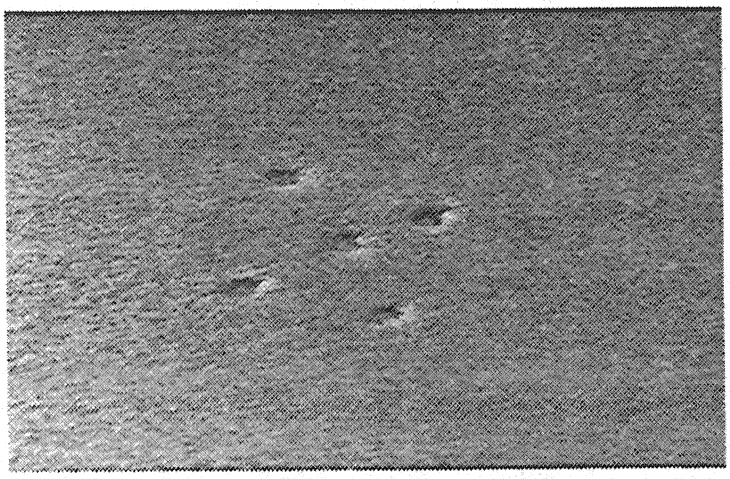


FIGURE 5. The "matchstick test" shows trophedema as lasting indentations.



Location of pain: All patients had musculoskeletal complaints that involved the spine. All had signs of neuropathy, with the exception of the one with psychosomatic problems, and another in whom we were unable to find any significant neurological signs. Many patients had symptoms at more than one level:

Neck Pain, 38 patients [76%]
Shoulder and arm pain, 37 patients [74%]. Note that neck and shoulder percentages are similar, as they belong to the same segments.
Low back pain, 32 patients [64%]
Dorsal back pain, 27 patients [54%]
Pain extending to the leg, 20 patients [40%]
Headache, 22 patients–occiput and vertex [44%]
Face and jaw pain, 3 patients [6%]–Voice was hoarse in 2.

Our findings show that WADs can affect the entire spine. For example, pain in the leg is almost as common as headache. Any examination or treatment of WADs, must therefore include every segment of the body.

RESULTS

Fifty consecutive WAD patients were examined. Forty-three accepted treatment and seven declined [Table 1]. Six [13.9%] of the forty-three were lost to the study as they had less than five treatments.

Of the remaining thirty-seven, thirty-four improved [79%] but three [6.9%] did not. The thirty-seven included five who had expressed improvement at discharge, but we were unable to reach for long-term follow-up.

Follow up: All patients were followed up for over one year–minimum 61 weeks, and a maximum of 164 weeks [average 128 weeks]. At the time of review, three patients still required occasional "booster" maintenance treatments.

NEUROPATHIC PAIN IS CAUSED BY INCREASED SENSITIVITY

The probable mechanisms in neuropathic pain are different from those in nociception and inflammation. Neuropathic pain is not caused by the arrival of noxious A-delta and C-fiber signals but by misperception and misinterpretation of ordinarily non-noxious signals.

Following damage to a peripheral nerve, e.g., irritation of a nerve root by spondylosis, or repetitive C fiber nociceptor input, e.g., from tissue inflammation [as in arthritis], interactions occur between peripheral and central mechanisms to produce post injury hypersensitivity (17-19).

In *peripheral* sensitization, the sensitivity of peripheral receptors is greatly increased and nociceptors become overly sensitive.

In *central* sensitization, a state of hyperexcitability occurs in the dorsal horn neuron. Central sensitization leads to a cascade of molecular events, such as activation of the N-methyl-D-aspartate [NMDA] channel, increase in intracellular calcium, wind-up/wide dynamic range neuron sensitization and other phenomena. There can be spontaneous activity of dorsal horn neurons, exaggerated response to afferent input, expansion of receptive field size, reduction in threshold, and prolonged after discharges. Together, these cause the spinal cord to modify or amplify incoming signals. Low intensity stimulation can be perceived as painful mechanical allodynia, and high-intensity stimulation, which is normally painful, sensed as hyperalgesia.

PHYSICAL THERAPY AND STIMULATION-INDUCED ANALGESIA

A critical review of controlled clinical trials for peripheral neuropathic pain had concluded that the pharmacologic management of neuropathic pain is very difficult. Overall there were no long-term data to support the analgesic effectiveness of any drug (20).

Although it has been suggested that the effect of stimulation on neuropathic pain and allodynia is due to inhibition of glutamate and aspartate release at NDMA receptor sites and activation of local gamma aminobutyric acid mechanisms (21), neuropathic pain has been shown to be a supersensitivity phenomenon and its treatment therefore requires desensitization. Animal experiments have shown that supersensitivity can be reversed by electric stimulation (22).

Physical therapy, which is widely used as treatment for neuropathic pain, likewise achieves its effect by stimulation. Physical therapy excites receptors [in skin and muscle]. For example, massage activates tactile and pressure receptors; exercise, manipulation, and dry needling stimulate muscle spindles and Golgi organs; heat and cold act on thermal receptors. The physical stimuli are sensed by their specific receptors, transduced into nerve impulses and relayed to the dorsal horn. All forms of physical therapy, including dry needling and acupuncture, are effective only when the nerve to the painful part is still intact, allowing reflex stimulation to release muscle shortening. The release provided by reflex stimulation can spread throughout the entire myotome.

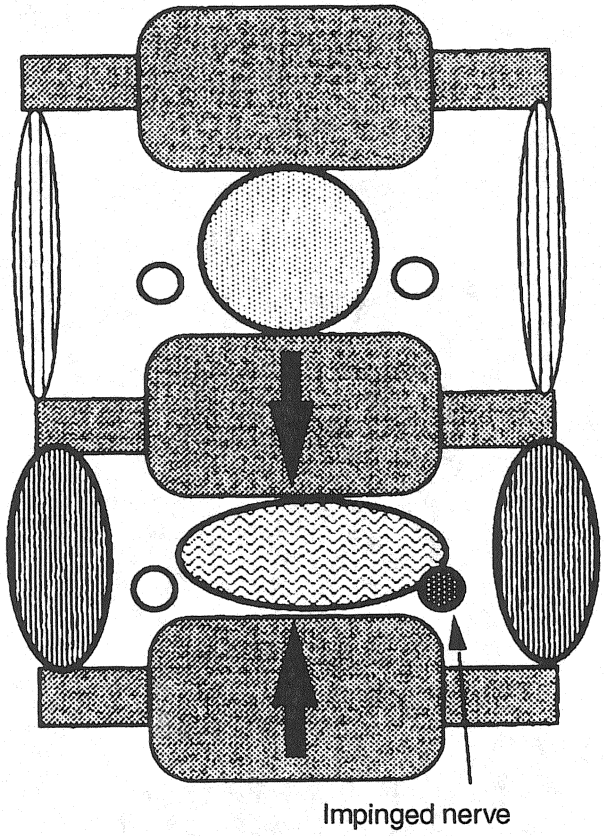
Sicne 1985, the Multidisciplinary Pain Center at the University of Washington has used a dry-needling technique to treat myofascial pain syndromes (23). We have called our technique intramuscular stimulation to distinguish it from classical acupuncture. We have found IMS to be an efficient method of stimulation having several distinct features. The needle is able to penetrate and release deep contractures. Stimulation lasts for several days, for as long as the current of injury persists. Intramuscular stimulation is also unique in that it promotes healing by the release of the platelet derived growth factor (24). For IMS to be effective, it is necessary that diagnosis, treatment, as well as progress be monitored and administered according to observable physical signs of neuropathy (9).

REMOVING THE CAUSE OF NEUROPATHY IS KEY TO TREATMENT

Spondylosis is, by far, the most common cause of radiculopathy, and treatment should be aimed at relieving the cause of impingement or entrapment of the nerve root [Figure 6]. Release of muscle shortening is the key to relief: local treatment, starting with simple measures such as massage, and if necessary, escalating to more effective modalities such as dry needling, should be used. Treatment should be given to all tender and shortened muscles in affected myotome[s], especially paraspinal muscles. The outcome depends on the modality used, and the skill of the therapist.

Effective points are usually situated at motor points or musculotendinous junctions. These points often coincide with acupuncture points and typically belong to the same segmental levels as the injury. Intramuscular stimulation can release a shortened muscle within minutes, or even seconds. The fine, flexible acupuncture needle used is a unique tool for finding and releasing contracture. Contractures are invisible to x-rays and can only be discovered by probing with a needle. The needle transmits feedback information on the nature and consistency of the tissues it is penetrating. When penetrating normal muscle, it meets with little hindrance; when penetrating a contracture there is firm resistance, and the needle is grasped by the muscle. This causes the patient to feel a peculiar, cramp-like sensation, referred to as the Deqi response. The Deqi response is an important finding as it confirms neuropathy: neuropathic pain has the Deqi, and nociceptive pain does not. This distinction is important because of the difference in nature and

FIGURE 6. In spondylosis muscle shortening causes entrapment of the nerve root.

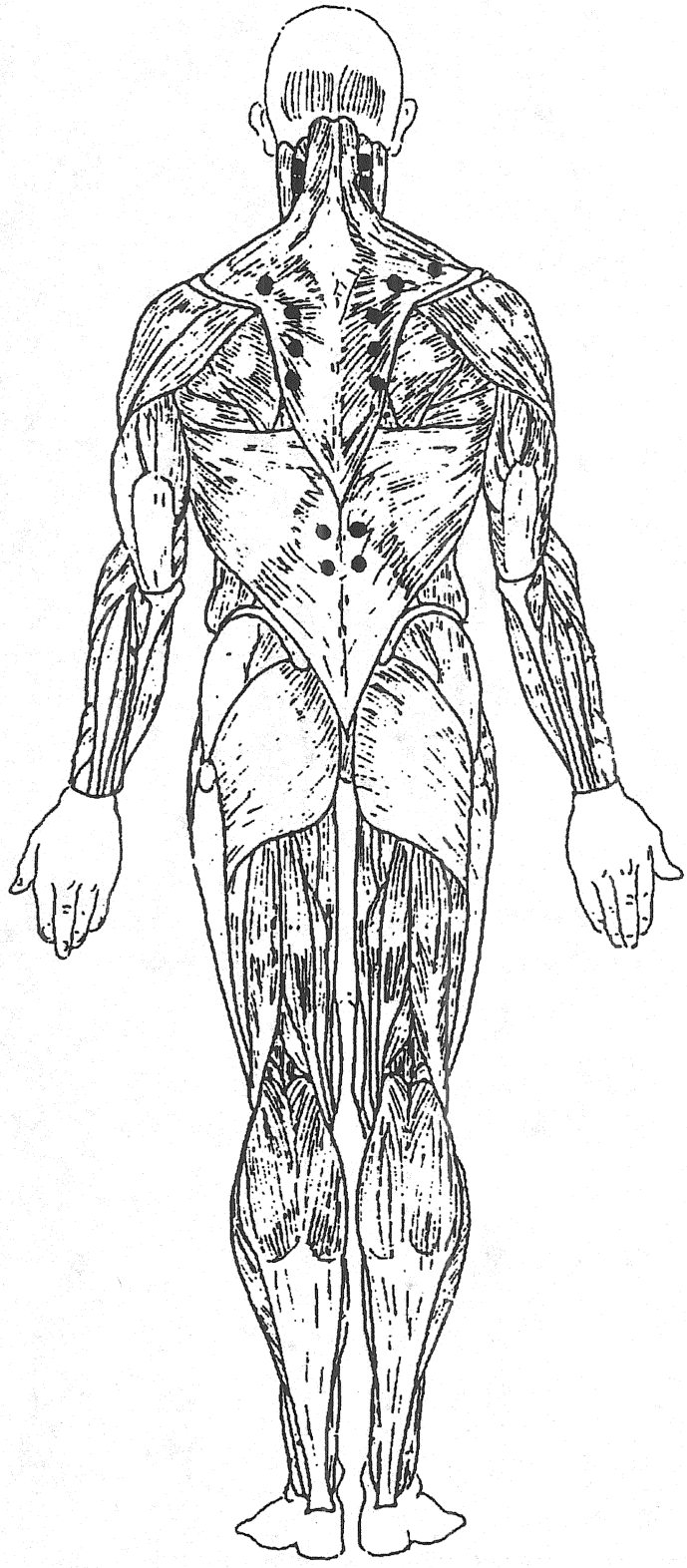


treatment of the two pains. Neuropathic pain, unlike nociceptive pain, is associated with proprioceptors that sense muscle length (25).

EVIDENCE-BASED RESULTS DEPEND ON THE RESOLUTION OF NEUROPATHIC SIGNS

The primary cause of pain in this series of WAD patients was not from ongoing nociception or inflammation, but from abnormal function in the nervous system. Treatment was for observed manifestations of spondylitic radiculopathy. Although we were guided by the patient's subjective report, the diagnosis, dry needling, and progress were primarily based on clinical signs of pathology. As the condition resolves, we require all evidence of neuropathy, such as trophedema, mechanical allodynia, palpable muscle knots, and limited joint range to disappear [Figure 7]. In our experience, direct evidence is more meaningful than randomized trials.

FIGURE 7. Tender motor points treated during patient's final visit.



In the QTF Clinical Classification, Grade I was designated as having no physical signs. However, stiffness results from muscle shortening [releasable by needling]; pain and tenderness result from mechanical allodynia [not nociception]. Therefore, QTF Grades 1 and 2 belong together as a neuropathic category.

It is encouraging that treatment, even when long-delayed, could still produce relief. For example, the patient who was admitted 272 weeks after the precipitating incident was discharged with significant improvement, and at her two year follow-up remained well.

Some health care providers believe that the incidence and prognosis of whiplash injury are related to eligibility for compensation. A recent publication stated, "The elimination of compensation for pain and suffering is associated with a decreased incidence and improved prognosis of whiplash injury" (26). However, the true incidence of injury is related to the actual number of patients with pathology. The accepted number of compensation claims generally bears no relation to true incidence, as the underlying condition may be missed, and claims are susceptible to claimant expectations or administrative manipulation to limit expenditures.

And as for prognosis: all our patients were either receiving compensation or undergoing litigation. Nevertheless, the majority improved when given correct diagnosis and effectual treatment, regardless of the status of their claims.

In this study, virtually all the patients with WAD showed manifestations of radiculopathy that appeared from traumatic spondylosis. Most of them responded favorably to IMS dry needling (11,12). The number of patients in our pilot study is small, but it suggests that an urgent reconsideration of the usual medical examination and treatment approach is necessary. Pain of WAD is usually a treatable *functional* disorder, yet most physical examinations look for nonexistent or nonrelevant structural changes.

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