Does EMG (dry needling) reduce myofascial pain symptoms due to cervical nerve root irritation?

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CASE REPORT

Chronic refractory myofascial pain and denervation supersensitivity as global public health disease

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SUMMARY

Chronic pain with a 30.3% global prevalence significantly impacts universal health. Low back pain has a 9.4% prevalence worldwide causing the most widespread disability. Neck pain ranks 4th highest regarding years lived with disability with a 4.9% prevalence worldwide. The principal cause of pain in 85% of patients visiting a tertiary pain clinic has a myofascial origin. The root cause is multifocal neuromuscular ischaemia at myofascial trigger points from muscle tightening and shortening following spondylotic radiculopathy induced partial denervation. Chronic refractory myofascial pain (CRMP) is a neuromusculoskeletal disease needing management innovations. Using electrical twitch-obtaining intramuscular stimulation (eToims), we provide objective evidence of denervation supersensitivity in multiple myotomes as cause, aggravation and maintenance of CRMP. This study underscores our previous findings that eToims is safe and efficacious for long-term use in CRMP. eToims aids potential prevention (pre-rehabilitation), simultaneous diagnosis, treatment (rehabilitation) and prognosis in real time for acute and CRMP management.

CASE PRESENTATION

A 63-year-old successful entrepreneur/mountaineer suffered disabling chronic low back pain (LBP) and left buttock pain after an 8 feet (2.4 m) fall in 2011 with pain aggravation 5 months later from a physically challenging expedition.[1–6] He had laminotomy with lumbar disc removal when contrast MRI in 2013 showed an L4-L5 broad-based left paracentral disc extrusion with central canal narrowing and mass effect on bilateral L5 roots. Other MRI findings included L4-L5 retrolisthesis, C5-C7 degenerative disc changes, lower thoracic Schmorl's nodes, L1-S1 small broad-based disc bulges, moderate sacroiliac joint arthritis bilaterally, left hip labral tear and old right total hip arthroplasty. Spine X-rays showed 24° lumbar levoscoliosis (figure 1).

Postspinal surgery pain worsened but did not alleviate with physical therapy, manual stretching, inversion spinal traction, epidural injections ×3, chiropractic/osteopathic manipulations, anti-inflammatory medications, as well as short-acting and long-acting opioids, acupuncture and alternative methods. Pain severely compromised going up inclines/steps and ambulation to 500–1000 feet (150–300 m), necessitating back and hip muscle stretches every 5–10 min. Pain scale was 6/10 on presentation for electrical twitch-obtaining intramuscular stimulation (eToims) on 7 August 2014. Examination showed moderate range of motion limitation of the neck, back, shoulders and hips with core muscle weakness, especially on the left. There were no sensory deficits or upper motor neuron signs.

The pain scale reduced from 6/10 to 2/10 with the first eToims session confirming predominant myofascial involvement. Satisfaction with the results made him voluntarily return frequently for fee-for-service eToims treatments. He was prescribed gabapentin 1200 mg which he took only before eToims.

He had no contraindications such as systemic/autoimmune/malignant diseases, pacemakers, angina, infections, disorders such as seizures, bleeding, profound psychiatric problems, skin scarring, obesity, central pain, sympathetically maintained pain, multiple failed spinal surgeries, use of multiple narcotics or severe pain in the 8–10/10 range. For females, pregnancy is a contraindication.

Materials and methods

Patient regularly self-measured and recorded mean of three blood pressure (BP) and pulse readings (Omron Hem 711 automated sphygmomanometer), before and immediately after treatment in sitting position in the same chair with steady respiration.

He selected 90 min eToims sessions, occasionally longer when pain relief was unsatisfactory. The corresponding author applied treatment using an eToims stimulator with bipolar probe to the C4-S1 paraspinal muscles and large muscles of C4-C7 and L2-S1 myotomes. Initially deep myofascial trigger points (MTrPs) were very difficult to seek and to stimulate and only weak force twitches were elicitable since tissue sensitivity prohibited the patient from tolerating pulse widths >500 μs and intensity >30 mA at 1.5 Hz.

After 4 months daily eToims, he tolerated stimulation with 1000 μs and 70–80 mA strength at 3 Hz to susceptible MTrPs. By session 49 (98±13 professional eToims hours), he exhibited rapid-firing, vigorous, antigravity lower limb movements. By session 76 (106.5±20.0 professional eToims hours), stimulation elicited twitch-trains that continued autonomously until abrupt fatigue (video 1). Occasionally after an eToims session, owing to muscle softening, he could mechanically initiate autonomous twitches with finger pressure on MTrPs with denervation supersensitivity (DS) (video 2). Mechanical stimulation through body/limb repositioning can maintain and/or improve the quality of electrically evoked twitches. Often,

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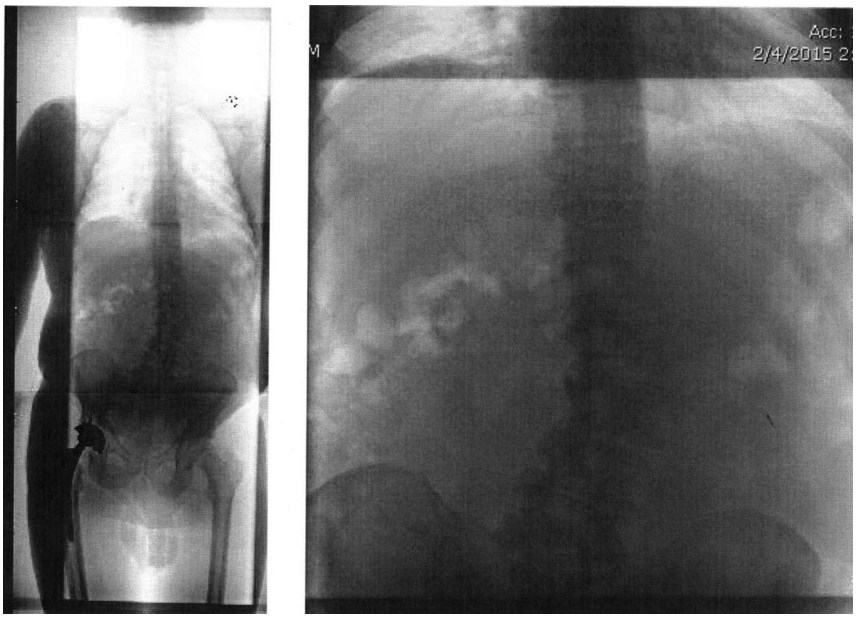


Figure 1 Erect spine X-rays showing 24° lumbar levoscoliosis.

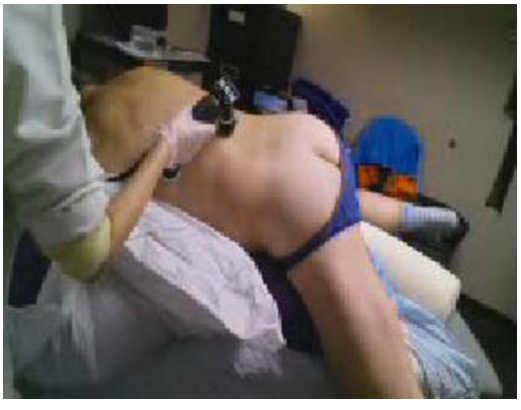
electrical supersensitivity was evidenced as forceful contraction of muscles remote from the stimulation site, for example, stimulating left gluteus maximus MTrPs, could cause upper trunk extension or simultaneous antigravity movements of the lower limbs. Stimulating the mid-thoracic paraspinal muscles or abdominal muscles caused lower limb movements. Stimulating the right latissimus dorsi MTrPs could induce forceful antigravity, simultaneous head/neck/trunk and right shoulder extension (video 1). Muscles most difficult to obtain fatiguing contractions were bilateral gluteal muscles, especially on the left. After 7 months of eToims, the pain level reduced to 1–1.3/10 and he started walking 4 miles (6.4 km) at 1.5–2 mph (2.6–3.2 km/h) including 45° inclines.

Nine months into treatment, he successfully completed travelling in an expedition, his first since 2011 during which he walked 4–6 miles (6.4–9.6 km) on most days. During the 60-day vacation hiatus beginning 1 June to 30 July 2015, he performed self-eToims but due to frequent, strenuous activities, the pain scale increased to 6/10. In the 45-day period prior to and after the expedition, his treatment sessions lasted 115±12.9 and 120±6.6 min, respectively, indicating more difficulty in eliciting large force twitches due to tightness of the muscles. Electrical supersensitivity related twitching at remote sites gradually returned but he lost the ability to mechanically provoke autonomous twitch-trains.

For further improvement and/or maintenance of quality of life (QOL), he requires and still receives ongoing, self-applied and professionally applied eToims.

Results and analysis

During the 14-month analysis period (7 August 2014–7 October 2015), he underwent 214 professionally applied eToims. Statistics were performed with SPSS V.12. The difference (Δ) between before-treatment ($\bar{\alpha}$) and after-treatment ($\bar{p}$) systolic BP (S) and diastolic BP (D), pulse pressure (PP) and pulse (P) were summed after each treatment and grouped into (SDPPP $\bar{\alpha}\,\&\,\bar{p}\Delta$ sum) elevated or reduced sessions. (SDPPP $\bar{\alpha}\,\&\,\bar{p}\,\Delta$ sum) reduced sessions showed higher ($\bar{\alpha}$) pain scales, S&PP. In the SDPPP reduced sessions, improved values were noted for post treatment S,D &PP with shorter treatment



Video 1 Grade 5 electrical twitch-obtaining intramuscular stimulation twitches in the left gluteus maximus and right latissimus dorsi muscles.

Video 2 Twitches elicited with mechanical stimulation using finger pressure on a hyperexcitable myofascial trigger points in the left gluteus maximus. He had just received electrical twitch-obtaining intramuscular stimulation and his muscles were soft enough to be mechanically stimulated to elicit twitches which were noted to continue autonomously without the need for further mechanical stimulation.

	MEAN± SD SDPPP reduced after tx (N=124)	MEAN± SD SDPPP elevated after tx (N=67)	Significance (p) (Mann-Whitney test)
Number of Txs	109.6±67.9	106.2±54.9	0.693
Tx interval	2.2±5.6	2.0±2.7	0.842
Tx Session Time	98.7±15.9	106.2±54.9	**0.001**
Pain Scale Before Tx	4.3±0.9	3.9±0.9	**0.009**
Pain Scale After Tx	1.9±0.8	1.9±0.8	0.554
Tx Related Pain Relief	2.4±1.3	1.9±1.1	**0.035**
Before Tx SBP	126.1±5.4	122.6±5.9	**0.000**
After Tx SBP	120.8±5.7	126.9±6.2	**0.000**
Before Tx DBP	69.1±3.4	68.3±3.2	0.094
After Tx DBP	68.5±3.7	70.9±4.3	**0.000**
Before Tx PP	56,7±5.5	54.1±6.0	**0.010**
After Tx PP	52.2±5.3	56.2±5.1	**0.000**
Before Tx P	76.5±9.8	74.4±7.7	0.367
After Tx P	71.1±8.9	72.0±10.9	0.116
Before and After Tx SBP Difference	- 5.3±4.9	4.3±4.6	**0.000**
Before and After Tx DBP Difference	- 0.7±4.3	2.7±4.2	**0.000**
Before and After Tx PP difference	- 4.5+5.4	2.3±4.9	**0.000**
Before and after Tx P difference	- 4.8±6.7	-0.7±4.8	**0.000**
SDPPP sum difference	-15.6±9.4	8.7±7.2	**0.000**
TW Force	3.7±1.1	3.9±0.9	0.525

Abbreviations: SBP= systolic blood pressure, DBP= diastolic blood pressure, PP= pulse pressure,P= pulse, SDPPP= Systolic, Diastolic,Pulse Pressure & Pulse, Tx= treatment, TW= twitch.

Note: SDPPP reduction sessions showed shorter treatment session time, higher pain scales before treatment, larger treatment related pain relief, higher SBP and lower PP before treatment. After treatment, this group had lower SBP, DBP and PP.

Figure 2 Means and SDs of different variables recorded for treatment related (systolic BP and diastolic BP, pulse pressure and pulse) SDPPP reduced sessions and SDPPP elevated sessions with significance in differences of the means.

sessions ($p<0.05$) that provided more significant pain relief. (figure 2).

Linear regression analysis showed that ($\bar{\alpha}$) pain scales were influenced negatively by treatment numbers and twitch force and positively by $\bar{\alpha}\&\bar{p}\Delta$ pain scale ($R=0.913$ and $R^2=0.833$ analysis of variance $F=91.5$ $p=0.000$), adjusted for numbers of treatment, session time, treatment interval, $\bar{\alpha}\&\bar{p}\Delta$ for S, D, PP&P. Twitch forces grades 1–5 included the most forceful twitch grade in any muscle in each session.

Correlation analysis (Spearman, figure 3) showed a strong positive correlation between twitch force and the total number of treatments ($r=0.665$, $p=0.000$) as well as $\bar{\alpha}\&\bar{p}\Delta$ pain scale ($r=0.479$, $p=0.000$) and negative correlation with pain scale $\bar{p}$ – treatment ($r=-0.642$, $p=0.000$).

Scatter plots (figure 4) showed an increasing number of treatments with larger force twitches in (SDPPP $\bar{\alpha}\&\bar{p}\Delta$ sum) reduced sessions compared to (SDPPP $\bar{\alpha}\&\bar{p}\Delta$ sum) elevated sessions.

GLOBAL HEALTH PROBLEM LIST

1. Poor recognition that neuromuscular ischaemia at deep MTrPs in tightened/shortened muscles from spondylotic radiculopathy related partial denervation is the root cause of chronic refractory myofascial pain (CRMP).
2. Incognisant that CRMP maintenance/aggravation results from significant mechanical and electrical hyperexcitability of MTrPs related to DS.
3. Lack of innovative, safe and efficacious cost-effective systems for prevention, simultaneous diagnosis, therapy and prognosis in real time for acute and CRMP management.

Figure 3 Spearman correlations of twitch forces to variables recorded.

	Correlation co-efficient (r) N=214	Significance (p) N=214
Total Tx Number	**0.665**	**0.000**
Tx Interval	0.003	0.968
Pain Scale Before Tx	0.015	0.835
Pain Scale After Tx	**-0.642**	**0.000**
Before and After Tx Pain Scale Difference	**0.479**	**0.000**
Before Tx SBP	**-0.261**	**0.000**
Before Tx DBP	-0.097	0.160
Before Tx PP	**-0.234**	**0.001**
Before Tx P	**-0.284**	**0.000**
After Tx SBP	**-0.378**	**0.000**
After Tx DBP	**-0.179**	**0.013**
After Tx PP	**-0.261**	**0.000**
After Tx P	**-0.216**	**0.002**
Tx Interval	0.003	0.968
Tx SessionTime	0.029	0.676

Abbreviations: SBP= systolic blood pressure,DBP= diastolic blood pressure, PP= pulse pressure,P= pulse, SDPPP= Systolic, Diastolic, Pulse Pressure & Pulse, Tx= treatment

Note: Very significant positive correlations between twitch forces and number of treatments and lowering of pain scales after the treatment.
The twitch forces correlated negatively with after treatment pain scale, before and after treatment SBP, PP and P and also to after treatment DBP.

DISCUSSION

Scope of chronic pain

Chronic pain (CP) is a worldwide public health problem affecting physiological, psychological and social well-being.[7] There are 1.5 billion CP sufferers worldwide (American Academy of Pain Medicine website), including 100 million American adults.[8] In the USA, annual CP care is estimated at $635 billion, which is more than the cost/year for cancer, heart disease and diabetes, costing $243, $309 and $188 billion, respectively. The total incremental cost of pain healthcare is $261–$300 billion; private insurers paid the largest share ($112–$129 billion), government programmes (Medicare and Medicaid) bore 25% ($66–$76 billion) and 8% ($20–$23 billion), respectively, with individuals paying an additional $44–$51 billion in out-of-pocket healthcare expense.[8] CP negatively impacts the annual number of work-days, work-hours and wages, resulting in lost productivity of $299–$334 billion.

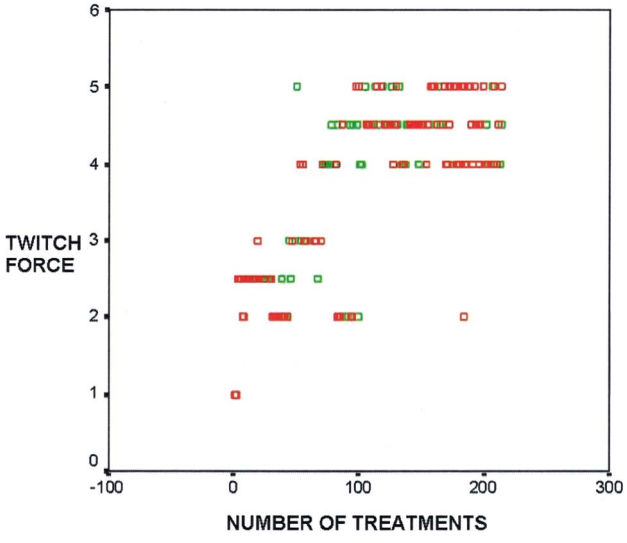


Figure 4 Scatter plot of relationship between twitch forces and number of treatments.

With global child survival improvement and increasing ageing populations, the number of people experiencing LBP and neck pain (NP) will escalate since CP increases with age.[9] CP dominates patients' lives, causing disabilities in family/home responsibilities, occupational, social, recreational, sleep and sexual activities. Pain-related investigations and treatments often make CP worse, affecting patients' interactions with coworkers, physicians, family and social network, creating alienation and isolation.

Constant pain interferes with the ability to concentrate and impairs cognition with mood/memory alterations from effects of medications. The WHO data obtained in primary care centres worldwide show that 22% of all primary care patients suffer from CP. They are four times more likely to have comorbid anxiety/depression than pain-free patients.[10]

LBP causes more global disability than any other condition. NP and LBP have no associated mortality but the morbidity rate for CP is higher than other diseases in the general population.[1] Years lived with disability and disability-adjusted life-years (DALY) are high. In 2010, DALY for NP rose to 33.6 million[3] and 83.0 million for LBP.[2] Systematic reviews of LBP treatments utilised in developed countries and treatments available in developing countries, as well as heat/ice/ultrasound/traction,[11] are discussed later.

Spine X-rays and imaging studies for establishing the presence of intervertebral disc pathology or spinal degenerative diseases for diagnosis of NP and LBP are not available/feasible in resource-poor settings. Despite significant multilevel spine imaging abnormalities, our patient had objective improvements in pain and QOL with eToims, indicating that X-rays/imaging studies correlate poorly with clinical symptoms.

It is essential to authenticate CRMP, the most common type of CP, as a ubiquitous neuromusculoskeletal disease resulting from spondylotic radiculopathies induced partial denervation with DS. Public health priorities necessitate an urgent need for a safe, efficacious, practical and objective cost-effective system with the potential for prevention (pre-rehabilitation) with simultaneous real-time ability to clinically diagnose, treat (rehabilitation) and provide prognosis in acute and CRMP management.

MTrPs/motor point identification

MTrPs are pathognomonic of myofascial pain (MP), clinically identifiable when pressure at this point causes referred pain and snapping palpation of the myofascial band produces a local twitch response.[12] Meta-analysis does not recommend physical examination as a reliable test for diagnosis of MTrPs.[13]

Electrophysiologically, a motor point is where single muscle contractions can occur with minimum intensity and short-duration electrical pulses.[14] Anatomically, it is the area where motor end plates, namely the terminal area of motor nerve fibres, are dense. Electrically evoked single muscle twitch contractions precisely locate MTrPs.

Twitches in DS

Within 6–8 days of denervation, DS develops due to acetylcholine (Ach) receptor increase and decrease in acetylcholinesterase activity.[15] DS can also occur in a prolonged conduction block.[16]

Twitches exercise and stretch individual muscles promoting local blood flow specifically to that muscle. Rat skeletal muscle experiments show that twitch contractions from 1 Hz stimulation increase muscle blood flow by 240%.[17]

eToims twitches in DS

Force, firing pattern, ease/difficulty of twitch elicitation of deep MTrPs objectively aids clinical differentiation of normal condition from partial denervation of spondylotic radiculopathy.[6] Grade 1 twitches result from focalised, partial contraction of stimulated muscle(s) at MTrP. A stronger twitch force on the electrode overlying MTrP with DS gives an asymmetrical, bouncy feedback on the bipolar probe with 6 inches (15 cm) separation between two water-wetted surface electrodes. Grade 2 twitches additionally show rocking/shaking limb and/or trunk movements from stimulation of MTrPs of deep muscles apposed to the bone and joint. Grade 3 twitches produce antigravity limb movements due to whole muscle(s) contraction. This indicates proximal stimulus spread to many and/or larger nerves from antidromic/ephaptic/direct stimulation, and/or distal spread of the current front due to DS. Grade 4 twitches produce antigravity limb movements with the firing rate slower than applied pulses due to the erratic stimulation of MTrPs with DS from the filter effect of tight and stiff overlying tissues. Ability to elicit grade 4 twitches is recognised when joint movements suddenly become stronger. On halting eToims, joint movements continue autonomously, lasting from a few seconds to >10 min before fatiguing. Grade 5 twitches produce antigravity movements with a firing rate faster than applied pulse frequency and rapidly fatigue within a few seconds indicating full, instantaneous depolarisation of MTrPs with DS in the non-tight muscle.

A pre-fatigue phenomenon heralds the onset of grade 5 twitches as multiple twitches/pulse instead of a normal single-twitch/pulse. On continuing stimulation, a sudden increase in the twitch-rate, rhythm and force occurs before erupting into autonomous fatigable twitches. When the twitch-cascade ends, eToims can be reapplied repeatedly for 1–5 min at this motor end plate zone until the entire muscle becomes refractory, at which time a different patient position is used for stimuli to reach other MTrPs with DS within the same muscle.

The pathophysiology of autonomous twitches is similar to cardiac dysrhythmias with problems in impulse re-entry (circus movement along anatomically defined pathways or micro re-entry in continuously varying directions), re-excitation (repetitive activity from local current flow) and/or focal automaticity due to enhanced activity of latent pacemakers from disturbances of ionic currents.[18]

Deep MTrPs are difficult to seek in CRMP due to muscle stiffness, tightness, tenderness and poor tolerance to electrical stimulation. In normal muscles, finding MTrPs is immediate, pleasant and painless. There is a non-forceful symmetrical feedback on both electrodes and grades 3–5 twitches do not occur.

To facilitate twitch elicitation, the joint over which the muscle of interest crosses over is kept at a position advantageous for muscle relaxation as well to incite a mild stretch-tension on the muscle, and stimulation performed along less electrically resistive intermuscular/intramuscular grooves. If elicited twitches are grade 1 force, patient needs to be repositioned differently into in supine/prone/side-lying, sitting, standing, etc, and/or clinician repositioning is necessary to obtain the correct angle to locate/effectively stimulate the MTrP with DS. To obtain pain relief, minimum grade 2 force is essential. Grades 3–5 forces in CRMP will not occur until many professional hours of consecutive treatments. Such twitches are elicitable at acute MTrPs with DS within non-tight muscles.

Stimulus parameters used for evoking twitches are similar to those used in electrodiagnostic medicine for peripheral nerve conduction studies. Repetitive stimulation at 2–3 Hz tests the stability of neuromuscular transmission by temporarily depleting Ach at diseased or immature end plates causing fatigue in neuropathic conditions.[14] Similarly, using 2–3 Hz, fatigable autonomous twitches elicited with eToims signify neurogenic involvement with unstable neuromuscular transmission in CRMP.

MP theories

The integrated hypothesis of Simons *et al*[12] suggests that muscle trauma, overload or strain causes end plate damage, resulting in excessive Ach release. This provokes local, partial muscle fibre contraction beneath the end plate and muscle fibre contracture leads to ischaemia and pain. The neuromuscular junction is the site most susceptible to acute ischaemia.[19] A dysfunctional end plate exhibiting increased Ach release may be the starting point for abnormal regional contractions, which may be essential for the formation of MTrPs.[20]

Gunn postulates that spondylotic radiculopathies cause MP from intramuscular entrapment of nerves and blood vessels. Partial denervation induced shortened/tightened muscle fibres produce tension on pain sensitive regions, for example, annulus fibrosus, bones and joints.[5] Others have also found MTrPs in radiculopathies. Intervertebral disc degeneration, with nerve root compression/angulation from reduced intervertebral space, causes neuropathy which leads to distal muscle spasm in radicular distribution.[21] Pain results from shortened/tight muscle fibres compressing small/large blood vessels leading to ischaemia.

Bradykinin and other neurochemical releases sensitise and/or excite nociceptors.[22]

Systematic reviews of treatments for CRMP

Many methods are available to directly treat MTrPs to inactivate, disrupt or suppress MTrP activity. Systematic reviews have not shown MTrPs treatments with botulinum toxin (Botox),[23] steroids,[24] acupuncture or dry needling[25] to be effective. In order to improve dry needling results in CRMP, the corresponding author first developed automated twitch-obtaining intramuscular stimulation (ATOIMS), which employs mechanical stimulation with a monopolar needle oscillated three times in 2 s. To facilitate twitching, she then created/engineered a needle eToims device that could deliver electrical pulses through a single automatic insertion and retraction of the monopolar needle.[26] These methods were discontinued when she implemented the safe, efficacious, non-traumatic and non-invasive eToims. In this updated eToims acronym, 'T' is capitalised to underscore the importance of twitches. Needling methods cause pain, bleeding, bruising and tissue trauma and are thus not indicated for repetitive/frequent applications throughout the body in patients with CRMP requiring lifelong regular treatments.

Systematic reviews of LBP treatments

Therapies for chronic LBP not showing high quality evidence for improving pain intensity, functional status, global improvement and return to work include lumbar supports,[27] traction,[28] superficial heat and cold,[29] ultrasound,[30] transcutaneous electrical nerve stimulation,[31] low level laser therapy,[32] muscle energy techniques,[33] spinal manipulation techniques[34] and chiropractic treatments.[35]

In acute and chronic LBP, massage improves pain and function only in the short term. Direct manual/mechanical stimulation mobilises superficial muscles but deep massage can produce pain as an adverse event.[36] eToims accurately focalises stimulation to MTrPs with DS and has a minimal tendency to cause post-treatment pain which can be resolved with longer/more treatment sessions.

In neuropathic conditions, in hypertensive patients, and the elderly with significant tightness and stiffness, it is necessary that eToims be applied essentially pain free using only stimulation parameters that the patient can tolerate and settling for grades 1–2 twitches. The probe must be lifted off the skin every 2–4 twitches so that the stimulus on the non-twitching/poor twitching muscle does not undergo repetitive subthreshold stimulation leading to spasm and pain during and after treatment. Patients may tolerate pain during treatment thinking erroneously that enduring strong stimulation will obtain larger twitches. Contrarily, pain-induced involuntary tightening of the muscles during eToims will inhibit deep penetration of electricity into the tissues causing pain during and after treatment. The clinician must watch patients' facial expressions and listen for sighs/moans or objective physical distress signs related to increased sympathetic tone such as pilomotor, vasomotor and sudomotor reflexes and reduce stimulation strength accordingly.

BP and pulse rate reduction have been noted after pain relieving massage attributable to increased parasympathetic tone and sympathetic tone inhibition.[37] Regular exercise in older active individuals lowers both SBP and PP compared to sedentary counterparts.[38] Similarly, regular eToims sessions are useful aerobic exercises that reduce BP and pulse proportional to the twitch force.

There is insufficient evidence to support the use of epidural injection to facet joints and nerve blocks in LBP.[24] The US Food and Drug Administration reports paraplegia, quadriplegia, spinal cord infarction and stroke from technique-related problems such as intrathecal injection, epidural haematoma, direct spinal cord injury, and embolic infarction after inadvertent intra-arterial injection.[39]

Systematic reviews on medications do not show clear evidence that antidepressants,[40] are more effective than placebo in chronic LBP. Non-steroidal anti-inflammatory drugs (NSAIDs) are effective for short-term symptomatic relief in patients with acute and chronic LBP without sciatica.[41] Muscle relaxants are effective in management of non-specific LBP, but adverse effects require cautious use.[42] Opioids compared to NSAIDs or antidepressants did not show differences regarding pain and function. There are no placebo randomised controlled trials (RCTs) supporting the effectiveness and safety of long-term opioid therapy for treatment of chronic LBP.[43]

Gabapentin at doses of 1200 mg or more is effective for some people with some painful neuropathic pain conditions.[44] Gabapentin (1200 mg) use on this patient an hour before eToims reduced pain which facilitated twitch elicitation.

Conflicting evidence exists on the short-term effect of radiofrequency lesioning in chronic LBP and disability of zygapophyseal origin. Intradiscal radiofrequency thermocoagulation is not effective for chronic discogenic LBP.[45]

Evidence for minimally invasive discectomy (MID), although associated with shorter hospital stay, has been found to be inferior in terms of relief of leg pain, LBP and rehospitalisation and our patient fits this profile. More research is needed to define appropriate indications for MID as an alternative to standard open discectomy.[46]

A systematic review of RCTs on stretching suggests that before, after or before-after exercise stretching does not produce clinically important reductions in delayed-onset muscle soreness in healthy adults.[47] Patients with chronic MP who did stretching for 3 weeks did not demonstrate effectiveness in improving muscle extensibility, although stretching increased tolerance to stretch-associated discomfort.[48] When stiff hamstrings are subjected to eccentric exercise, strength loss, pain, muscle tenderness and increased creatine kinase activity occurs. This is consistent with the sarcomere strain theory of muscle damage showing experimental evidence of association between flexibility and a tendency to muscle injury.[49]

Mechanical stretch forces delivered from the surface occur to many muscles simultaneously and are not effective in stretching shortened muscle fibres at deep MrTPs. The solution to make stretching consistently more effective lies in finding new methods including eToims. Effective summation of twitch-induced stretch forces which are focused to MTrPs are best obtained with repetitive 1–3 Hz stimulation.

Not commonly recognised is thixotropy of muscle which is a ubiquitous and functionally important phenomenon since it results from a tendency of actin and myosin filaments to stick together when inactive for a period of time. Passive properties of thixotropy can be reduced with previous movements as evident with preventive warm-up activities of athletes before strenuous sports. Overcoming thixotropy may be the basis by which eToims is able to clinically improve function in muscle tightness without pain, fibromyalgia, stroke or Parkinsonism. Reduced muscle thixotropy/stiffness persists as long as motion

persists but will return to its previous state.[50] Stiffness reduction afforded by a twitch exercise allows more mobility and the increased mobility and increased blood flow perpetuates to improve muscle function and QOL.

Improving denervation supersensitivity related CRMP

Partial denervation and/or conduction block in the presence of DS leads to ongoing MTrPs formation in many myotomes at various times daily with activities of daily living. Morphological and electromyographic studies have demonstrated atrophy and delayed activation of deep muscles of the spine in patients with chronic NP[51] and chronic LBP.[52] A decrease in maximum force of deep back muscles improves resultant joint moments and reduces the stabilisation function provided by these muscles to the lumbar spine.[53] Exercise therapy appears to be slightly effective at decreasing pain and improving function in adults with chronic LBP.[54] There is conflicting evidence for effectiveness of exercise in reducing the number of recurrences or the recurrence rate.[55]

eToims is aerobic exercise therapy to individual muscles. If there is no pain relief with the first eToims session, the primary diagnosis of CP is not CRMP and other causes need consideration, for example, neuropathic, inflammatory, psychiatric or nociceptive. Further eToims sessions are advised even in such patients to treat comorbid CRMP and/or muscle tightness to facilitate management of the primary pain.

Prior to implementation of grades 3–5 twitch elicitation technique, a previous eToims study on 92 patients with CRMP treated during 2006–2008[56] and 133 patients with CRMP treated during 2009–2011 showed that 31% of patients underwent $\geq$10 treatments paying out of pocket in the long term.[6] In comparison, during the same 14-month treatment period, 42 patients with CRMP, who had failed many conventional treatments similar to the presented patient, self-paid $200/h for treatments and 21/42 (50%) self-selected to pay out of pocket for $\geq$10 treatments. Of these, 14/20 patients (67%) were able to empower themselves by learning how to perform self-eToims at no additional cost as part of the eToims programme. They facilitated effects of professional treatments by performing self-eToims for 60 min as a warm-up and a cool-down after the clinician's treatment, similar to common athletic practice.

The eToims treatment model is based on the traditional medical ethics of physician advice patient consent. Since patients paid out of pocket, direct involvement of the patient pocket appears to provide a direct, strong incentive for the patient's active involvement in this relationship. Yet, that relationship could not be maintained over time without the patient's perception of accruing benefit from consenting to treatment. This helps explain the strong patient involvement, demonstrated by regularly keeping eToims appointments and also performing self-treatments.

For best functional results, optimal treatment in CRMP includes these five muscle areas: trapezius, latissimus dorsi, gluteus maximus, adductor magnus, and paraspinal muscles from the neck to sacral areas. This is needed even if the patient presents only with NP/upper limb pain or LBP/lower limb pain as in this patient. Additionally, other muscles connected to the thoracolumbar fascia and along the kinetic chain must be treated proximodistally, starting with the largest muscles that cross multiple joints to small muscles of the hands and feet as needed. Treatments begin with weakened muscles exposed to injurious eccentric contractions before directing treatments to strong muscles used primarily for concentric contractions. In the presence of weak symptomatic side muscles,[52] asymptomatic side muscles are stronger by default and from overuse developing MTrPs that need treatment. This balances the chronic strong pull of muscles towards the asymptomatic side that more weakens the symptomatic side. Treatments begin on the symptomatic side starting with the upper trapezius MTrPs with DS which can be easily located. Through its myofascial connections, other muscles on the symptomatic side become easier to treat. Provided MTrPs with DS are stimulated, grades 3–5 twitch elicitation is facilitated by aged neuromuscular junctions exhibiting enhanced presynaptic nerve terminal branching, postsynaptic distribution of neurotransmitter receptor sites, increased Ach quantal content and a more rapid decline of end plate potential strength during continuous presynaptic neuron stimulation.[57]

Additionally, central sensitisation amplifies DS. Noxious stimuli and/or misinterpretation of non-noxious stimuli (secondary hyperalgesia and allodynia) can induce CP. Injury induced functional and adaptive changes include ineffective synapses unmasking, receptive field shifts and reorganisation or altered effectiveness of surviving neural networks at the brain cortex level as well as at peripheral nerves and receptors.[58]

eToims role in CRMP

With eToims, we have originated an algorithm with consistent pain/discomfort relief and reproducible results without concurrent use of multiple medications or other therapies. The presence of DS in CRMP requires that treatments be safe and effective for regular lifelong use on the entire body. We studied our case with statistical process control (SPC). Studying one case in detail sequentially over time can produce statistical results superior to that of an RCT.[59] In these circumstances, SPC has greater statistical power to exclude chance as an explanation.

Future RCTs could examine specific subgroups related to age (including children), gender, race/ethnicity, culture, socioeconomic status, urban/rural settings, geography, occupational status, mental and physical stress at work, education, comorbid diseases, etc, and include recovery, return to work, QOL and costs of care. In acute MP management, eToims need to be applied as soon as possible, preferably within 24 h of injury while nerves are still viable and are in the stage of conduction block and/or in early stages of partial denervation[60] to prevent progressing into CRMP with DS from ischaemia producing intense muscle spasms.

In developed countries, the ultimate goal of eToims is to have government and private third party payers to cover the cost of eToims treatments and make eToims accessible to all those suffering from acute and CRMP. These payers can easily perform comparative studies to analyse the expenses associated with providing coverage for eToims for a given individual with that which has been paid out already for the same individual for invasive and non-invasive therapies, investigations (radiological and otherwise) and medications.

eToims is suitable for use in developing countries since it is cost-effective. The corresponding author, who is of Myanmar/Burma heritage, has worked with Myanmar patients and academic physicians such that eToims is available at Yangon General Hospitals through approval by the Ministry of Health. Clinicians in other developed and developing nations can utilise a similar model since CRMP is a public health disease. eToims clinical training can be done using online

videos, real-time video-conferencing and corresponding author's review of clinicians short video tapes of eToims treatments on their patients.

Patient's perspective

- I, had a spinal surgery 2 years ago in July 2013. The hypothesis was that my inability to walk uphill effectively was severely compromised by a herniated disc at L4-L5. I had had several injuries and trauma which probably contributed to the problem, whether it was a herniated disc or some other cause of compromising function—especially walking uphill. One of these was an expedition trip to Ecuador during which I was on a boat which slammed up and down for 4 h. I had to tighten my buttock intensely and hold on for the entire boat ride. The next day I was on a horse which trotted causing me more bouncing effects on my spine for 4 h. This was an extreme challenge as I had to tighten my buttock again to endure the bouncing up and down. These two back-to-back incidents followed about 5 months after I fell from a rock climb gym wall. After that fall, I lay on the padded mat for several minutes thinking I had severely hurt myself. However, I was able to get up and I seemed to be okay. I thought that these injuries did not apparently compromise my function. However, on hindsight, it probably did—especially in conjunction with the traumatic incidents in Ecuador which I mentioned. Going further back in time, about 10 years ago, I attempted to water ski and ended up in a very compromising position and felt some tremendous strain on my hamstrings. I let go of the rope and thought that I had damaged my hamstrings severely. However, again, I was able to function and forgot about the injury. Going further back in time, I did 'pull my groin' as they say, in high school football. There was no good treatment available. Lots of heat and inappropriate exercise probably contributed to the injury. However, once again, I moved on because I was generally very fit and probably have a high tolerance for pain and compromised function.
- I have tried just about every treatment possible including many versions of physical therapy, gravity-assisted traction, yoga, Feldenkrais exercises, spinal manipulations, acupuncture with four different practitioners, chiropractic release, medications, epidural injections, many anti-inflammatory medications including opioids and even spinal surgery. In addition, I have a stretching and myofascial release programme which does give me relief. Pain is on my mind 23 h/day and I sought relief with eToims. After treatment with eToims within 6 months, I can feel my affected musculature, namely the gluteal and hamstring muscles, returning to function. The deep twitching has released most of the spasms and the muscles feel more functional and I'm ready to start light exercise. In June of 2015, I went on my first expedition since 2011. I went to Crete and I was able to walk on level and inclines for 3–6 miles daily for 2 weeks. This has been a dramatic development after years of frustration with all the other modalities I tried.

Learning points

- Chronic refractory myofascial pain (CRMP) is a neuromusculoskeletal disease caused by spondylotic radiculopathies following acute or chronic cumulative trauma with denervation supersensitivity induced peripheral and central, mechanical and electrical hyperexcitability.
- The root cause of CRMP is neuromuscular ischaemia at deep myofascial trigger points in tightened/shortened/stiffened muscles from spondylotic radiculopathy related partial denervation that maintains/aggravates CRMP.
- Systematic reviews show lack of effective treatments for CRMP. Since CRMP is a global public health problem with a huge economic toll on society, governments of developed and developing nations should invest in safe, efficacious, cost-effective novel systems such as eToims for its prevention and management.
- eToims is a safe and efficacious innovation for repetitive, lifelong whole body treatments for CRMP management as a real-time preventive, diagnostic, therapeutic and prognostic armamentarium. It empowers patients in their own healthcare since it can also be self-performed.
- The commonly available sphygmomanometer is useful as an inexpensive, practical, objective, real-time pain monitor for clinical follow-up of eToims treatments.

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