Utilizing trigger points

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The trigger mechanism in association with myofascial pin syndromes has long been acknowledged as a common problem in medical practice.^{1,2}

This syndrome is characterized by the existence of a small hypersensitive area—the "trigger point" or "region" connecting with a related "target" or "reference" area of pain. The trigger point is described as a small circumscribed hypersensitive area in muscle or in connective tissue from which impulses arise and produce referred pain. The resulting severe and disabling pain has caused confusion in

the diagnosis of shoulder, neck and low back disorders. When the pain occurs in chest muscles, it has been mistaken for effort angina and myocardial infarction.³

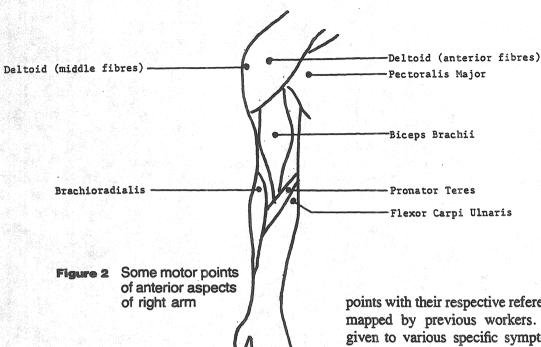
Although the peculiar symptomatology has been recognized, the true nature of the syndrome has not yet been defined. There is very little information regarding the exact histology of a trigger point or the neurophysiological mechanism of the syndrome.

We will briefly summarize existing concepts and add our observations from three recent studies. A neurophysiological mechanism is postulated.

Current concepts The exact etiology of the syndrom is not known. Symptoms often follow acute trauma but may also Trapezius (upper fibres)-Supraspinatus Deltoid(posterior fibres) Deltoid (middle fibres) Triceps (long head) Triceps (lateral head) (Brachioradialis) Triceps (medial head) Extensor Carpi Radialis Longus & Supinator Brevis Extensor Digitorum Extensor Carpi Ulnaris Extensor Digiti Minimi Extensor Pollicis Longus & Brevis Adductor Pollicis 1st Dorsal Interosseus

Figure 1 Some motor points of the posterior aspect of right arm

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follow trauma of no consequence or develop insidiously. Among the many precipitating factors blamed for its occurrence are "arthritis", poor posture, general fatigue, chilling of muscles, low metabolic rate, creatinuria, visceral ischemia, dyskinesia, chronic infection and even psychogenic stress.^{1,2}

The symptomatology is well known. Stimulatin of a hypersensitive trigger point leads to tenderness, pain and muscles spasm in the area of reference. Spasm and resistance to stretching may give rise to apparent shortening of affected muscles with limitation of motion. The referred pain is dull and aching in character and varies from mild discomfort to severe disabling distress. The reference area, though generally constant and predictable, has been confusing as it does not appear to conform to a dermatomal pattern or nerve root distribution. Frequently, apart from the basic or "essential" reference area of pain, there is a "spill-over" zone where symptoms are less severe.

In addition to the above features, there may be autonomic effects such as abnormal vasomotor reaction, sudomotor hyperactivity and a pilimotor effect or cutis anserina.

The topographical distributions of many trigger

points with their respective reference areas have been mapped by previous workers. Names have been given to various specific symptom-complexes such as the "scapulocostal syndrome" ^{4,5}, levator scapulae syndrome, infraspinatus syndrome, fatigue-postural paradox, etc.²

Trigger points are not necessarily always hypersensitive. They may be dormant or subclinical until caused to flare up into the vicious circle of painspasm-pain by some stress factor. As a pragmatic approach to the problem, the injection of trigger points with local anesthetic or saline has long been in use. Transcutaneous neural stimulation has also been used with good results.

Material

Our observations and deductions are based on three recent clinical studies:

(I) An investigation of 50 patients having upper limb pain with "myalgic" points, 8 showed that these tender points occurred at muscle "motor points". It was demonstrated that these tender motor points represented the reflex localization of pain from radiculopathy secondary to cervical spondylosis. Clinical, radiological and electromyographic findings supported this. Treatment directed to the cervical spine gave relief.

This series of 50 patients with pain in the arm and labelled as "resistant tennis elbows" was referred by attending physicians to the Rehabilitation Clinic of the Workers' Compensation Board of British Columbia for management. They had defied the

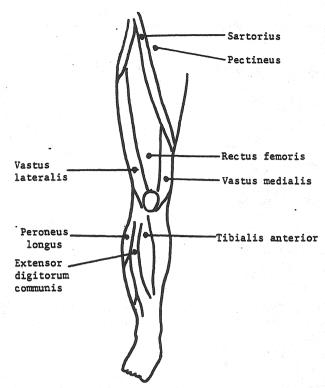


Figure 3 Some motor points on the anterior aspect of leg

usual conservative office measures, such as injections of steroids and local anesthetic, manipulation, ultrasound, friction massage, immobilization, etc. On examination, points of maximal tenderness were found (usually 4 or 5) at muscle motor points in the elbow region. These were confirmed as such by electrical stimulation. The motor points generally found were those of the brachioradialis, extensor carpi radialis, supinator, extensor digitorum and extensor carpi ulnaris, all these being closely situated in an area of about 2 inches in diameter. Other motor points with tenderness were also found in muscles of the same myotome(s). Frequently, both arms were involved. Roentgenograms of the cervical spine in 34 patients showed degenerative changes at involved levels. Forty-two patients given an electromyographic examination showed some abnormal E.M.G. findings of early neuropathy or radiculopathy in affected myotomes. Twenty-three of the 50 patients were given treatment directed to the cervical spine (mobilization or cervical traction) and had relief of symptoms within 4 weeks. The other 27 patients were given local elbow treatment (ultrasound, friction massage, ice, etc.) but their symptoms persisted. After 4 weeks, treatment was changed to that for the neck and this gave good response. Forty-four out of 47 patients who had responded to treatment were followed up at least 3 and 6 months after discharge and they had no further symptoms. These findings led us to conclude that the arm pain was related to the cervical spine and thus when treatment to the elbow failed, neck treatment was tried with good results. In many of these patients, some degree of cervical degeneration preceded the arm condition.

(II) A study of muscle tenderness in relation to low back pain in 200 subjects, ¹⁰ also showed that muscle tenderness, maximal at motor points, occurred in affected myotomes secondary to lumbar spondylosis. Again, clinical, radiological and electromyographic evidence supported the findings.

In 1974, total attendance for all types of injuries at the Workers' Compensation Board Rehabilitation Clinic number almost 5,000. Of these, 33% were for injuries to the lumbar spine. In the study, four subject groups were examined:

Group A: "Low back sprain"—50 consecutive patients.

Group B: "Disc degeneration"—50 consecutive patients.

Group C: (control) 50 normal individuals with no history of backache.

Group D: (control) 50 normal individuals, no previous medical treatment, but admitting to the occasional backache after unusual stress or activities. The study showed:

- (1) Tender motor points of a mild and transient nature may occasionally be found in normal, asymptomatic individuals, especially in those subjected to unusual activities.
- (2) Mild to moderately tender points are usually present in individuals with a history of a "vulnerable" back or those with clinical, radiological and E.M.G. evidence of spondylosis.
- (3) Moderately to acutely tender points are almost constantly found in patients admitted for "disc degeneration", the degree of tenderness and the number of tender points paralleling the patient's condition.
- (4) Tenderness in muscles of both anterior and posterior rami places the lesion at root level.
 - (5) An important finding of this study was that

patients admitted to the Clinic with "low back sprains" without tender points were disabled for an average of 6.9 weeks, yet, "low back sprains" with tender points (22.4 weeks) were almost as disabled as patients with radicular signs (25.7 weeks).

(III) An as yet unpublished study on shoulder pain referred from the cervical spine from spondylosis. It is well recognized that shoulder pain may emanate from the cervical spine; yet this pain often goes misdiagnosed. Pain in the shoulder may be the only presenting symptom of cervical disc degeneration since the presenting symptom of lesions in the cervical spine is seldom pain in the neck but commonly pain in the shoulder region.

In the fourteen months from November 1974 through January 1976, there was a total of 357 patients referred by attending physicians to the Clinic, as having primary shoulder pain. Of these, 50 patients were recognized, on admission, as having referred pain from the neck (see Tables I & II).

Roentgenograms of the shoulder and cervical spine (including lateral, in flexion and extension, anterior-posterior, open-mouth and oblique views) were taken and electromyography conducted on all patients (C.C.G.).

Thirty-seven of the 50 patients were males and thirteen, females. Their average age was 41.84 years.

Roentgenograms of the shoulder joint were normal in all patients except two who had some degenerative changes. Cervical roentgenogram findings were normal in twenty-three patients and showed minor degenerative changes in ten and moderate to severe changes in seventeen. Electromyography revealed some degree of neuropathy in every patient including slight denervation in three. The levels of cervical involvement and neuropathy corresponded with the myotomes where tender motor points were found. The degree of tenderness at motor points and apophyseal joints paralleled the degree of neuropathy found. All patients responded to conservative treatment directed to the cervical spine.

Observations

(1) Tender "myalgic" points are found to be located at muscle motor points and correspond with described sites of "trigger points".

Table I
Spinal cord root derivations of motor nerves supplying some arm & shoulder muscles

Muscle	Root innervation	Peripheral nerve					
Trapezius (upper)	C3-4						
	00.45	accessory					
Levator scapulae	C3-4-5	C3-4 and Dorsal scapular					
Supraspinatus	C5-6	Suprascap.					
Infraspinatus	C5						
Deltoid	C5-6	Axillary					
Biceps brachii	C5-6	Musculo- cutaneous					
Pectoralis major	C5-6	Anterior					
		thoracic					
Supinator.	C5-6	Radial					
Ext. carpi radialis	C5-6-7	Radial					
Ext. carpi ulnaris	C6-7-8	99					
Ext. dig. communis	29	99 99					
Triceps brach.	99						
Felx. carpi ulnaris	C7-8, D1	Ulnar					
Add. poll. brevis	C8, D1	 					

Most muscles receive their innervation from more than one segment of the spinal cord, as indicated in this table. The segments listed are those generally accepted as the predominant source of innervation of the muscles in question, all of which are innervated by the anterior rami whose fibres pass along the nerves indicated on the right. The posterior rami from these same cord segments supplies skin and muscles of the back of the neck (semispinalis capitis and cervicis, splenius capitis and cervicis).

In our studies, mild to moderate neuropathy was found to be an early product of spondylosis, that is, the skeletal changes resulting from degeneration of the intervertebral disc. Clinically, neuropathy was reflected in motor, sensory and autonomic findings in the affected myotomes, dermatomes and sclerotomes. In involved myotomes, muscles innervated

Table II

Tender motor points in 10 selected patients with referred shoulder pain

Patient	Age,	Age, Diagnosis on Tender motor points*					•	Tender								
	Sex	referral	Tz	Ss	Is	D	Р	Bi	Ext	Sup	TRI	FC	APB		apophys. joints	
G.D.	29M	Contusion to shoulder.	1	1	1	2			3 1	1		1	3		C6-7	
G.K.	24M	Fell on shoulder.	2	2	1		2								C5-6	
P.S.	46M	Pull on shoulder.		3	2	3	3								C5-6	
A.W.	44F	Contusion to shoulder & elbow.	2	2	2	2			2	2		2			C4-6	
R.P.	48M	Pulled shoulder.				3					3				C3-7	
A.P.	64M	Fell on shoulder.	2	3		3			3	2	•				C4-6	
R.S.J.	32M	Acute shoulder & arm strain.	3	3	2	2			3	3			1		C3-6	
U.L.	33F	Acute shoulder strain.	2	2	2	2			2	2		2			C5-6	
c.c.	36M	99	3	3	3	3		3	2	2					C4-6	
P.E.	34M	Struck on shoulder.	3	3		3	2								C4-5	

by compromised nerve roots were tender to digital pressure, especially at the region of the motor point. The locations of these motor points are noted to correspond with previously described sites of "trigger points". (See Figures 1-6).

Motor points have been in use for many years for the electrical stimulation of innervated muscles and are known to any physiotherapist. Charts showing their locations are generally available.¹¹

Tenderness graded 1-3 (see text).

*Tz=Trapezius

Ss=Supraspinatus

Is=Infraspinatus

D=Deltoid

P=Pectoralis Major

Bi=Biceps Brachii

Ext=Extensor Common Origin

Sup=Supinator

TRI=Triceps

FC=Flexor Carpi Ulnaris

APB=Adductor Pollicis Brevis

LS=Levator Scapula

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Roentgenograms (cervical spine)	Roentgenograms (shoulder)	E.M.G. (nerve root) Mild neuropathy C7-8				
Normal.	Normal.					
Normal.	Normal.	Slight fall of amplitude & frequency C6.				
Degen. C5-6.	Normal.	Moderate neuropathy C5-6.				
Slight limit'n of motion C5-7.	77	Mild neuropathy C4-5.				
Severe narrow'g C3-7.	99	Moderate neuropathy C4-7.				
Mild degen'n with slight	99	Moderate neuropathy C4-6.				
narrowing C5-6. Normal.	. 53	Moderate neuropathy C3-4 & 7.				
"		Mild C.5-6 neuro- pathy.				
,,		Mod. C4-6 neuro-				
Slight roto- scoliosis.	"	Mod. C4-6 neuro- pathy.				

A motor point is defined as the skin region where an innervated muscle is most accessible to percutaneous electrical excitation at the lowest intensity. It generally lies close to the point at which the nerve enters the muscle, the neurovascular hilus, 12 and approximates but is not identical to the muscle zone of innervation. Here, terminal branches of the nerve lie nearer the skin surface. The exact location of a motor point may vary from person to person, but

it is a fixed anatomic site.

Tender "myalgic" points in both studies were demonstrated to be located at motor points where muscle twitches were evoked in response to minimum electrical stimulation with a pluse generator/chronaxie meter.

(2) The degree of tenderness is variable.

Tenderness at motor points varied from mild to severe and in our studies was graded as:

Grade 0—No tenderness whatsoever to firm digital pressure (normal).

Grade 1—Patient is aware of some tenderness but it is not unpleasant.

Grade 2—Tenderness is present and unpleasant.

Grade 3—Acute tenderness; the patient is often surprised and the response is vigorous (latent "trigger point").

Grade 4—When tenderness and pain are self-evident, persistent and associated with referred pain—a "trigger point" results.

(3) Tender points rarely occur singly.

It was rare to find an isolated tender point. There may be only one symptomatic or "herald" point, but a diligent search over known motor point locations generally revealed others where pain and tenderness of a similar nature could be produced, though of a lesser degree. For example, in the first study of 50 patients presenting as "lateral epicondylitis", herald points found at the elbow were usually of the supinator, brachioradialis, extensor digitorum longus, extensor carpi radialis and extensor carpi ulnaris, but other tender motor points (see Fig. 6) always within the same myotome(s) which could be elicited included pectoralis, biceps, deltoid and supraspinatus. (See Tables I & II.)

(4) Relationship to spine.

Noting that these tender points occurred in muscles having common nerve root origins (C5, C6), we also examined the spine at these levels. In the cervical spine, slight limitation of lateral rotation and tilting towards the affected side was usually found, and the apophyseal joints of affected levels were always tender to digital pressure and showed resistance to passive movements.¹³ Simi-

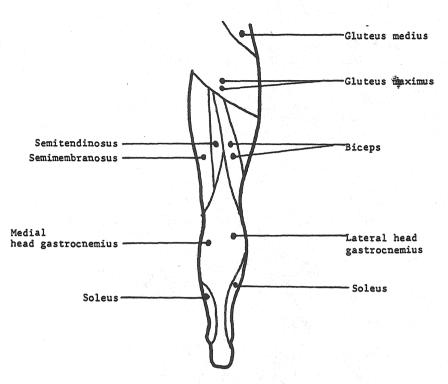


Figure 4 Some motor points on the posterior aspect of leg

larly, in the lower limbs tender motor points were found in relation to low back degenerative disease and sometimes associated with clinical signs, e.g. straight leg raising restriction, etc.

(5) Presence of nerve root neuropathy.

X-rays of the spine frequently showed degenerative changes commensurate with age in the affected level(s) and electromyography in almost all patients showed evidence of mild to moderate neuropathy of related nerve roots. Muscles demonstrating E.M.G. changes always belonged to the same myotome(s).

According to Buchtal & Pinelli, findings of neuropathy include: Increased needle insertion activity or a relative increase of mechanically induced discharge of action potentials by electrode movement; with voluntary contraction, the mean duration of action potentials becomes prolonged but amplitude may be normal or reduced; typically, polyphasic potentials appear in abnormal numbers, the interference pattern is reduced or in severe cases lost altogether and the action potentials of individual units could be identified even during maximal contraction. These E.M.G. abnormalities, typical of neuropathy, are not generally overwhelmingly obvi-

ous but present as subtle changes from the normal state. Fasciculation potentials, positive sharp waves and fibrillation potentials develop only when denervation is present. It is fallacious to accept the occurrence of fibrillation potentials as the only criteria for establishing the diagnosis of neuropathy. Fibrillatin potentials are late arrivals if they are to appear at all. 14,21

(6) Tender points resolved following treatment directed to the affected spinal levels.

In all patients, if and when symptoms were relieved following treatment to the affected spinal levels (conservative or surgical), the tender points disappeared and E.M.G. findings reverted to normal.

(7) Intact nerve root essential for trigger reflex mechanism.

When the innervating nerve root is disrupted, the trigger reflex mechanism is absent.

It was observed in a patient with tears to the fifth and sixth cervical roots that a pilimotor effect could be induced in the entire limb by pressure over the tender motor points, excepting the areas supplied by C5 and C6 over the deltoid and radial surfaces of the forearm and hand (axillary and musculocutaneous). Injection of local anesthetic around the muscle nerve supply can also abolish the trigger reflex.

Neurophysiology

To explain the multiphasic nature of the myofascial syndrome concepts are reviewed.

The morphologic and functional unit of the nervous system is the neuron. From the functional point of view, each neuron acts as an integrator, conductor and transmitter of coded information. Neurons are organized in circuits. Although the simplest circuit is the monosynaptic chain, most are open or closed multisynaptic chains. Closed multisynaptic chains may form feedback circuits that permit the reverberation of impulses to raise or lower the excitability of the various neurons in the chain. Normally, feedback circuits operate against a background of tonic descending influences. These feedback circuits may be unbalanced in neuropathy.

The segmental nerve is a mixed nerve and includes motor, sensory and autonomic components.

Sensory fibres arise from nerve endings in a variety of somatic structures—cutaneous and subcutaneous tissues, tendons, joint tissues, periosteum and skeletal muscles. Recently, attention has been focused on cutaneous nociceptors and spinal neurons activated specifically by noxious stimuli. These cutaneous nociceptors have a powerful, excitatory action in the dorsal horn of the spinal cord on at least two kinds of dorsal horn neuron. These neurons normally are subjected to a selective inhibitory input from the mechanoreceptors, presumably through an internuncial pathway. ¹⁵

Primary autonomic neurons, from their origin in the central nervous system, synapse with the secondary neurons in the autonomic ganglia whence the

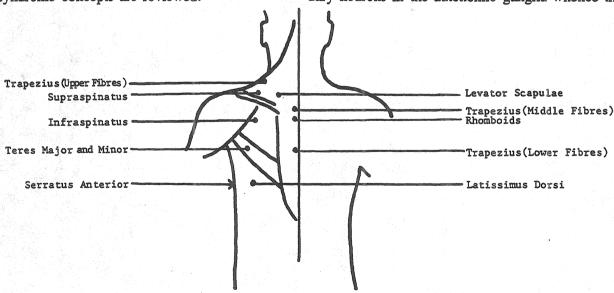


Figure 5 Motor points of the back

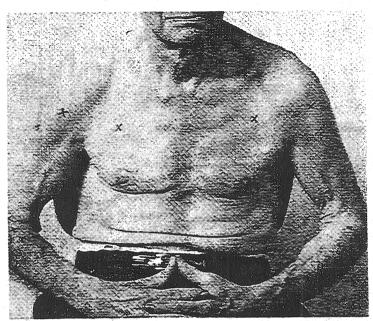


Figure 6 Photograph of patient with tennis elbow resistant to treatment, showing "herald points" at the elbow and other "subclinical points." X-rays showed extensive degenerative changes.

postganglionic fibres pass to their terminal distribution in smooth muscle and glands. Since postganglionic outnumber preganglionic neruons by a ratio of 32:1, the target area affected by one central connection can be extensive.¹¹

The motor innervation of a muscle is plurisegmental and the axons of all the motor units for any one muscle leave the central nervous system by two or more ventral roots.

Stimulation of a trigger point situated on either the anterior or posterior primary ramus of a spinal nerve can cause referred symptoms to both rami but posterior myotomes generally extend lower than the corresponding anterior myotomes.¹⁶

Taking the above neurophysiological features into consideration, afferent barrages from sensory endings at the trigger point can produce motor, sensory and autonomic effects with a referred symptomatology that is generally constant but transmitted to a complex geographic reference area consisting of mixed dermatomal, myotomal and sclerotomal patterns. (See Figure 7.) When the stimulating impulse is of sufficient intensity, the "spill over" manifestation occurs via interconnecting open multisynaptic neuronal chains or internuncial neurons.

Discussion

The term "referred pain" was first used by Sir Henry Head in 1893 in connection with visceral disease, but it is now recognized that referred pain may be associated with both visceral and somatic disease.

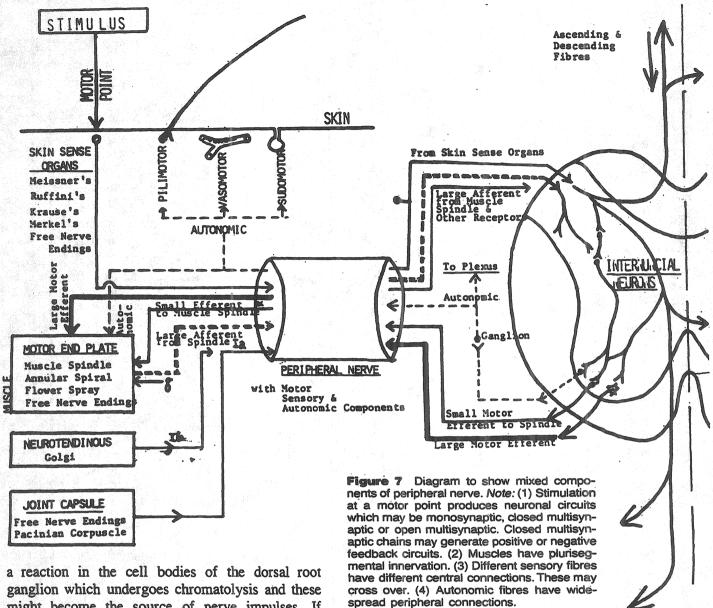
In our clinical studies, we have observed that the locations of trigger points coincide with and can be demonstrated to be situated at muscle motor points and muscle zones of innervation.

A muscle motor point is a known anatomical entity situated in cutaneous tissue, but the trigger zone where tenderness is felt is deeper in muscle at the neurovascular hilus. For instance, the skin over the zone of innervation can be drawn aside. shifting the cutaneous point which responds to minimal electrical stimulation, but subjective tenderness which is felt deeper remains unaltered. Muscle zones of innervation are normally not tender to digital pressure but can be so in the presence of neuropathy. Sometimes this tenderness may be present but is mild or asymptomatic and does not draw attention to itself until caused to erupt into consciousness by some precipitating factor. When the condition is severe, persistent and self-perpetuating, it then acts as a "trigger point" and the resulting disability has been labelled as the "myofascial pain syndrome."

Tender motor zones do not occur singly but occur in muscles belonging to the same affected myotome(s). Since both anterior and posterior divisions are involved, the neuropathy is at root level. Electromyographic findings of radiculopathy associated with radiological and clinical evidence of spondylosis are usually present—myofascial syndromes and spinal arthrosis are both diseases of the middle-aged.

Pain may result from the stimulation of specific receptors (nociceptors) or from partial damage to the peripheral nevous system. The crucial question, therefore, remains whether the apin and tenderness of neuropathy are to be explained by the generation of nerve impulses at some irritant focus or by the block of inhibitory large-afferent fibres following damage. A combination of factors probably exists. As stimulation of large afferent fibres relieves pain, it seems likely that some of the pain is due to a loss of the normal inhibitory barrage. 17,18

When a nerve is damaged at root level, there is



a reaction in the cell bodies of the dorsal root ganglion which undergoes chromatolysis and these might become the source of nerve impulses. If regeneration fails and chromatolysis continues, changes may occur in the axons within the roots themselves and in the terminal arborisations of peripheral afferents in the cord. This seems unlikely to us as we have found in our studies that the degree of tenderness can vary from week to week, even from day to day, suggesting a quickly reversible process.

In neuropathy, it is also possible for sensory receptors, including nociceptors, to be come hypersensitive. The sensitivity of peripheral nociceptors has been shown to be related to autonomic activity¹⁵

which may be unbalanced and affects the normal ongoing afferent barrage. P. D. Wall agrees that an "amplifier" undoubtedly exists and that a search in animal models should be conducted to determine whether it is at the periphery, in the root, or in the cord.¹⁹

The "trigger mechanism" probably occurs as follows. It is the sequel of spondylosis, that is, the skeletal changes resulting from the degeneration of the intervertebral disc. Trigger points commonly occur in the middle age group, in myotomes be-

longing to the commonly affected spinal levels, C5, C6 and L5-S1 and in the presence of radiculopathy. The mixed myotomal, sclerotomal and dermatomal effects have confused the previous recognition of a single segmental level involvement and that trigger points correspond to motor points. Whatever the actual pathogenesis of spondylosis may be-compression by degenerative intervertebral disc material, interference with the blood supply, tethering by ligamenta denticulata, hypertrophy of the ligamentum flavum, excessive movement between adjacent vertebrae, tethering of nerve roots or constitutional stenosis of the spinal canal—at root level it attenuates the normal selective inhibitory afferent barrage and at the same time affects the efferent autonomic vasomotor regulatory function (via ventral ramus) upon which the sensitivity of peripheral receptors and nociceptors depends. The resulting excessive afferent barrage is relayed back to the central nervous system by the related posterior ramus, but again because of pathology at root level, the normal inhibitory barrage is reduced.

The trigger reflex mechanism does not exist in the absence of intact innervation and may be interrupted by depressing the nerve supply—local anesthetic injections or by reducing the number of functioning nociceptors—saline injections.

Some clinical applications are now described. In the management of pain with transcutaneous neural stimulation, we are using motor points which correspond to acupuncture points with good effect.

Following our study on referred pain from cervical spondylosis, we found that "true tennis elbow," caused by local injury to the lateral epicondyle, form only a small percentage of cases seen at the Clinic (estimated at less than 5%). The majority are due to referred pain manifesting as tender motor points in the muscles around the elbow. Awareness of these closely-grouped points around the elbow has resolved many a misdiagnosed tennis elbow and treatment directed to the cervical spine has shortened many weeks of discomfort.

Pain in the shoulder from cervical spondylosis often goes misdiagnosed. When neuropathy is mild and clinical signs sparse, tender motor points can clarify the picture. Thus, a motor point of the supraspinatus need not be mistaken for "tendinitis"

or a deltoid motor point, which overlies the biceps tendon, need not be subjected to surgery as "bicipital tendinitis." Much anxiety may also be allayed when pectoralis tenderness is distinguished from effort angina or myocardial infarction.

Palpation for tender motor points in the leg is most helpful in the diagnosis and prognosis for low back injury, particularly when there are no "positive physical signs." Motor points are not tender in the normal individual but mild and transient points may occasionally be found in asymptomatic subjects after unusual activities, such as jogging.

History of vulnerable back

Mild to moderate tender motor points are usually present in individuals who give a history of a vulnerable back or who have had lesser degrees of trauma. Newman²⁰ has suggested that these vulnerable backs with few symptoms and signs probably represent the first stage of lumbar weakness and are likely to progress to other syndromes of lumbar insufficiency. Radiological findings of minor degenerative changes in these asymptomatic patients are usually dismissed by clinicians as unimportant, but following back injury their disability tends to be prolonged. The presence of tender motor points may therefore be significant in pre-employment medical examinations.

Acutely tender motor points are almost constantly found in patients with disc degeneration. The degree of tenderness and the number of points tend to parallel the patient's condition and may serve as indicators of progress.

An important finding was that patients with simple "low back sprain" and no tender points were disabled for a relatively short period (on an average, less than 8 weeks) while those having tender motor points were disabled as long as patients showing radicular signs (on an average, over 20 weeks). Tender motor points may therefore be considered as a sensitive sign of radicular involvement. Patients seen for the first time who show no physical signs except tender motor points deserve further surveillance, whilst those without tender motor points should be carefully assessed to rule out other causes of low back pain of psychosomatic problems.

Tender motor points around the hip, as in the

shoulder, have often been mistaken for other conditions. A gluteus medius motor point over the upper lateral quadrant of the buttock is frequently attributed to "gluteal bursitis," a tensor fascia lata motor point, for "trochanteric bursitis" and tenderness at the gluteus maximus has been mistaken for the sciatic nerve.

Palpation for tender motor points has also diagnostic and localizing value. Tenderness in muscles innervated by the upper lumbar roots implies lesions at high lumbar levels for which the straight leg raising and femoral stretch tests are of no assistance. Similarly, a search for tender points may serve as a rough and ready substitute for electromyography. Radiculopathy may be detected by tenderness occurring in a group of muscles belonging ot an affected myotome but receiving their segmental innervation through different peripheral nerves.

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References

- 1) Bonica, J. J.: "Management of Myofascial Pain Syndromes in General Practice." J.A.M.A. June 15, 1957. P. 732-738.
- 2) Simons, D. G. "Muscle Pain Syndromes-A Historical Review." Am J. of Phys. Med. 54:289-311, 1975. 55:15-39, 1976.
- 3) Travell, J. & Rinzcer, S. H.: "Pain Syndromes of the Chest Muscles: Resemblance to Effort Angina and Myocardial Infarction, and Relief by Local Block." C.M.A.J. 59:333-338, (Oct.) 1948.
- 4) Michele, A. A.ff Davies, J. J., Krueger, F. J., Lichtor, J. M. "Scapulocostal Syndrome." New York State J. M. June 1950. p. 1353-1356.
- 5) Russek, A. S.: "Diagnosis and Treatment of Scapulocostal Syndrome." J.A.M.A. Vol. 150. No. 1. Sept. 6, 1952. P. 25-27.
- 6) Sola, A. E. & Kuitert, M. C.: Myofascial Trigger Point Pain in the Neck and Shoulder Girdles Treated by Injection of Normal Saline." Northwest Med. Sept. 1955. P. 980-984.
- 7) Gorrell, R. L.: "Treatment of Skeletal Pain with Procaine Injections." Am. J. S. P. 102-104.
- 8) Gunn, C. C. & Milbrandt, W. E.: "A Review of 100 Patients with 'low Back Sprain' Treated by Surface Electrode Stimulation of Acupuncture Points." Am. J. of Acupuncture. Sept. 1975.
- 9) Gunn, C. C. & Milbrandt, W. E.: "Tennis Elbow and the Cervical Spine." C.M.A.J. Vol. 114. May 8, 1976.
- 10) Gunn, C. C. & Milbrandt, W. E.: "Tender Motor Points-A Diagnostic and Prognostic Aid for Low Back

- Injury." J. Bone & Joint Surgery. Sept. 1976. Chusid, J. G.: "Correlative Neuroanatomy." 15th Ed. Lange. P. 236-7 and P. 132.
- 12) Coers, C.: "Note sur une technique de prelevement des biopsies neuromusculaires." Acta. Neurol. Psych. Belg. 53: 759-65 (1953).
- 13) Cyriax, J.: "Textbook of Orthopaedic Medicine." Vol. 1. 5th Ed. P. 167. Bailliere, Tindall & Cassell.
- 14) Buchthal, F. & Pinelli, P.: "Analysis of Muscle Action Potentials in Muscular Atrophy of Neurogenic Origin."
- Neurology (Minneap). 3. 691-603. 1953. 15) Iggo, A.: "Activation of Cutaneous Nociceptors and Their Actions on Dorsal Horn Neurons. Advances in neurology. Vol. 4. International Symposium on Pain. Raven Press. New York. P. 1-9.
- 16) Gough, J. E. & Koepke, G. H.: "Electromyographic Determination of Motor Levels in Erector Spinae Muscles.' Arch. Phys. Med. & Rehab. Vol. 47. 1. Jan. 1966.
- 17) Wall, P. D.: "The Future Attacks on Pain." Advances in Neurology. Vol. 4. International Symposium on Pain. Raven Press. New York. 1974. P. 301-308.
- 18) Pain/ "Editorial," Lancet. September 6, 1975. P. 442-443.
- 19) Wall, P. D.: "Personal Communication," April 1976.
- 20) Nassim, R. & Jackson Burrows, H.: "Modern Trends in Diseases of the Vertebral Column." Butterworths & Co. London. 1959. P. 268.
- 21) Goodgold, J. & Eberstein, A.: "Electrodiagnosis of Neuromuscular Diseases." Williams & Williams. P. 163.