eToims Twitch Relief Method in Chronic Refractory Myofascial Pain (CRMP)

J. Chu[1], **I. Schwartz**[2]

Abstract

Introduction: *CRMP management involves electrical stimulation of motor points.*

Objective: *To demonstrate that Electrical Twitch-Obtaining Intramuscular Stimulation (eToims) using ET127 system for noninvasive motor point stimulation is safe and efficacious in CRMP management.*

Method: *Longitudinal observation of consecutive self-pay outpatients treated from 10/06 through 4/08, divided into Preferred Group ("PG", N = 49, 3 Hz, 4 stimuli/site) and Basic Group ("BG", N = 43, 1 Hz stimulation, 1 stimulus/site). PG and BG had comparable ages, symptom durations, treatment session durations and treatment intervals. Each session involved treatment to large muscles of C4-C7 and L3-S1 myotomes. Outcome measures include prior week's verbal pain levels, pre and immediate post-session pain levels, blood pressure (BP), pulse rate (PR), symptomatic (**S**) and asymptomatic (**A**) side range-of-motion (ROM) for neck rotation (NR), shoulder external rotation (ER), shoulder internal rotation (IR), straight leg raising (SLR) and FABERE testing.*

Results: *PG and BG showed significant improvements ($p < 0.01$) in immediate post-session pain levels and measured ROM. Significantly higher ER**S** (pre and post session percentage changes) noted for BG over PG ($p < 0.05$). Post-session PR decreased in both groups, more so in PG. Systolic BP was mildly elevated in PG but was mildly reduced in BG. Both groups showed no diastolic BP changes. Significant negative correlation noted between increasing number of treatments and pain level only in PG ($r = -0.3$, $p = 0.00$). Increasing number of treatments in PG correlated significantly with improvement in NR**S**, NR**A**, IR**S**, SLR**S**, LR**A**, FABERE**S** and FABERE**A** whereas BG significantly correlated only for improvement in LR**S**. PG had lower average pain levels than BG (3.4 ± 1.9 vs. 4.3 ± 2.5, $p < 0.02$).*

Conclusion: *eToims using ET127 electrical stimulator appears safe and efficacious in CRMP management.*

Key-words: chronic myofascial pain – range of motion – noninvasive electrical stimulation – motor point – muscle twitch

Introduction

Myofascial pain with presence of myofascial trigger points (MTrPs) is a major source of musculoskeletal pain. Lower back pain alone is experienced by 70-85% of all people at some time in life with an annual prevalence of 15-45% (1). US 3-month prevalence of back and/or neck pain was 31% [low back pain: 34 million, neck pain: 9 million, both back and neck pain: 19 million] (39). Among musculoskeletal disorders, the total prevalence of myofascial pain in patients referred for lower limb and upper limb electrodiagnosis was 32% and 42% respectively (4, 5). Due to the high prevalence of myofascial pain, finding safe and efficacious methods for treatment of this ubiquitous condition is essential.

Myofascial pain is accompanied by the presence of myofascial trigger points (MTrPs), which appear

[1] Department of Physial Medicine and Rehabilitation University of Pennsylvania School of Medicine, USA.
[2] Department of Surgery, Division of Urology, University of Pennsylvania School of Medicine, USA.

pathognomonic. Prevalence of motor endplate noise highly correlates with MTrP irritability in active MTrPs, more than latent ones (23). Whereas other myofascial pain treatments appear predicated upon inactivating, disrupting or suppressing MTrP activity (27), a novel approach is advocated that locates the most irritable, active MTrPs, and then electrically exercises them to twitch, promoting healing.

We modified the manual dry needling intramuscular stimulation method initially described by Gunn (18) for management of radiculopathic myofascial pain. Initial work using automated needle intramuscular stimulation at 2 Hz to mechanically induce twitch elicitation found that to be a pre-requisite for relief of pain associated with MTrPs (11). Our later work showed that needle electrical twitch obtaining intramuscular stimulation (ETOIMS) of involved motor end-plate zones/MTrPs had more pain relieving effects than dry needling of the same muscle or overlying skin (10). Dry needling of the same muscle and overlying skin did not induce noticeable twitches. This suggested that electrically induced twitches produce myofascial pain relief. Studies using an automated needling device that also provided electrical stimulation to MTrPs verified that electrically induced twitches produce myofascial pain relief in treating self-pay patients with neck and lower back pain (8, 9). However, due to the invasive nature of the needling method, a safe, efficacious and noninvasive electrical twitch obtaining intramuscular stimulation method (retaining the acronym eToims, capitalizing the "T" to signify the importance of the twitch) was developed in 2006. The development of the bipolar probe ET127 prototype constant current stimulator system (eToims Medical Technology LLC, Philadelphia, Pennsylvania, USA) made this noninvasive twitch elicitation method possible.

Methods and materials

Longitudinal observation was performed on consecutive self-pay outpatients with CRMP treated noninvasively from 10/1/06 through 4/30/08 using the ET127 constant current system with bipolar probe. The bipolar probe has a stimulating surface of 2.5 cm and has an adjustable interelectrode distance. The patients were divided in 2 groups, Preferred Group ("PG", N = 49, 3 Hz stimulation, 4 stimuli/site) and Basic Group ("BG", N = 43, 1 Hz stimulation, 1 stimulus/site). For both groups, the utilized stimulus strength was adjusted up to 100 mA and the pulse width used was 0.1 - 0.5 ms, titrated according to the size of the muscle and patient tolerance to electrical stimulation.

Routinely included for treatment were muscles of bilateral cervical myotomes: trapezius (C3, **C4**), rhomboid major (**C5**), deltoid (C5, **C6**), triceps (**C7**, C8) and latissimus dorsi (C6, **C7,** C8). Treated muscles of bilateral lumbosacral myotomes involve: gluteus maximus (L5, **S1**), tensor fascia latae **(L5**, S1), adductor magnus (L2-S1), and quadriceps (L3, **L4**). Also treated were bilateral paraspinal muscles from C4 - S1 levels. The principle of treatment was to find irritable MTrPs that, when stimulated, elicited brisk, rapid twitch contractions at a stimulus intensity tolerated by the patient. To locate such MTrPs, stimulus intensities were utilized befitting the classic motor point definition, an area requiring the shortest duration pulse with least stimulus intensity for muscle contraction, i.e., twitch elicitation. A countdown timer in the ET 127 system terminated the session after sounding a warning audio signal during the last five seconds. The patients were usually positioned in supine, prone, side-lying and opposite side-lying positions during treatment.

An assistant collected outcomes data consisting of patient pain levels (highest, average and lowest) from the week before each session (0-10 in full or half grades), pre and immediate post session pain verbal levels (0-10 in full or half grades), blood pressure (BP), pulse, symptomatic (**S**) and asymptomatic side (**A**) range of motion (ROM) of neck, shoulders and lower limbs. The ROM parameters in cm include: NR (neck rotation) measured distance between the middle of the chin to the ipsilateral acromioclavicular joint; ER (external rotation of shoulder) measured distance between tip of middle finger of tested side to contralateral angle of the mouth, when the tested upper limb is externally rotated, flexed at shoulder and elbow, and the patient places the tested limb behind the neck, with forearm in pronation; IR (internal rotation of the shoulder) measured distance between the tip of the middle finger of the tested side to the contralateral midpoint of the spine of the scapula, when the tested upper limb is extended and adducted at the shoulder, with the elbow flexed behind the trunk, with the forearm

Table 1. – *Presenting features of patients*

	PG (49 patients M: F = 26:23)	BG (43 patients, M: F = 21:22)
Upper body pain	28 (57.1%) M: F = 9:19	27 (63%) M: F = 16:11
Lower body pain	21 (42.9%) M: F = 13:8	16 (37%) M: F = 8:8
Stated cause of pain:		
Auto-accident	12 (24.5%)	12 (27.9%)
Lifting injuries	8 (16.3%)	9 (20.9%)
Falls	6 (12.2%)	6 (13.9%)
Repetitive stress injuries	6 (12.2%)	7 (16.3%)
No trauma	17 (34.7%)	9 (20.9%)

Abbreviations: M = males, F= females, PG = Preferred Group, BG = Basic Group

Table 2. – *Characteristics of patients in treatment*

Age (years)		Symptom duration (months)		Tx interval (number)		Treatment (number)		Tx session duration (minutes)	
PG	BG	PG	BG	PG	BG	PG	BG	PG	BG
48.2 ± 17.7	45.1 ± 15.2	97.4 ± 130.4	89.1 ± 105.8	9.5 ± 13.9	10.0 ± 17.8	*60.7 ± 41.9	28.9 ± 21.9	28.2 ± 18.6	24.6 ± 16.0

*P < 0.01, Abbreviations PG = Preferred Group, BG = Basic Group

Table 3. – *Comparison of MRI diagnoses of PG and BG patients*

	PG (49 patients)	BG (43 patients)
Normal	6 (12.2%)	2 (4.7%)
Spinal DJD	5 (51.0%)	5 (58.0%)
Bulge	2 (4.1%)	2 (4.7%)
Herniation	2 (4.1%)	4 (9.3%)
Stenosis	10 (20.4%)	8 (18.6%)
Scarring post-laminectomy	4 (8.2%)	2 (4.7%)

Abbreviations: MRI = Magnetic Resonance Imaging, DJD = Degenerative Joint Disease, PG = Preferred Group, BG = Bsic Group

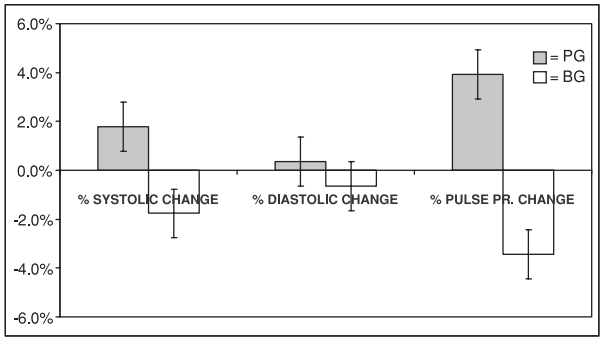

Fig. 1. – Comparison of blood pressure changes for Preferred Group (PG) and Basic Group (BG) before and immediately after treatment (% changes).

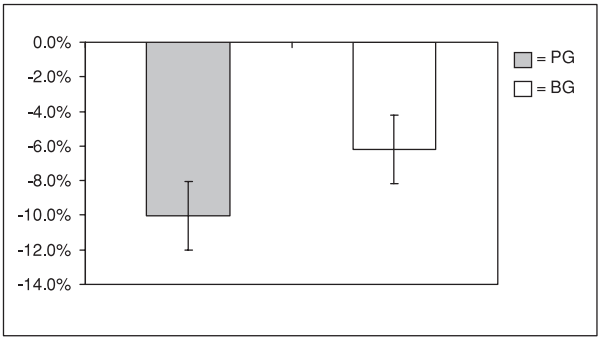

Fig. 2. – Comparison of pulse rate changes for Preferred Group (PG) and Basic Group (BG) before and immediately after treatment (% changes).

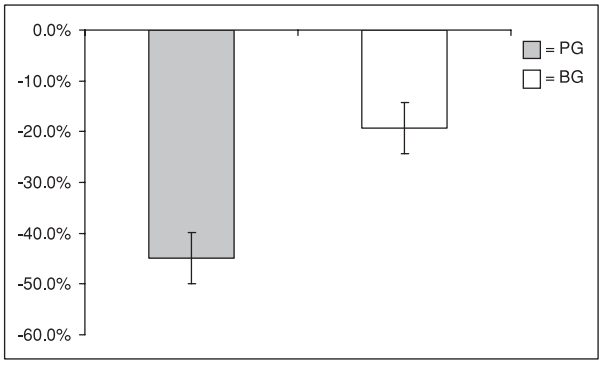

Fig. 3. – Comparison of pain level changes for Preferred Group (PG) and Basic Group (BG) before and immediately after treatment (% changes).

in supination; and FABERE (the ability to flex, abduct and externally rotate the tested hip) measured vertical distance between the most lateral knee flexion crease of the tested side to the surface of the bed, when the tested side heel is placed above the superior border of the contralateral patella. The other ROM parameter measured utilizes degrees, SLR (straight leg raising test), the angle between the surface of the bed and the plane bisecting the middle of the thigh and the leg.

Patients were advised to return weekly for eToims treatments at will and continue their pre-eToims treatment prescription medications throughout the course of eToims therapy. Only two patients in PG and one patient in BG were on oral narcotics for pain control. Only three patients in PG preferred to take a fast acting narcotic medication 30 minutes before the treatment to better tolerate it.

Results

Patients were divided into two groups. There were 49 patients in PG and 43 patients in BG. No statistically significant differences in age, duration of symptoms and treatment interval were found. PG patients had significantly higher number of treatments. Patient characteristics are presented in Tables 1-3.

For both groups, there were statistically significant improvements ($p < 0.05$ with T-test for dependent variables) for the following before and after treatment measures: pain level, NR, ER, IR, SLR and FABERE for both symptomatic and asymptomatic sides. Significant after session changes were noted for systolic BP and pulse pressure, which was mildly elevated in PG but was mildly reduced in BG. No significant diastolic BP changes noted in both groups after treatment. There was significant reduction in post-session pulse rate in both groups, more so in PG.

By Mann-Whitney U test, after treatment changes favoring PG when compared to BG were significant percentage reduction in pulse rate and percentage reduction in pain levels. However, PG showed significant percentage elevation in systolic blood pressure and pulse pressure ($p < 0.01$). There were no significant differences for percentage changes in diastolic pressure (Figures 1-3). However, BG patients had significant improvement in the percentage change over PG's improvement in symptomatic side shoulder external rotation (ER**S**, $p < 0.05$).

Using Spearman correlation, significant negative correlation was noted between increasing number of treatments and pain levels in PG for high, average and low pain levels ($r = -0.3$, $p = 0.00$) and not BG. Increase in number of treatments in PG correlated significantly with improvement in NR**S** and NR**A** ($r = 0.5$ with $p = 0.000$, and $r = 0.3$ with $p = 0.000$ respectively), IR**S** ($r = 0.2$ with $p = 0.000$), SLR**S** and SLR**A** ($r = 0.2$ with $p = 0.000$ respectively), and FABERE**S** and FABERE**A** ($r = 0.2$ with $p = 0.000$ and $r = 0.3$ with $p = 0.000$ respectively). Increasing number of treatments in BG significantly correlated only for improvement in LR**S** ($r = 0.2$ with $p = 0.000$). PG had significantly lower average pain level ($3.4 + 1.9$) than BG ($4.3 + 2.5$), $p < 0.02$.

Regression analyses showed that after treatment pulse rate had no significant relationship to after treatment systolic and diastolic blood pressures and pulse pressure. SPSS program for windows (version 12) was used for statistical analyses.

Discussion

Myofascial pain is accompanied by presence of myofascial trigger points (MTrPs). MTrPs are localized taut bands with tender points that are more circumscribed than the band itself and when compressed produce stereotypical referred pain patterns. Snapping palpation of the myofascial band also produces a local twitch response (40). The integrated hypothesis of Travell and Simons for MTrP formation suggests that muscle trauma, overload, or strain cause damage to the endplate which results in release of excessive acetylcholine (Ach). This causes a local, partial contraction of a muscle fiber beneath the endplate. Muscle fiber contracture leads to ischemia and pain.

Myofascial pain, as postulated by Gunn, is caused by spondylotic radiculopathies in which pain arises from mechanical traction of muscle fibers shortened by denervation, causing intramuscular entrapment of nerves and blood vessels and a tension effect on pain sensitive regions, such as annulus fibrosus, bones and joints (18). Gunn's postulated traction effects of the muscle, secondary to denervation, constitutes one

instance and, possibly, the major instance of how, in the theory of Travell and Simons, injury can initially befall the muscle from excessive release of ACh. Another mechanism by which muscle fibers become shortened involves denervation supersensitivity to Ach, which develops within two weeks of denervation (2). An increase in ACh receptors at extrajunctional areas and a decrease in acetylcholinesterase activity contribute to the phenomenon of denervation supersensitivity (27). Additionally, denervation supersensitivity can occur in muscles subjected to prolonged conduction block (26).

Muscle fiber shortening compresses small blood vessels and the tissue becomes ischemic. Ischemia leads to the release of bradykinin and sensitization or excitation of nociceptors (29). Reflex spasm in a given muscle can be induced by nociceptive input from neighboring joints or muscles. If the force generated by a spasm is relatively high, it will compress the large blood vessels supplying the muscle causing more ischemia. This can lead to a drop in pH. The resulting acidic environment and bradykinin release, are known stimulants for muscle nociceptors, resulting in myofascial pain (28).

Shah verified that active MTrPs possessed relatively higher acidic levels (lower pH) than latent MTrPs. Such findings in the active MTrPs could be associated with conditions attributed to hypoxia and ischemia. Similarly, levels of bradykinin, serotonin, norepinephrine, substance P, CGRP, TNF-[alpha], and IL-1[beta] were all at relatively higher concentrations in active MTrPs (36).

It has been found that active MTrPs and consequent MTrP irritability are highly correlated with the prevalence of endplate noise more than that noted in latent ones (24). Our own findings with monopolar EMG needle insertion at MTrPs have shown elicitation of end-plate spikes, grouped single-muscle fiber discharges, fasciculations, and myokymic discharges through mechanical stimulation. These elicited discharges or micro-twitches cause immediate muscle fiber contraction and then relaxation, which may be the basis for pain relief with needling tender MTrPs (12). Based on that, clinically visible muscle contraction and relaxation induced with surface-applied, noninvasive Electrical Twitch-Obtaining Intramuscular Stimulation (eToims Twitch Relief Method) of tender MTrPs consequently should produce definitive musculoskeletal pain relief.

Our technique makes use of the hypothesis that spondylotic radiculopathy with denervation supersensitivity is the underlying cause of myofascial pain. Consequently, denervation leads to formation of MTrPs in many myotomes. Axonal microstimulation and single fiber electromyography testing demonstrate evidence of neuroaxonal degeneration and neuromuscular transmission disorders in MTrPs. The jitter values measured in such MTrPs positively correlate with disease duration. It has been theorized that the etiologic mechanism of myofascial pain syndrome possibly involves degeneration of motor neurons (7).

Treating the underlying etiologic lesion responsible for MTrP activation is the most important strategy for others, but not eToims. For others, CRMP treatment centers on procedures that involve inactivation, disruption or suppression of MTrP activity. Such procedures include dry needle intramuscular stimulation and injection of sterile water, saline, local anesthetics, steroids or Botox. In direct opposition to these approaches, eToims electrically excites MTrPs.

eToims searches for and locates the most irritable and active MTrPs. Identification of such MTrPs has therapeutic pain relieving effects. This identification involves MTrP electrostimulation, generating twitches. eToims promotes healing of irritable MTrPs through twitch elicitation, stretching problematic tight and shortened muscle fibers, thereby reducing traction effects on pain sensitive structures, such as entrapped intramuscular nerves and blood vessels, bone surfaces and joint capsules. Within muscles, twitch induced exercise effects promote local blood flow improving tissue oxygenation and removing local accumulation of pain producing neurochemicals.

Low-intensity, low-frequency electrostimulation delivered within MTrPs by needle stimulation has also been used as intervention to deactivate MTrPs (31). Our use of low-intensity, low-frequency electrostimulation to elicit MTrPs to twitch is a therapeutic procedure in the management of myofascial pain (8-10). This noninvasive eToims method promotes the healing process, instead of directly disrupting or destroying a neuromuscular junction that is already undergoing denervation. Through central effects, low-frequency electrical stimulation can also relieve pain by transmission of non-painful stimuli

transmitted via large-diameter A-fibers (Gate theory) and possibly from stimulation of the periaqueductal gray matter (25, 31). Additionally, animal studies have shown that repetitive stimulation of peripheral nerves relieves pain through diffuse depression of nociceptive transmission in the sensory pathway, while mobilizing anti-nociceptive action in the affective pathway, as well as involving non-nociceptive spinal cord activity (19, 43).

Intramuscular contraction and immediate relaxation of treated muscle fibers with eToims produces simultaneous eccentric contraction of the antagonist muscle. This may relieve pain through stretch effects on the agonist and antagonist muscle fibers. In clinical rehabilitation, stretching of tight muscles is common to relieve pain, to diminish muscle tension and tenderness, and to enhance range of motion. Muscle stretching exercises are commonly used in sports activities to gain flexibility (21, 23).

Intermittent muscle contractions improve skin and muscle circulation. Electrical stimulation-induced contractions improve circulation of the lower leg by the physiologic pumping action of muscle, reducing venous stasis/pooling and edema (17, 30). Immediately following muscle contraction, muscle microvessels exhibit increased convective (flow of red blood cells) and diffusive (perfused capillary surface area) transport (32). The use of low frequency TENS (2 Hz), producing moderate muscle contractions, leads to a transient, local increase in blood flow in muscle and skin (14, 37, 42). Blood flow measurements in the common femoral artery show that surface twitch contractions at 3 Hz increase perfusion in human leg muscles (20).

Neuromuscular electrical stimulation can generate contractions through peripheral and central mechanisms. Direct motor axon activation recruits motor units either in a random order or by first activating fast fatigable muscle fibers. In contrast, synaptic activation recruits motor units in their natural order, starting with fatigue-resistant muscle fibers. The activation of sensory axons can produce contractions through a central mechanism, providing excitatory synaptic input to spinal neurons that recruit motor units in the natural order. However, a stimulation frequency above 20 Hz (preferably 40-60 Hz) was required to activate the central mechanism with pulse widths of 1 ms (16). At low frequency stimulation of 1-3 Hz with pulse width less than 1 ms used for eToims, a central mechanism is an unlikely cause of large force twitches.

It is suggested that motor axons may be more susceptible than cutaneous afferents to conduction block due to presence of neuromuscular junctions which when impaired reduce the safety margin for impulse conduction. If denervation is temporary, such as neurapraxia (conduction block due to focal and restricted demyelination of the nerve), active MTrPs get a chance to be completely healed. Twitch-obtaining intramuscular stimulation has been found useful in relieving conduction block. This is probably related to focal muscle contraction and relaxation that removes tight tissues away from the site of conduction block, as well as associated increase in circulation to that area. eToims has potential curative benefits for neurapraxic axons (13). By increasing blood flow to areas with some permanent nerve damage, potential exists to aid better quality axonal regeneration in axons that have undergone degeneration. It has been shown that 2 Hz percutaneous stimulation with 0.8-1 mA can promote nerve regeneration. When successful regeneration was stimulated by such a method, increase in axon density, blood vessel number and blood vessel area occurs (6).

The use of 3 Hz stimulation to produce neuromuscular fatigue and block of transmission, an established neurophysiologic test, may also aid in the transient depletion of Ach causing neuromuscular block at susceptible neuromuscular junctions (34, 38). Simons and Mense proposed use of Botox to inhibit pre-synaptic release of Ach to prevent its excessive accumulation at MTrPs (28). Noninvasive eToims using 3 Hz stimulation at MTrPs to aid in the transient depletion of Ach causing neuromuscular block at susceptible neuromuscular junctions is safer for chronic use than Botox injections, which cause denervation through the destruction of neuromuscular junctions. Our long term observations show that eToims can be safely repeated multiple times on a regular basis, without injuring neuromuscular junctions.

The noninvasive nature of eToims Twitch Relief Method allows many active MTrPs to be treated in one session. This is not achievable with needling methods for treating MTrPs. MTrPs are often difficult to locate in patients with chronic pain. This MTrP localization difficulty is either due to significant tightness of overlying muscles or due to the

presence of activity dependent hypo-excitability with axonal hyperpolarization (41). In such situations high-intensity stimulation is needed, but not well tolerated. Therefore, 3 Hz stimulation at lower stimulus intensities with concomitant elicitation of many twitches, albeit low force, can also produce pain relief. This is verified by the fact that PG patients who received repetitive stimulation at 3 Hz, which allowed more twitches to be elicited per unit time, showed progressive reduction of pain levels and lower pain levels immediately after treatment and through follow-up, which was not noted in BG patients stimulated at 1 Hz. Due to its noninvasive nature, the use of the prototype ET 127 device with bipolar probe facilitated treatments by enabling a rapid search and localization for these irritable MTrPs. It also facilitates treatments to tender but asymptomatic, latent MTrPs in bilateral cervical and lumbosacral myotomes associated with "silent" denervation resulting from co-existent multi-level spondylotic radiculopathies.

Activity related fluctuation of symptoms in CRMP is common. It may result from transient conduction block. Even natural activity results in substantial hyperpolarization of active axons and, for similar discharge rates, the degree of hyperpolarization is greater in motor axons than cutaneous afferents. The greater affect of activity on the excitability of motor axons could be caused by less inward current rectification and less persistent sodium conductance than in sensory axons. Suggested is that motor axons may be more susceptible than cutaneous afferents to conduction block at sites of impaired safety margin for impulse conduction (41). When such conduction block is not relieved as soon as possible, it has potential to proceed to axonal degeneration. The increased susceptibility of MTrPs to further trauma, whether induced by violent muscle contractions, associated new injuries, such as falls, lifting injuries and auto accidents or even repetitive contractions associated with activities of daily living can keep these patients in a constant state of ongoing and chronic pain. The repeat decision of these patients to continue to pay for myofascial pain treatments over time is a positive indicator of patient appreciation for pain relief and improvement in quality of life attributable to eToims. Review of our longitudinal observations revealed no appreciable treatment side effects.

After the treatment sessions, pulse rate decreased significantly in both groups, especially in PG. This was accompanied by a mild elevation in systolic blood pressure in PG and a mild reduction in systolic blood pressure in BG, but without significant change in diastolic blood pressure in both groups. The literature notes that increase in blood pressure is associated with potentially painful massage techniques, including trigger point massage therapy and sports massage (3). This raises the possibility that increase in systolic blood pressure in PG may be related to pain production from repetitive stimulation at 3 Hz in the presence of significant underlying pain. BG patients had a reduction in systolic blood pressure, which may be related to relaxation effects of 1 Hz stimulation which causes a massage effect from tissue mobilization. This is similar to Swedish massage used for mechanical activation of muscular tissue, skin, tendons, fascias, and connective tissue. Swedish massage may indirectly regulate the tonus of the autonomic nervous system and has been shown to have greatest effect on blood pressure reduction from sympathetic inhibition (3).

Reduction in PG pulse rate could be related to increase in systolic blood pressure due to treatment pain but the treatment pain was not significant enough to produce a corresponding increase in diastolic blood pressure. However, BG patients also showed reduction in pulse rate, even when accompanied by mild reduction in systolic blood pressure. Since regression analyses showed that the after treatment pulse rate had no significant relationship to after treatment systolic and diastolic blood pressures as well as pulse pressure, vagal stimulation may play a significant role in pulse rate reduction. Vagus nerve stimulation reduces pain in humans, which may be related to central inhibition rather than alteration of peripheral nociceptive mechanism (22). That patients in PG had better pain relief and more decrease in pulse rate than BG suggest that more muscle contractions and movements of the neck and upper limbs due to 3 Hz stimulation may have stimulated the vagus nerve directly or through central mechanisms.

Potential bias was inadvertently introduced in observations because treatments were not randomized, controlled or double blinded. However, BG patients receiving 1 Hz stimulation were a relative control group versus PG patients receiving repeti-

tive stimulation at 3 Hz. Both patient groups were sufficiently treated with frequencies thought sufficient for pain relief. Hence, the lesser treated group was the "Basic Strength Group." The fact that BG patients showed no correlation between increasing number of treatments and pain reduction, as well as also maintained higher pain levels, advocates eToims greater potential for pain reduction using repetitive stimulation at 3 Hz. However, BG patients did have sufficient pain relief, before and after treatment, and also had improvement in range of motion parameters, such that they also self-selected to return for further treatments for which they also paid, similar to PG patients. The fact that BG patients who received stimulation at 1 Hz showed significantly more improvement in measured range of motion of the symptomatic side shoulder external rotation immediately after the session than those in PG, indicated that 1 Hz stimulation produced less treatment pain. This allowed BG patients to show an immediate improvement in range of motion of the symptomatic side. BG patients may require longer follow-up to adequately demonstrate similar correlation between increasing number of treatment sessions and decrease in pain level. This lack of correlation may also be related to fewer and less forceful twitches produced per session.

There were no complications or adverse effects related to eToims followed longitudinally over 18 months. Treatment pain can occur due to application of inadequate stimulus to the underlying muscle fibers, allowing them to shorten and tighten without relaxation, aggravating the pain. Also, as the motor point, i.e. MTrP, is the most tender area (33, 35), the patient feels tenderness to stimulation, especially in the presence of high levels of underlying pain. This is related to stimulation of higher numbers of nociceptors per unit area, when the muscle is tight and shortened, which increases the pain during electrical stimulation (33, 35). Only when patients are unable to tolerate submaximal stimulation dosages are pre-treatment medications indicated to enable satisfactory treatment sessions. The fact that very few patients needed pretreatment medication indicates that eToims is well tolerated by the majority of patients, as the electrical stimulus can be titrated to patient tolerance.

Chronic pain management is common using transcutaneous electrical stimulation and other various neuromuscular stimulators. Our longitudinal follow-up of eToims used chronically has also been found to be safe. However, studies have shown that there is electromagnetic interference in those with implantable cardioverter defibrillators. We have avoided use of eToims in such patients and recommend that presence of such implanted devices and stimulators, be considered a contraindication for the use of eToims (15). Other contraindications include patients with pacemakers, seizure disorders, history of cerebrovascular accidents including transient ischemic attacks, bleeding disorders, therapeutically anticoagulated, active infections, active inflammations, fractures, acute illnesses, organ failures, neuroendocrine disorders (e.g. pheochromocytoma), hyperkinetic heart syndrome, coronary artery disease with angina, debilitation, significant profound psychiatric disorder, suicidal or homicidal ideas, inability to follow commands, disruptive or noncooperative patients and pregnant patients. Those patients with very severe and high levels of pain, pain associated with significant denervation, failed multiple spinal surgeries, need for multiple narcotics for pain control, pain due autoimmune diseases, central pain or sympathetically maintained pain, those with thickened and scarred skin, edema, and obesity are not candidates for eToims.

Longitudinal observation added a quantifiable clinical electrophysiological dimension regarding the force and ease of twitch elicitation to the identification of trigger points, presently identified by clinical palpation and laboratory methods. Subjectivity enters when patient participation is needed to help identify MTrPs. Additionally, manual palpation is unable to identify deep MTrPs. Objective parameters were utilized to identify MTrPs for their therapeutic pain relieving effects by use of classic motor point definition, describing these points as areas requiring the shortest duration pulse with least stimulus intensity for muscle contraction, i.e., twitch elicitation.

Previous non-controlled clinical studies suggested an analgesic effect of needle ETOIMS in musculoskeletal pain (8-10). This present longitudinal observation confirmed that non-invasive eToims Twitch Relief Method has pain relieving effects that appear safe and efficacious when used regularly and repeatedly and over time in the chronic long-term care of patients with CRMP. In CRMP management, muscle twitches provide the local key to pain relief.

References

1. Andersson, G.: Epidemiological features of chronic low-back pain. *The Lancet*, 354: 581-585, 1999.
2. Antony, M.T. and Tonge, D.A.: Effects of denervation and botulinum toxin on muscle sensitivity to acetylcholine and acceptance of foreign innervation in the frog. *J Physiol.,* 303: 23-31, 1980.
3. Cambron, J.A., Dexheimer, J. and Coe, P.: Changes in blood pressure after various forms of therapeutic massage: a preliminary study. *J. Alternat. Comple. Med.*, 12(1): 65-70, 2006.
4. Cannon, D.E., Dillingham, T.R., Miao, H., Andary, M.T. and Pezzin, L.E.: Musculoskeletal disorders in referrals for suspected lumbosacral radiculopathy. *Am. J. Phys. Med Rehabil.*, 86(12): 957-61, 2007.
5. Cannon, D.E., Dillingham, T.R., Miao, H., Andary, M.T. and Pezzin, L.E.: Musculoskeletal disorders in referrals for suspected cervical radiculopathy. *Arch. Phys. Med. Rehabil.*, 88(10): 1256-9, 2007.
6. Chen, Y.S., Hu, C.L., Hsieh, C.L., Lin, J.G., Tsai, C.C., Chen, T.H. and Yao, C.H.: Effects of percutaneous electrical stimulation on peripheral nerve regeneration using silicone rubber chambers. J. *Biomed Mat. Res.,* 57(4): 541-549, 2001.
7. Chang, C.W., Chen, Y.R. and Chang, K.F.: Evidence of neuroaxonal degeneration in myofascial pain syndrome: A study of neuromuscular jitter by axonal microstimulation. *Eur. J. Pain*, 2008 march.
8. Chu, J., Schwartz, I. and Aye, H.H: Efficacy of Electrical Twitch Obtaining Intramuscular Stimulation (ETOIMS) in Chronic Lower Back Pain. *Arch. Phys. Med. Rehabil.*, 86 (9): e50-e52, abstract 194, 2005.
9. Chu, J., Schwartz, I. and Aye, H.H: Efficacy of Electrical Twitch Obtaining Intramuscular Stimulation (ETOIMS) in Chronic Neck Pain. *Arch. Phys. Med. Rehabil.*, 86 (9): e50-e52, abstract 195, 2005.
10. Chu, J., Yuen, K.F., Wang, B.H., Chan, R.C., Schwartz, I. and Neuhauser, D.: Electrical twitch obtaining intramuscular stimulation in lower back pain: A pilot study. *Am J Phys Med Rehabil.,* 83: 104-111, 2004.
11. Chu, J., Neuhauser, D.V. and Schwartz, I., et al.: The efficacy of automated/electrical twitch obtaining intramuscular stimulation (ATOIMS/ETOIMS) for chronic pain control: evaluation with statistical process control methods. *Electromyogr Clin Neurophysiol.*, 42: 393-401, 2002.
12. Chu, J. and Schwartz, I.: The muscle twitch in myofascial pain relief: Effects of acupuncture and other needling methods. *Electromyogr Clin Neurophysiol.*, 42: 307-11, 2002.
13. Chu, J.: Twitch-Obtaining Intramuscular Stimulation (TOIMS) in acute partial radial nerve palsy. *Electromyogr Clin Neurophysiol.,* 39: 221-226, 1999.
14. Cramp, A.F., Gilsenan, C. and Lowe, A.S., et al.: The effect of high- and low-frequency transcutaneous electrical nerve stimulation upon cutaneous blood flow and skin temperature in healthy subjects. *Clin. Physiol.*, 20: 150-157, 2000.
15. Crevenna, R., Stix, G., Pleiner, J., Pezawas, T., Schmidinger, H., Quittan, M. and Wolzt, M.: Electromagnetic interference by transcutaneous neuromuscular electrical stimulation in patients with bipolar sensing implantable cardioverter defibrillators: a pilot safety study. *Pacing & Clinical Electrophysiology*. 26(2 Pt 1): 626-9, 2003.
16. Dean, J.C., Yates, L.M. and Collins, D.F.: Turning on the central contribution to contractions evoked by neuromuscular electrical stimulation. *J. Appl. Physiol.,* 103(1): 170-6, 2007.
17. Faghri, P.D., Van Meerdervort, H.F. and Glaser, R.M., et al.: Electrical stimulation-induced contraction to reduce blood stasis during arthroplasty. *IEEE Trans Rehabil Eng* 5: 62-69, 1997.
18. Gunn, C.C: Treatment of Chronic Pain: Intramuscular Stimulation for Myofascial Pain of Radiculopathic Origin. Churchill Livingston, London, UK., 1996.
19. Ignelzi, R.J., Nyquist, J.K. and Tighe, W.J. Jr.: Repetitive electrical stimulation of peripheral nerve and spinal cord activity. *Neurol Res.*, 3(2): 195-209, 1981.
20. Janssen, T.W., and Hopman, M.T.: Blood flow response to electrically induced twitch and tetanic lower limb muscle contractions. *Arch. Phys Med. Rehabil.,* 84: 982-987, 2003.
21. Kinser, A.M., Ramsey, M.W., O'Bryant, H.S., Ayres, C.A., Sands, W.A. and Stone, M.H.: Vibration and stretching effects on flexibility and explosive strength in young gymnasts. *Med. Sci. Sports & Exer.,* 40(1): 133-140, 2008.
22. Kirchner, A., Birklein, F., Stefan, H. and Handwerker, H.O.: Left vagus nerve stimulation suppresses experimentally induced pain. *Neurology*, 55(8): 1167-71, 2000.
23. Kokkonen, J., Nelson, A.G., Eldredge, C. and Winchester, J.B.: Chronic static stretching improves exercise performance. *Med. Sci. Sports & Exer.,* 39(10): 1825-1831, 2007.
24. Kuan, T.S., Hsieh,Y.L., Chen, S.M., Chen, J.T., Yen, W.C. and Hong, C.Z.: The myofascial trigger point region: correlation between the degree of irritability and the prevalence of endplate noise. *Am. J. Phys. Med Rehabil.*, 86(3): 183-189, 2007.
25. Liu, W.C., Feldman, S.C. and Cook, D.B., et al.: fMRI study of acupuncture-induced periaqueductal gray activity in humans. *Neuroreport*, 15: 1937-1940, 2004.
26. Lorkovic, H.: Supersensitivity to ACh in muscles after prolonged nerve block. *Arch Internat Physiol Biochimie*., 83(4): 771-81, 1975.
27. McConnell, M.G. and Simpson L.L.: The role of acetylcholine receptors and acetylcholinesterase activity in the development of denervation supersensitivity. *J Pharm Exp Therap.,* 198(3): 507-17, 1976.
28. Mense, S.: Neurobiological basis for the use of botulinum toxin in pain therapy. *J. Neurol.*, 251 Suppl 1: I1-7, 2004.
29. Mense, S., Simons, D.G., Hoheisel, U. and Quenzer, B.: Lesions of rat skeletal muscle after local block of acetylcholinesterase and neuromuscular stimulation. *J. Appl. Physiol.,* 94(6): 2494-501, 2003.
30. Morita, H., Abe, C., Tanaka, K., Shiratori, M., Oguri, M. and Shiga, T.: Neuromuscular electrical stimulation and an ottoman-type seat effectively improve popliteal venous flow in a sitting position. *J Physiol Sci*., 56(2): 183-186, 2006.
31. Niddam D.M., Chan R.C., Lee, S.H., Yeh, T.C. and Hsieh, J.C.: Central modulation of pain evoked from myofascial trigger point. *Clin. J. Pain,* 23(5): 440-448, 2007.
32. Pittman, R.N.: Oxygen supply to contracting skeletal muscle at the microcirculatory level: Diffusion vs. convection. *Acta Physiol Scand.,* 168: 593-602, 2000.

33. Qerama, E., Fuglsang-Frederiksen, A., Kasch, H., Bach, F.W. and Jensen, T.S.: Evoked pain in the motor end-plate region of the brachial biceps muscle: an experimental study. *Muscle Nerve*, 29(3): 393-400, 2004.
34. Rowley, K.L., Mantilla, C.B., Ermilov, L.G. and Sieck, G.C.: Synaptic vesicle distribution and release at rat diaphragm neuromuscular junctions. *J Neurophysio*. 98(1): 478-87, 2007.
35. Sener, H.O., Gokdemir, O. and Mutluer, N.: EMG needle causes severer pain in the end-plate region compared to the silent site of the muscle. *Eur Neurol.* 44(4): 219-221, 2000.
36. Shah, J.P., Phillips, T.M., Danoff, J.V. and Gerber, L.H.: An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. *J. Appl. Physiol.,* 99: 1977-1984, 2005.
37. Sherry, J.E, Oehrlein, K.M. and Hegge, K.S., et al.: Effect of burst-mode transcutaneous electrical nerve stimulation on peripheral vascular resistance. *Phys Ther* 81: 1183-91, 2001.
38. Slater, C.R., Fawcett, P.R., Walls, T.J., Lyons, P.R., Bailey, S.J., Beeson, D., Young, C. and Gardner-Medwin, D.: Pre- and post-synaptic abnormalities associated with impaired neuromuscular transmission in a group of patients with 'limb-girdle myasthenia'. *Brain,* 129(Pt 8): 2061-76, 2006.
39. Strine, T.W. and Hootman, J.M.: US national prevalence and correlates of low back and neck pain among adults. *Arth. Rheum.*, 57(4): 656-65, 2007.
40. Travell, J.G. and Simons, D.G.: Myofascial Pain and Dysfunction. The Trigger Point Manual. 2nd ed. Williams & Wilkins, Baltimore, 1992.
41. Vagg, R., Mogyoros, I., Kiernan, M.C. and Burke, D.: Activity-dependent hyperpolarization of human motor axons produced by natural activity. *J Physiol*., 507 (Pt 3): 919-25, 1998.
42. Wikstrom, S.O., Svedman, P. and Svensson, H., et al.: Effect of transcutaneous nerve stimulation on microcirculation in intact skin and blister wounds in healthy volunteers. *Scand. J. Plast. Reconstr. Surg. Hand Surg.* 33: 195-201, 1999.
43. Wang, J.Y., Zhang, H.T., Han, J.S., Chang, J.Y., Woodward, D.J. and Luo, F.: Differential modulation of nociceptive neural responses in medial and lateral pain pathways by peripheral electrical stimulation: a multichannel recording study. *Brain Res*., 1014(1-2): 197-208, 2004.

Address reprint requests to:
Jennifer Chu, M.D.
CEO & Principal
eToims® Medical Technology, LLC
eToims® Soft Tissue Comfort Center, LLC
3401 Market Street, Suite 135
Philadelphia, PA 19104-3315

Tel: (215) 387-0550
Fax: (215)387-0556
Email: jchu@etoims.com
www.stopmusclepain.com