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SURFACE ELECTROMYOGRAPHY CHARACTERIZATION OF THE LOCAL TWITCH RESPONSE ELECTED BY TRIGGER POINT INJECTION AND SNAPPING PALPATION IN MYOFASCIAL PAIN PATIENTS

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ABSTRACT OF THESIS

SURFACE ELECTROMYOGRAPHY CHARACTERIZATION OF THE LOCAL TWITCH RESPONSE ELECTED BY TRIGGER POINT INJECTION AND SNAPPING PALPATION IN MYOFASCIAL PAIN PATIENTS

Local twitch responses (LTRs) can be elicited by snapping palpation of myofascial trigger points (TrP) or TrP injections. Objective: To characterize the LTR elicited by TrP injection and snapping palpation on surface electromyography (sEMG) in subjects with myofascial pain in 14 female subjects. Methods: Surface EMG electrodes were placed around the TrP and a control site on the trapezius muscle. Then the following protocol was carried out: tension and contraction of the ipsilateral trapezius muscle, baseline resting activity (five minutes), snapping palpation of the TrP and the control sites, TrP injection, and final resting activity (five minutes). The following data were recorded: pain ratings, areas of referred pain, presence of LTR, and sEMG recordings. Results: During the TrP injection, the investigator found LTRs in only 36% of the subjects, while 64% of the subjects reported that they felt the LTR, and the sEMG recorded only one LTR in one subject. Despite the low percentage of LTRs elicited clinically (36%), a large number of subjects (71%) reported more than 50% immediate reduction in pain intensity after the TrP injection. Conclusion: The sEMG is unable to register the LTR elicited by snapping palpation and TrP injection.

KEYWORDS: Myofascial Pain, Trigger Point Injection, Snapping Palpation, Surface Electromyography, Local Twitch Response

Pei Feng Lim, B.D.S.

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THESIS

Pei Feng Lim, B.D.S.

The Graduate School
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2004

SURFACE ELECTROMYOGRAPHY CHARACTERIZATION OF THE LOCAL TWITCH RESPONSE ELECTED BY TRIGGER POINT INJECTION AND SNAPPING PALPATION IN MYOFASCIAL PAIN PATIENTS

THESIS

A thesis submitted in partial fulfillment of the
requirements for the degree of Master of Science in the
College of Dentistry at the University of Kentucky

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1. Introduction

1.1. Myofascial Pain

Myofascial pain [1 2 3] is a muscle pain disorder characterized by the presence of trigger points (TrP) in taut muscle bands. The taut band refers to a group of tense muscle fibers. The TrP is an area in the taut band that exhibits exquisite spot tenderness on palpation of the taut band. Upon compression of the TrP, referred pain is often elicited in a commonly reported pattern according to the location of the TrP and the muscle. TrPs are classified as latent or active. While both active and latent TrPs can produce pain referral upon digital compression [4], only the active TrP reproduces the clinical pain complaint [1 2 3 5 6 7 8 9].

Numerous theories have attempted to explain the neurophysiology of the myofascial TrP [10 11 12]. The energy crisis theory proposed by Simons [2] states that a local energy crisis is responsible for the formation of myofascial TrPs. Stress or trauma to muscle fibers cause the release of calcium from the sacroplasmic reticulum. This increase in intracellular calcium causes shortening of the myofibrils and increases local metabolic activity. The shortened muscle fibers impair local circulation resulting in ischemia and hypoxia. This sets the stage for a local area of energy crisis (the TrP). Hubbard [13] proposed a muscle spindle hyperactivity theory which states that TrPs are located at the muscle spindles, and the increased activity at the TrP is related to hyperactivity of the muscle spindle. On the other hand, the end-plate hyperactivity [12 14 15] theory states that activity at the TrP is due to hyperactive extrafusal motor end plates.

1.2. Local twitch response (LTR)

The local twitch response (LTR) is a palpable and/or visible transient reflex contraction of the taut band that traverses a TrP [2 16]. This brisk contraction of the taut band (but not the surrounding normal muscle) occurs in response to mechanical stimulation of the TrP by snapping palpation or needling. Mense [3] stated that the LTR can be elicited from both active and latent TrPs. The LTR is considered the most specific clinical test of a trigger point [2 17 18]. Simons [1] regarded the elicitation of a LTR by snapping palpation or needle insertion into the TrP as one of the three minor criteria for the diagnosis of myofascial pain syndrome.

The exact neurophysiological mechanism of the LTR is unclear. Hong and Torigoe [19] investigated the electrophysiological characteristics of the LTR in rabbit skeletal muscles. Mechanical stimulation of the rabbit TrP by

snapping it manually with a blunt probe, tapping it with a blunt probe driven by a solenoid device, or inserting an electromyography (EMG) needle into the TrP produced visible and needle EMG (nEMG) demonstrable rabbit-LTRs in the responsive bands. In the rabbit, the LTR seems to be mediated via a spinal reflex [19] [20]. In a case report, Hong [21] investigated the LTR produced by snapping palpation using nEMG in a patient with complete loss of nerve conduction involving the posterior cord of the right brachial plexus. At six months following injury, nEMG activity of the LTRs in the third finger extensor digitorum communis muscle was significantly reduced on the paralyzed side compared with the normal side. However, at seven, eight and seventeen months post-injury, when the sensory and motor functions had recovered considerably, EMG activity of LTRs was similar on both sides. The author concluded that the LTR in humans is also mediated by the spinal cord, similar to LTRs in rabbits. It is likely a polysynaptic spinal reflex activity, but the exact pathway and interneurones involved in the spinal cord are unknown [22] [16]. Rivner [10] hypothesized that the LTR may be a miniature stretch reflex.

Snapping palpation is performed with the tip of the finger pulled across the muscle fibers at a right angle to the direction of the fibers [2]. However, it is very difficult to reliably elicit a LTR by snapping palpation [6] [23] [24]. Hong et al [25] found that LTR was more frequently elicited by needling than by palpation. They reported that LTR was elicited in 39% of the TrPs by snapping palpation, and in 100% of the TrPs by TrP injection and needling. Fricton et al [26] recorded the LTR elicited by snapping palpation of taut bands with trigger points versus normal muscle using nEMG. Results showed that the palpable band had statistically significantly higher motor activity while the normal muscles showed minimal or no activity. Simons & Dexter [27] [28] compared the nEMG and surface EMG (sEMG) characteristics of LTR elicited by snapping palpation and needling in nine subjects. They reported that the number, duration and density of the discharges were the same whether the LTR was elicited by snapping palpation or needling. Very little or no EMG activity was detected by the surface electrode compared to the intramuscular (needle) electrode.

The LTR is believed to inactivate the myofascial TrP. Electrophysiological studies have demonstrated spontaneous electrical activity (SEA) unique to animal TrSs [29] [30] and human TrPs [31] [32] [33]. This is also referred to as the endplate noise [34]. Chen et al [35] studied the effect of dry needling on the SEA of the rabbit myofascial TrS using nEMG. The authors described repetitive and rapid needle insertion into the experimental TrSs in order to elicit LTRs. Control TrSs were stimulated by very slow needle insertion for minimal LTR elicitation. A mean of 30.2 ± 4.7 LTRs were elicited in the experimental TrSs, while a mean of 8.4 ± 1.4 LTRs were elicited in the control TrS. The study concluded that rapid needling to produce multiple LTRs resulted in statistically significant inhibition of the SEA of the TrSs, compared to the control TrSs which were minimally needled. The authors believed that the multiple LTRs elicited were responsible

for the reduction in electrical activity, rather than other factors such as traumatic effects of needling (e.g. edema, hematoma formation). Later studies showed that verapamil (calcium channel blocker) [36], phentolamine (sympatholytic agent) [30] and botulinum toxin (Botox) [37] also effectively inhibited the SEA of the myofascial TrSs. Possibly, LTRs, calcium channel blockers, sympatholytic agents and Botox [37 38 39 40 41 42 43 44 45] have similar ability to reduce TrP activity.

Both dry needling [17 46 47] and injection of the TrP with local anesthetic solution [17 48 49 50 51] have been shown to effectively alleviate myofascial pain. Esenyel et al reported [50] statistical significant reduction in pain intensity following TrP injection at two weeks and at three months. Wreje and Brorsson's [52] study showed that pain reduction persisted at two weeks following TrP injection. Considerations and recommendations regarding TrP injections have been reported previously [53 7]. Fine et al [54] reported that the effects of trigger point injections were naloxone reversible, suggesting that the pain reduction was mediated by an endogenous opioid system. In a review article on TrP injections, Cummings [55] concluded that the needling or placebo effect is more important than the type of agent injected. The presence/absence of LTR was not recorded in these studies.

Hong [17] investigated the importance of the LTR in a study which compared lidocaine injection to dry needling of the myofascial TrP. Visible and/or palpable LTR was not elicited in nine (26%) out of 35 subjects who received lidocaine injection and eight (35%) out of 23 subjects who received dry needling. The results showed that there were little or no immediate treatment effects in these subjects where no LTR was elicited. Further injection or needling was continued in these subjects in an attempt to elicit the LTR because the authors felt that the lack of immediate therapeutic effect would probably signify a lack of therapeutic effect at 2 weeks post injection. The study, however, did not investigate the amount of pain reduction at 2 weeks in subjects in whom no LTR was elicited. Further investigation is also needed to determine if the extensive needling described by Hong [17] produces tissue damage. In a separate review, Hong [53] speculated that rapid insertion of the needle minimized muscle fiber damage. However, he provided no scientific data to support this statement. In his opinion and in Simons' [12] experience, LTRs were elicited more easily if the needle was moved quickly rather than slowly. Simons [12] suggested that Hong's needling technique using small needles will cause less muscle fiber trauma because when the needle elicits a LTR, the fast-out stroke withdraws the needle from the taut band before the contracting muscle can damage itself by pulling against the needle. However, this theory needs to be validated with further research. Also in Hong's study [17], dry needling was found to require more needle insertions to inactivate the TrP than TrP injection, and hence the former group suffered greater intensity and longer duration of post-injection soreness.

Due to studies which stress the importance of the LTR during TrP injections, numerous review articles [56 57 58 59 60 61 16 5 62 63 64] on trigger point injections have recommended eliciting the LTR, and numerous research papers [65 17 25 47 66] investigating TrP injections have included eliciting the LTR as a part of their protocol. Borg-Stein and Stein [59] recommended repeated insertions of the needle "until a twitch response can no longer be elicited". Fischer [61] stated that the LTR "proves that the needle is on target". Ruane and Roberts [62] stated that the LTR "verifies correct needle placement". Doggweiler-Wiygul [63] advised that the LTR was used to "verify successful needle piercing of a TrP", and routinely warns the patient that the TrP injection may cause the muscle to twitch. Schneider [60] cautioned that "as long as the LTR can continue to be elicited, the TrP has not been eliminated…. If the LTR is still present after the (TrP) injection, the patient will still have pain and the TrP nodule will remain palpable". However, none of these claims were substantiated by scientific data.

In fact, the LTR became so important that classification of response to TrP palpation began to incorporate the presence or absence of LTR. In Ardic's study [66] comparing the effects of two techniques of electrotherapy on myofascial pain, TrPs were classified according to a four level scale: 0 – no pain with palpation; 1- pain with palpation but no LTR; 2- pain with palpation and LTR; and 3- LTR even with light touch. Remember that back in 1990, Simons [1] considered the elicitation of a LTR by snapping palpation or needle insertion into the TrP as one of the three *minor* criteria for the diagnosis of myofascial pain syndrome. Even in 1999, the 2nd edition of Travell and Simons' Trigger point manual [2] listed the LTR as a confirmatory rather than a essential criterion. Based on Gunn's [67] work on intramuscular stimulation and the belief in the therapeutic importance of the LTR, Chu reported a technique, known as Twitch-Obtaining Intramuscular Stimulation (TOIMS), in which neurogenically evoked muscle twitches was used to relieve myofascial pain [68 69 70 71 72]. The technique uses repetitive mechanical stimulation to elicit twitch responses at abnormally excitable motor end-plate zones. The author believed that twitches produce exercise-induced stretch to the shortened muscle fibers, and thus restore the muscle fibers to optimal length. The efficacy of Chu's technique in the management of myofascial pain remains to be established using sound research principles. At present, there is inadequate data to help in understanding the therapeutic effect of LTRs, if any.

In fact, numerous studies investigating the effects of TrP injections did not mention eliciting the LTR as part of their protocol [73 74 75 76 77 54 46 48 78 52 79 80 81 82 51 50]. Nevertheless, the subjects in these studies also reported pain reduction following the TrP injection. Moreover, Campbell [18] recommended infiltration of the TrP until the area was no longer tender and neither mentioned the fast-in-fast-out needling technique nor the LTR.

Studies investigating the effect of acupuncture on myofascial TrPs have reported it's success in relieving myofascial pain [83] [47]. Based on Hong's study [17], Irnich et al [47] compared the immediate effects of dry needling and acupuncture at distant sites on neck pain. In the dry needling group, the needle was inserted and manipulated until at least one LTR was elicited. The authors found acupuncture to be superior to dry needling. Acupuncture techniques described in these studies did not involve elicitation of the LTR either. In a survey of 1663 American Pain Society members conducted by Harden et al [84] in 2000, only 14.6% of the respondents felt that the LTR was essential to the diagnosis of myofascial pain. 62.6% felt that the LTR was associated with the diagnosis and 22.8% felt that it was irrelevant to the diagnosis.

In order to study the LTR, the method of recording its presence is important. The LTR has been very well characterized in nEMG studies in animals [19] [20] [35], especially in rabbits. Both nEMG [85] [21] [26] [27] [28] and sEMG [27] [28] have been used in investigations of the LTR phenomenon in humans with myofascial pain. Objective measurement of the LTR (e.g. using sEMG or nEMG) avoids examiner bias. The nEMG method is precise, but technique sensitive and invasive [29] [14] [34] [27]. It is difficult to insert the recording needle electrode close enough to the TrP to measure its activity, and yet not too close such that it disturbs the TrP activity. The very insertion of the needle electrode close to the TrP often produces a LTR [29] [36]. This is a distinct disadvantage since the object of interest is the LTR. In the nEMG studies, the LTR is described as a transient burst of EMG activity [21] [27] [19] [20]. The sEMG, on the other hand, is noninvasive, less technique sensitive, and easy to use clinically [86]. Fujimoto and Nishizono [87] found that surface electrodes was comparable to needle electrodes in measuring muscle contractions. In addition, surface electrodes do not interfere with the activity of the TrP. A few studies used sEMG successfully to investigate the effect of TrP injections on muscle activity. Hendler et al [76] recorded the resting activity of the trapezius muscle (harboring TrPs) using sEMG before and after TrP injection in the same muscle. The authors showed that resting muscle activity in the trapezius decreased after injection. Carlson et al [49] recorded resting sEMG activity in painful masseter muscles before and after trigger point injection in ipsilateral trapezius TrPs. They reported reduction of resting EMG activity in masseter following trapezius TrP injection. Neither study assessed the presence or absence of LTRs. Dexter and Simons [27] [28] [85] reported no or only a distant response being recorded using the sEMG electrode to record the LTR elicited by snapping palpation and needle penetration of the trigger point. Yet Hong's [17] study, which documented the LTR based on clinical observation (visible and/or palpable LTR), reported that approximately 5-30 LTRs were elicited from the needling or lidocaine injection of the TrP. This reveals an interesting discrepancy in these reports since one would think that sEMG should be more sensitive in detecting muscle twitch response than the human eye or hand.

1.3. Objectives

The aim of this study is to characterize the LTR elicited by TrP injection and snapping palpation on sEMG in subjects with myofascial pain. This is to determine if sEMG can be used as an objective clinical tool to study the LTR.

2. Methods

2.1. Study protocol and consenting procedures

The experimental protocol, informed consent form, Health Insurance Portability and Accountability Act (HIPAA) Authorization form, and advertisement for this study were reviewed and approved by the Human Investigational Review Boards (IRB) of the University of Kentucky. All subjects were provided with oral and written information describing the nature and duration of the study. Subjects' informed consent was obtained at the University of Kentucky Orofacial Pain Center and documented on the consent form. This study was conducted between January 2003 and May 2004 at the University of Kentucky Orofacial Pain Center. One investigator carried out all procedures in the protocol. Subjects were paid $80 for their participation in this study.

2.2. Subject recruitment and inclusion/exclusion criteria

Subjects were recruited by flyers placed at the Chandler Medical Center and the University of Kentucky campus grounds, and from patients who sought consultation or treatment at the University of Kentucky Orofacial Pain Center. Inclusion criteria for enrollment were: (a) female, age 18 years or older; (b) diagnosis of myofascial pain based on the following criteria [2]: exquisite spot tenderness on palpable taut muscle bands (known as a TrP) in the upper trapezius muscle, and restriction and/or pain or stiffness on cervical range of motion; and (c) an active TrP in the upper trapezius muscle, based on the subject's recognition of her pain complaint or reproduction / intensification of her usual pain on digital compression of the TrP.

Subjects were excluded from this study if they had: (a) significant medical illness (e.g. heart disease, cancer); (b) extreme fear of needles; (c) allergy to local anesthetics; (d) bleeding disorders or tendencies (e.g. hemophilia, thrombocytopenia, anticoagulant therapy); (e) cognitive impairment or exhibited inadequate cooperation; (f) fibromyalgia; (g) acute trauma and/or infection in the trapezius muscle (TrP region); (h) cervical spine injury and/or surgery within last one year (e.g. cervical radiculopathy or myelopathy, segmental instability, fracture and/or surgery of the cervical spine); and (i) a cardiac pacemaker or other electronic devices implanted into the body that interferes with sEMG recording.

2.3. Armamentarium

Armamentarium consisted of the following (see Figure 1): skin pen, ruler, alcohol swab, sterile gauze, 1 ml of 2% lidocaine HCl without epinephrine (Xylocaine. Lidocaine HCl injection USP. AstraZeneca LP, Wilmington, DE 19850), 27-gauge (2.5cm) needle, needle protector, sEMG electrodes (HC-1 Individual disposable silver/silver chloride electrodes. American Biotec Corporation, 24 Browning Drive, Ossining, NY 10562), electrolyte (Staodyn Conductive Gel. Staodynamics, Inc. Longmont, CO 80501 USA), and sEMG (MP Systems. AcqKnowledge software. Biopac Systems, Inc. 42 Aero Camino, Goleta, CA 93117.) (see Figure 2).

2.4. Pain and medical history

All subjects were interviewed regarding their pain and medical history. Subjects were asked questions regarding the location, onset, quality (e.g. dull, aching, sharp, shooting, burning, throbbing, etc), intensity (based on a 0 to 10 Verbal Analogue Scale (VAS), with '0' being no pain and '10' being the worst possible pain), frequency, and duration of their pain. Precipitating factors, aggravating factors, relieving factors, and other associated factors were also noted. Subjects' medical history status was also obtained.

2.5. Muscle palpation

All subjects were placed in a semi supine position throughout the examination [88]. The following non-biased statement [89] was read to the subjects prior to muscle palpation: "Sometimes when we push on sore spots in muscles you may feel pain somewhere else. I am going to push on a sore spot in the muscle. Let me know if you feel pain in an area of your head away from where I am pushing". Palpation of the upper trapezius muscle was carried out with approximately 2 lbs. of digital pressure [90] while the subject rated the pain on palpation on a 0-3 rating scale ("0" being no pain/tenderness on palpation; "1" being tenderness on palpation; "2" being pain on palpation; and "3" being pain on palpation with the subject withdrawing from the external pressure applied to the muscle).

TrPs and their pattern of pain referral on palpation were charted. An active TrP in the upper trapezius muscle was defined by the following criteria [2]: exquisite spot tenderness on palpable taut muscle bands in the upper trapezius muscle, with subject's recognition of her pain complaint or reproduction/intensification of her usual pain on digital compression of the TrP. For subjects with bilateral active TrP in the trapezius muscles, the more painful side was used

as the experimental side. The right TrP was used as the experimental side when the subject reported same pain intensity on both sides. The TrP was located by palpation and marked as a dot on the skin with a skin pen (see Figure 3). An ipsilateral site, 2 cm away from the TrP, but not on the same taut band, was marked as a cross on the skin. This latter site is known as the control site.

2.6. Surface electromyography (sEMG) recording

Surface EMG recording was carried out according to the guidelines by Van Boxtel [91]. The skin around the TrP and the control site region was cleansed with alcohol. One set of sEMG electrodes (sEMG 1) was placed around TrP (previously marked with a dot on the skin), 3 cm mesial and 3 cm distal to the TrP (see Figures 4, 6, and 7). A second set of electrodes (sEMG 2) was similarly placed around the control site (previously marked with a cross on the skin) (see Figures 5, 6, and 7). These electrodes were placed parallel to the trapezius muscle fibers. Each ground lead was placed at an equal distance from the two active leads. The same set of recording electrodes, sEMG 1 and sEMG 2, were used for all subjects. sEMG recording was continuous throughout the study. Subjects were positioned such that they could not view the monitor of the sEMG recording. sEMG signals were amplified (gain = 1000) and passed through a 10-5000 Hz bandpass filter. The sampling rate was 20/s.

The following records were made in the same order for all subjects: (a) a 5 second sEMG recording with the subject tensing the ipsilateral trapezius muscle without visible shoulder movement; (b) a 5 second sEMG recording with the ipsilateral shoulder lifted towards the subject's ear (see Figure 9); and (c) a 5 minute resting sEMG recording with the subject resting quietly with her teeth apart and mandible relaxed [92 93] (see Figures 10 and 11).

2.7. Snapping palpation

Snapping palpation was performed with the tip of the investigator's finger pulled across the trapezius muscle fibers, perpendicular to the direction of the fibers [2]. This was carried out, with simultaneous sEMG recording, twice on the TrP site (see Figure 12) and then twice on the control site (see Figure 13) in the same order in all subjects.

2.8. TrP Injection

Subjects were asked to note the pattern of referred pain or pain intensification produced (if any) during the injection. All subjects were read the following statement: “During the injection, you may feel pain somewhere else. If you feel pain in an area of your head away from the injection site, mark it with an ‘X’ on the drawing of the face where you felt the pain. You may mark as many places as you wish or no place at all if there is no pain in any area away from the injection site”. They were also told to note the presence or absence of twitching of the trapezius muscle during the TrP injection. The following statement was read to each subject: “A muscle twitch is a sudden contraction of part of the muscle. During the injection, you may feel the shoulder muscle twitch. After the injection, mark on the chart whether or not you felt the shoulder muscle twitch”. Subjects were instructed to record this information on a pain diagram after the injection was carried out. They were then instructed not to move their body during the injection.

One injection was performed at the TrP site for each subject, with simultaneous sEMG recording. The TrP site was cleansed with an alcohol swab (see Figure 14) . Then, the TrP was framed by two fingers flat on the muscle (see Figure 15) . Following that, local anesthetic was injected while using the tip of the needle to penetrate the TrP in a fast-in-fast-out manner [2] (see Figure 16). The presence of any LTR (visible and/or palpable) during the injection was noted by the investigator, and then recorded on the chart after the injection. Post injection hemostasis was achieved by firm compression of the injection site for 10-15 seconds (see Figure 17).

2.9. Post-injection procedures

Following the TrP injection, resting sEMG was recorded for 5 minutes with the subject resting quietly with her teeth apart and mandible relaxed. sEMG electrodes were removed after recording. A pain diagram was then presented to the subject for the purpose of drawing the pattern of referred pain or pain intensification produced during the injection (if any), and for documenting the subject’s perception of the presence or absence of twitching of the trapezius muscle during the TrP injection. The subjects were blinded to the investigator’s record of the presence or absence of LTR during the TrP injection. The investigator was also blinded to the subject’s record of the presence or absence of LTR. Finally, subjects were asked to state their current pain intensity on a 0 to 10 VAS (‘0’ being no pain; ‘10’ being the worst possible pain).

2.10. Statistical analysis

Statistical analyses were performed with SPSS for windows 11.5 program (SPSS Inc., Chicago, IL). The data were analyzed using Student's *t*-tests and Pearson's correlation coefficients. Significance level was set at $p<0.05$ for all analyses.

Figure 1. Armamentarium (see text for description).

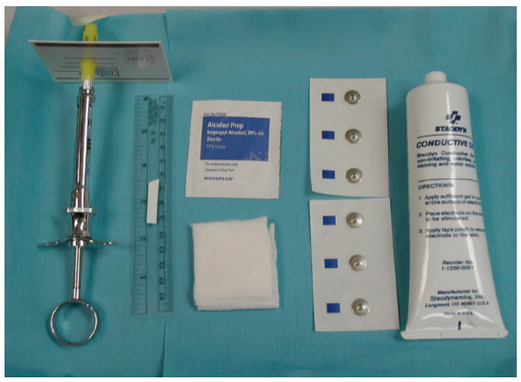


Figure 2. sEMG equipment.

Figure 3. TrP site marked as a dot and control site marked as a cross on the skin.

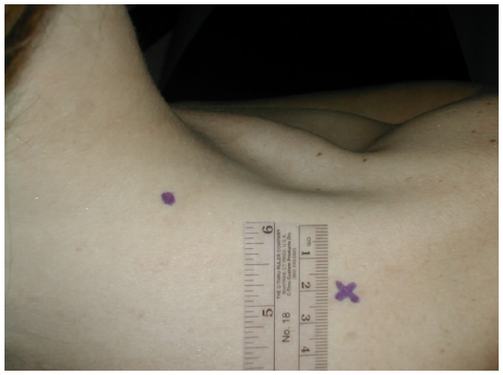


Figure 4. Sites marked for the placement of sEMG electrodes 3cm mesial and 3cm distal to the TrP (white arrows).

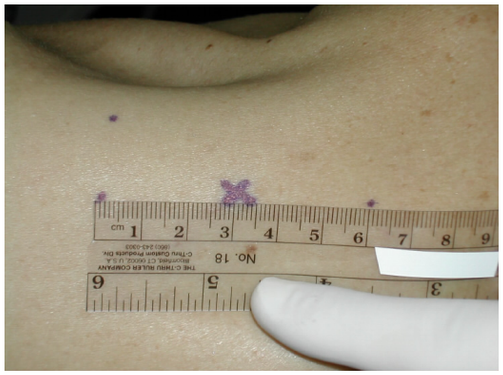


Figure 5. Sites marked for the placement of sEMG electrodes 3cm mesial and 3cm distal to the control site (white arrows).

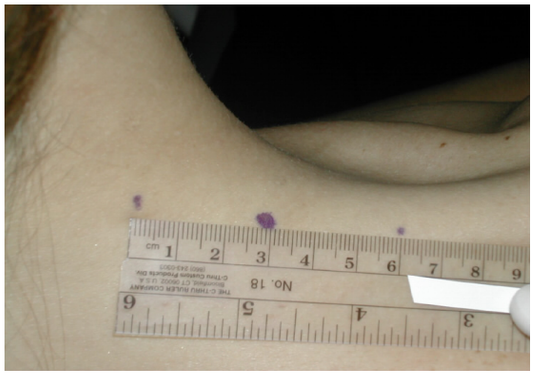


Figure 6. Sites marked for the placement of sEMG electrodes.

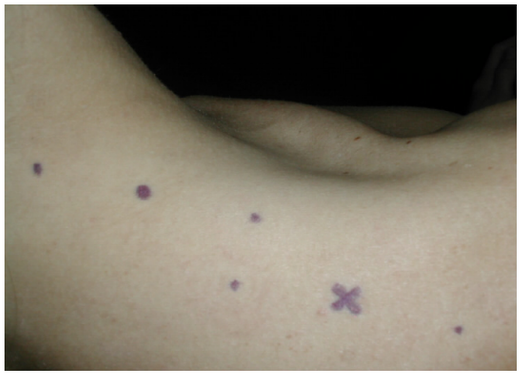


Figure 7. Placement of sEMG 1 around the TrP.

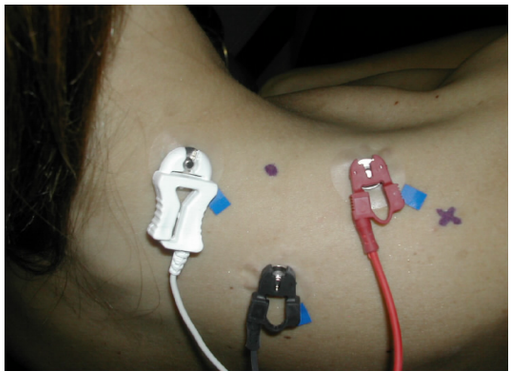


Figure 8. Placement of sEMG 1 and sEMG 2 around the TrP and the control sites.

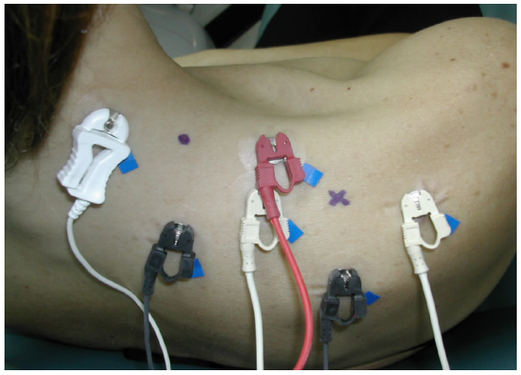


Figure 9. sEMG recording with the subject lifting her shoulder towards her ear.

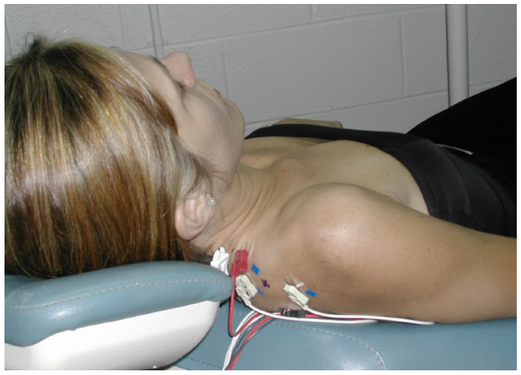


Figure 10. sEMG recording with the subject resting quietly.

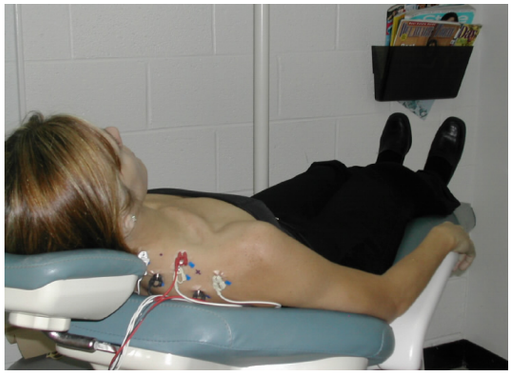


Figure 11. sEMG recording monitor.

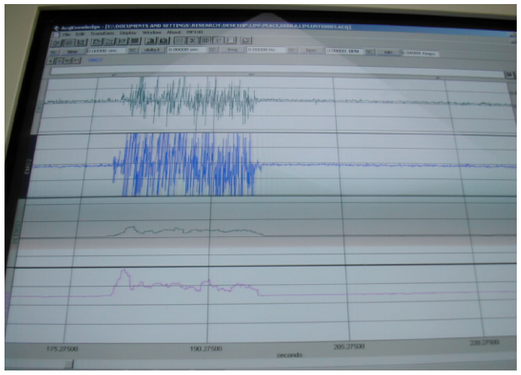


Figure 12. Snapping palpation on the TrP site.

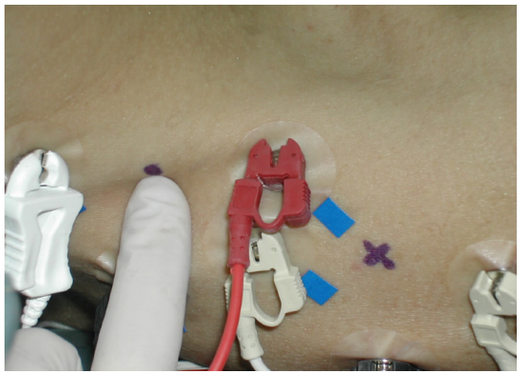


Figure 13. Snapping palpation on the control site.

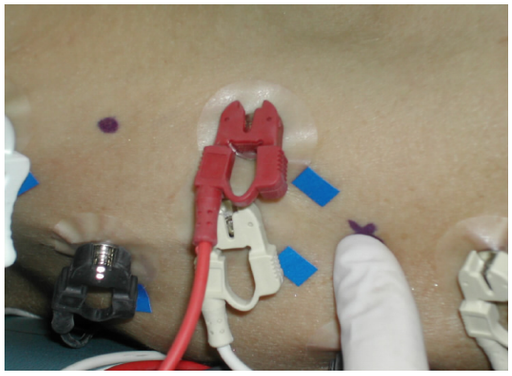


Figure 14. TrP site cleansed with an alcohol swab.

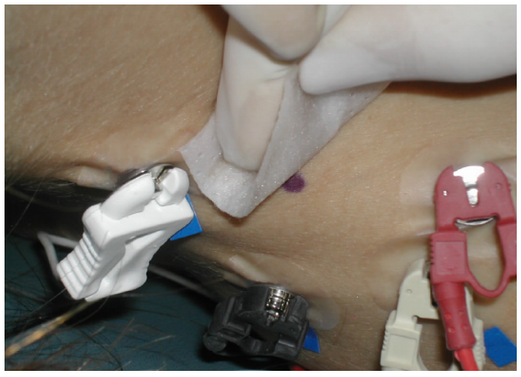


Figure 15. Framing the TrP with two fingers flat on the muscle.

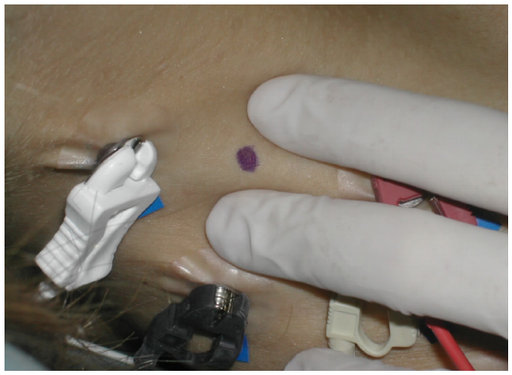
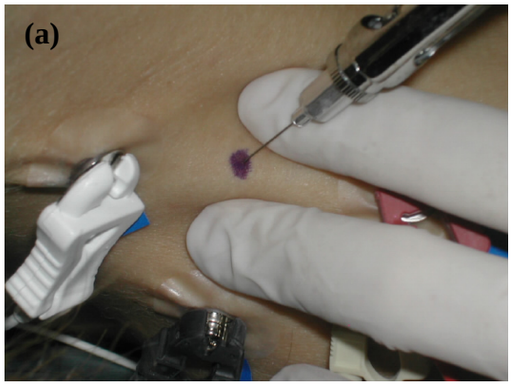


Figure 16 (a) and (b). TrP injection carried out in a fast-in-fast-out manner.



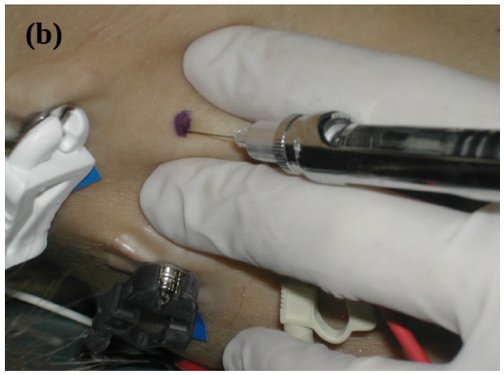
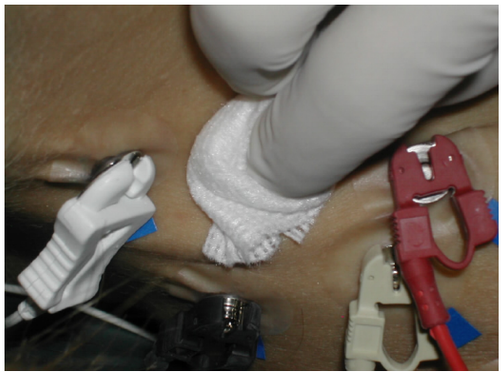


Figure 17. Post injection hemostasis achieved by firm compression of the injection site.



3. Results

3.1. Demographic and medical history data

Of the 17 subjects recruited for this study, three subjects were excluded due to incorrect recording of a defective control sEMG electrode (sEMG 2), which was subsequently replaced. The mean age of the remaining 14 subjects was 32.9 years with a range of 23 – 50 years. Eight (57%) of these subjects were married and 10 (71%) of them were employed. None were reportedly disabled or involved in litigation. Four (29%) subjects were smokers, three (21%) subjects consumed alcohol regularly, and 13 (93%) subjects consumed caffeinated beverages daily.

Table 1 shows the various medical disorders reported by these subjects. Eight (57%) subjects reported sinus trouble. At the time of the study, three (21%) subjects were being treated for both anxiety disorders and depression, and one (7%) subject was treated for depression only. None of the subjects reported to be pregnant. Table 2 shows the subjects' current medications.

Table 3 lists the number of subjects who responded positively to the seven questions related to sleep in the Pain Questionnaire (see Appendix 4). Eight (57%) subjects responded positively to three or more of the seven questions. One (7%) subject responded positively to all, whilst another responded positively to none, of the sleep questions.

Table 4 lists the number of subjects who responded positively to the nine questions related to stress in the Pain Questionnaire (see Appendix 4). Five (36%) subjects responded positively to four or more of the questions. Two (14%) subjects did not endorse any of the questions.

Table 1. Medical disorders.

Medical disorder	**Number of subjects (%)**
Sinus trouble	8 (57%)
Hay fever	4 (29%)
Depression	4 (29%)
Anxiety disorder	3 (21%)
Anemia	3 (21%)
Vision problems	3 (21%)

Table 2. Current medications.

Medications	**Number of subjects (%)**
Antidepressant	5 (36%)
Oral contraceptive	4 (29%)
NSAIDs	4 (29%)
Muscle relaxant	3 (21%)
Antihistamine	3 (21%)
Benzodiazepine	3 (21%)
Narcotic analgesic	1 (7%)

Table 3. Questions related to sleep in the pain questionnaire.

Question	**Number of subjects who responded positively to the question (%)**
Do not feel rested in the morning	10 (71%)
Do not sleep well	6 (43%)
Restless sleeper	6 (43%)
Pain interferes with sleep	5 (36%)
Awaken frequently during the night	5 (36%)
Vivid dreams or nightmares	4 (29%)
Go to bed more tired than daily activities justify	2 (14%)

Table 4. Questions related to stress in the pain questionnaire.

Question	**Number of subjects who responded positively to the question (%)**
Stress makes the pain worse	11 (79%)
Feel I am under stress much of the time	10 (71%)
My hands and feet are often cold or hard to keep warm	6 (43%)
The pain prevents me from performing my normal activities	4 (29%)
Feel lightheaded or dizzy	3 (21%)
Feel depressed much of the time	2 (14%)
Have been under the care of a psychiatrist or psychologist	2 (14%)
Do not enjoy my job	1 (7%)
There are times when I feel as though I cannot breathe in enough air	1 (7%)

3.2. Pain characteristics

Although all subjects had an active TrP in their trapezius muscle, only nine (64%) subjects presented with neck and shoulder pain. Nine (64%) subjects had headaches (three temporal, one frontal, two fronto-temporal, one temporal-occipital, one frontal and retro-ocular, one occipital), four (29%) subjects had face pain (two subjects reported pain in the masseter region and two subject reported in the mandibular region), and one (7%) subject reported an earache.

Five (36%) subjects presented with unilateral pain, and nine (64%) subjects had bilateral pain. Four of the latter subjects reported the same pain intensity on both sides and hence, their right side was used as the experimental side. The mean pain intensity at baseline was a mean of 5.6/10 (s.d. = 1.91) ranging between 3–9/10. There was a statistically significant negative correlation (Pearson's correlation; $p = 0.027$; $r = -0.588$) between the initial pain intensity and the mean baseline resting sEMG activity at the TrP site (sEMG 1), i.e. subjects who reported higher initial pain intensity had lower muscle activity at the TrP site. There was no significant correlation (Pearson's correlation; $p = 0.376$; $r = 0.257$) between the initial pain intensity and the mean baseline resting sEMG activity at the control site (sEMG 2).

3.3. sEMG data

Muscle activity at the control site remained largely unchanged from the baseline resting record to the final resting record (see Table 5). However, there was a statistically significant decrease in the muscle activity from the baseline to the final resting activity (*t*-test; $p = 0.008$). The mean sEMG activity during contraction was three times higher than that during the mean baseline resting activity.

Muscle activity at the TrP site (see Table 6) showed a significant increase from the baseline resting activity during palpation of the TrP site (*t*-test; $p = 0.013$) and during the TrP injection (*t*-test; $p = 0.002$). Muscle activity during the TrP injection was also statistically significantly higher than during the final resting activity (*t*-test; $p = 0.001$). The mean sEMG activity during contraction was 7.5 times higher than that during the mean baseline resting activity.

The sEMG data for all subjects during snapping palpation of the TrP and control sites and during the TrP injection are shown on Figure 18. We did not observe a trend in the sEMG tracings on sEMG 1 (electrode placed around the TrP

site) and sEMG 2 (electrode placed around the control site) during snapping palpation of the TrP and control sites, and during the TrP injection.

The sEMG tracings did not show any burst in activity in the electrode around the TrP site (sEMG 1) relative to the electrode around the control site (sEMG 2) during snapping palpation of the TrP and the control sites. In fact, in Subject 1, a burst of activity was recorded at the control site (sEMG 2) when snapping palpation was performed at the TrP site. Only in Subject 13 did there appear to be a burst of activity at the TrP site (sEMG 1) compared to the control site (sEMG 2) during snapping palpation of the TrP site. The sEMG tracings at sEMG 1 and sEMG 2 appeared similar in subjects 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12 and 14 during snapping palpation of the TrP site.

There was no observable trend in the sEMG tracing during the TrP injection. Subject 1's sEMG 1 tracing appeared to have captured a burst of activity at the TrP site without a corresponding burst of activity at the control site (sEMG 2). But both the investigator and the subject failed to record a LTR (Table 7). The sEMG tracing in Subject 6 shows a burst of activity at sEMG 1 but no corresponding burst of activity at sEMG 2. For this subject, both the investigator and the subject indicated that the LTR was present during the TrP injection (Table 7). This seemed to be the only subject in whom the LTR was both clinically present, as recorded by the investigator and subject, and appeared as a burst of activity at the TrP site only (sEMG 1) on sEMG recording. The sEMG tracings of the TrP site (sEMG 1) and the control site (sEMG 2) appeared similar in subjects 2, 3, 4, 5, 7, 8, 9, 10, 11, 12, 13 and 14 during the TrP injection.

Table 5. sEMG recording at the control site (sEMG 2).

	Mean (μV)	s.d. (μV)	Range (μV)
5 sec tension	50.34	12.42	44.50 – 91.90
5 sec contraction	137.46	105.55	46.90 – 390.40
5 min baseline resting	45.58	0.87	44.60 – 48.30
Palpation of TrP site	45.57	1.59	42.60 – 48.70
Palpation of control site	45.86	1.39	44.10 – 48.70
TrP injection	45.47	0.72	44.40 – 46.90
5 min final resting	45.39	0.89	44.20 – 48.00

Table 6. sEMG recording at the TrP site (sEMG 1).

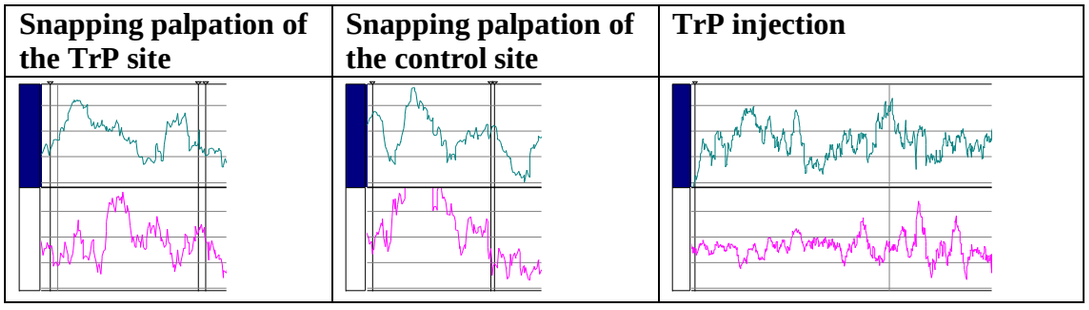
	Mean (μV)	s.d. (μV)	Range (μV)
5 sec tension	12.75	5.13	6.90 – 26.90
5 sec contraction	55.21	46.13	16.80 – 188.70
5 min baseline resting	7.41	0.50	6.80 – 8.40
Palpation of TrP site	8.64	1.80	5.50 – 11.20
Palpation of control site	7.88	1.16	6.20 – 10.10
TrP injection	8.13	0.81	6.70 – 9.50
5 min final resting	7.36	0.59	6.70 – 8.80

Table 7. Presence or absence of LTR based on investigator (a) and subject's (b) evaluation; (c) subject's report of the presence/absence of referred pain; and (d) subjects reporting ≥50% decrease in pain intensity on the VAS. '+' indicates present; '-' indicates absence.

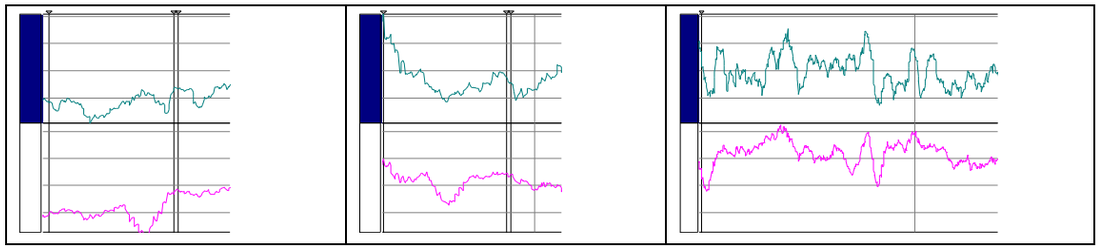
Subject	(a) Investigator LTR	(b) Subject LTR	(c) Subject referred pain	(d) ≥50% ↓ in pain VAS
1	-	-	+	+
2	-	+	+	+
3	+	+	+	+
4	-	-	+	+
5	-	+	+	+
6	+	+	-	+
7	+	-	+	+
8	+	+	+	-
9	-	+	-	-
10	+	+	-	-
11	-	-	+	+
12	-	-	+	-
13	-	+	+	+
14	-	+	-	+

Figure 18. sEMG record of snapping palpation of the TrP site and control site, and the TrP injection. The single vertical dotted line marks the start of the palpation or injection. Double vertical dotted lines mark the end of the palpation. sEMG 1 around the TrP site is displayed in blue, while sEMG 2 around the control site is displayed in pink.

Subject 1



Subject 2



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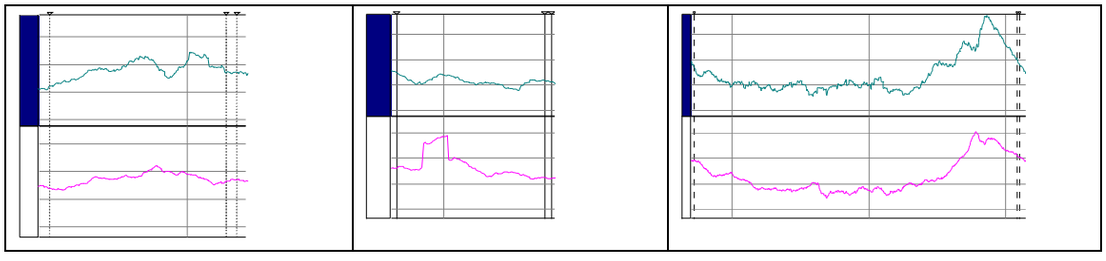


Subject 4

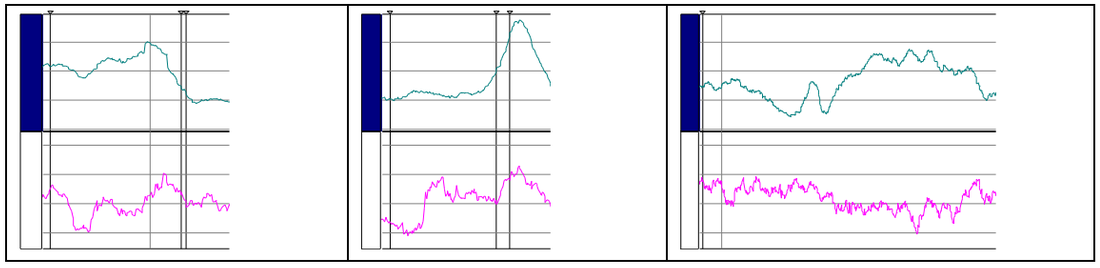
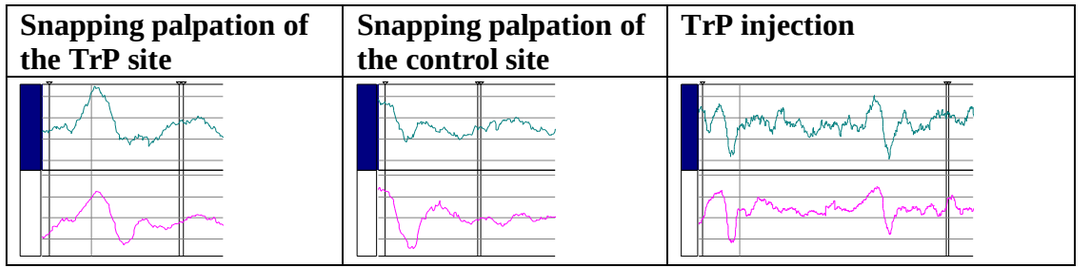
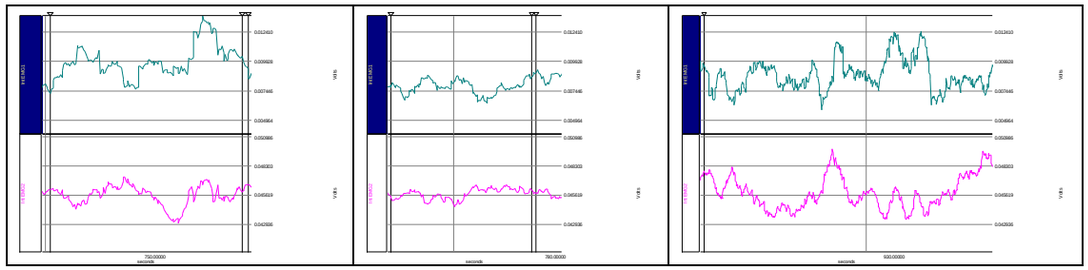


Figure 18 (continued)

Subject 5



Subject 6



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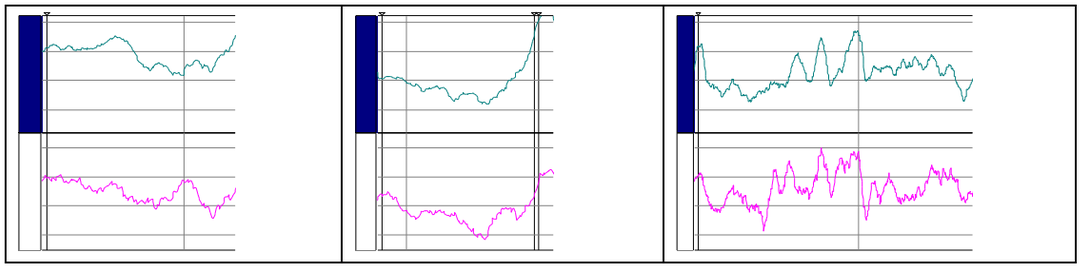
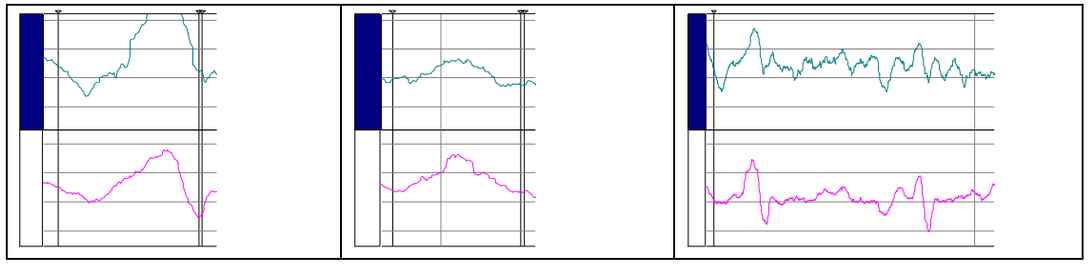


Subject 8



Subject 9

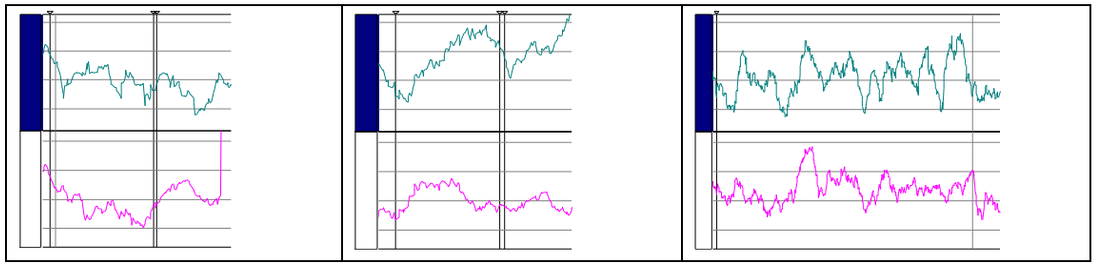
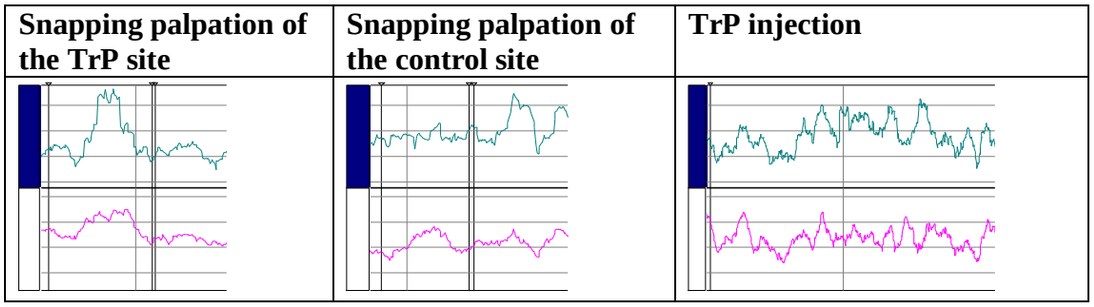
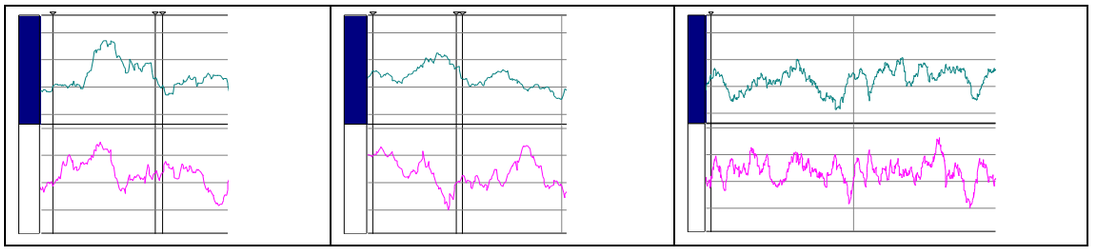


Figure 18 (continued)

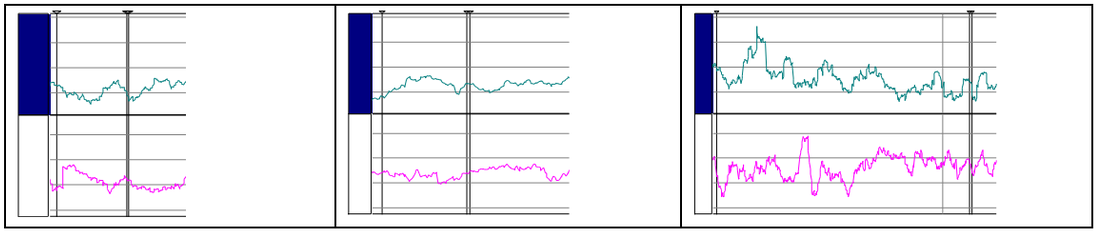
Subject 10



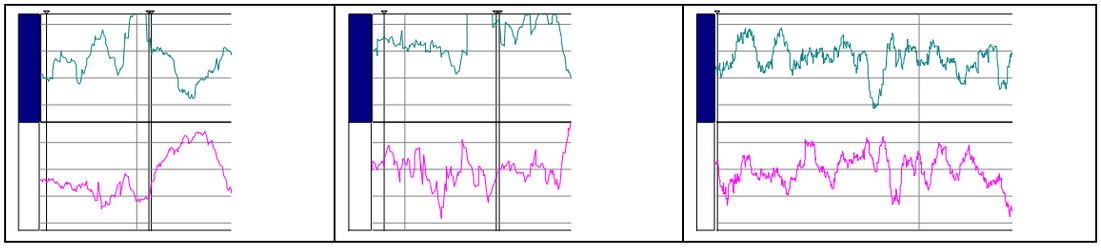
Subject 11



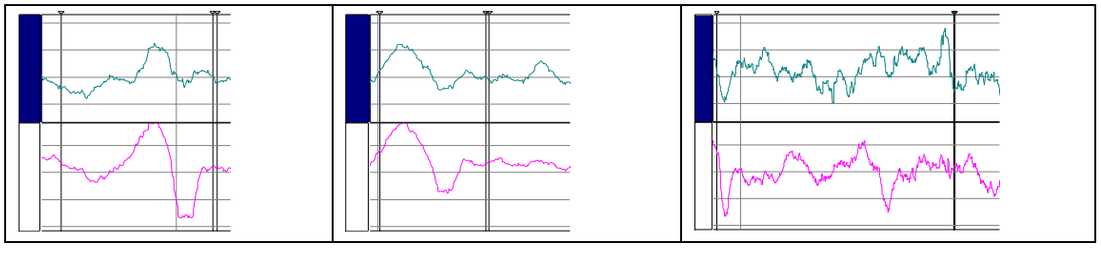
Subject 12



Subject 13



Subject 14



3.4. Presence or absence of LTR during the TrP injection based on investigator and subjects' report

LTR was observed in five (36%) of the 14 subjects (Table 7). It was visible and palpable in four of these five subjects, and was palpable, but not visible, in one subject. In seven of the nine (64%) subjects in whom the LTR was recorded as absent by the investigator, the pain reduction was more than 50% following the TrP injection (Table 8). Seven of these nine subjects reported referred pain during the TrP injection (Table 9). Six of the seven subjects who reported referred pain when the investigator did not observe or palpate the LTR had greater than 50% reduction in pain intensity following the TrP injection (Table 7). Of the seven subjects in whom the investigator did not observe LTR but the subject reported referred pain, six had greater than 50% reduction in pain intensity.

Nine (64%) subjects reported that they felt the LTR during the trigger point injection (Table 7). There was poor agreement between the investigator and the subjects' assessment of the presence or absence of LTR (Table 10). More subjects reported the presence of LTR (64%) than the investigator (36%) (Table 10). Eight out of ten subjects (80%) who reported referred pain had more than 50% reduction in pain intensity following the TrP injection (Table 8). Six out of nine subjects (67%) who reported a LTR had >50% decrease in pain intensity following the TrP injection (Table 8). Of the subjects who reported referred pain (ten subjects), only half of them reported that the LTR was present (and vice versa) (Table 10). None of the subjects reported both the absence of the LTR and referred pain (Table 10).

During the TrP injection, the sEMG tracings of the TrP site (sEMG 1) and the control site (sEMG 2) appeared similar in subjects 2, 3, 4, 5, 7, 8, 9, 10, 11, 12, 13 and 14. However, the LTR was recorded by the investigator as clinically visible and palpable in subjects 3, 7, 8, and 10, and subjects 2, 3, 5, 8, 9, 10, and 14 recorded that they felt the LTR. Hence, in subjects 3, 8 and 10, in whom both the investigator and subject agreed that an LTR was present during the TrP injection, the sEMG tracing did not show a corresponding burst of activity at the electrode around the TrP (sEMG 1) relative to the control electrode (sEMG 2).

Table 8. Relationship between the investigator and subjects' report of presence '+' or absence '-' of LTR and subjects' report on perceived referred pain, and pain reduction following TrP injection.

	≥50% ↓ in pain intensity	**<50% ↓ in pain intensity**
Investigator LTR +	3 (21%)	2 (14%)
Investigator LTR -	7 (50%)	2 (14%)
Subject LTR +	6 (43%)	3 (21%)
Subject LTR -	4 (29%)	1 (7%)
Subject referred pain +	8 (57%)	2 (14%)
Subject referred pain -	2 (14%)	2 (14%)

Table 9. Comparison of subjects' perception of referred pain with investigator's record of the LTR.

		Subject		
		Referred pain +	**Referred pain -**	**Total**
Investigator	**LTR +**	3 (21%)	2 (14%)	5 (36%)
	LTR -	7 (50%)	2 (14%)	9 (64%)
Total		10 (71%)	4 (29%)	14 (100%)

Table 10. Investigator and subjects' observation of the presence '+' or absence '-' of the LTR, and subjects' perception of referred pain during the TrP injection.

	Subject	
	LTR +	**LTR -**
Investigator LTR +	4 (29%)	1 (7%)
Investigator LTR -	5 (36%)	4 (29%)
Subject referred pain +	5 (36%)	5 (36%)
Subject referred pain -	4 (29%)	0 (0%)

3.5. Subject's evaluation of the presence or absence of referred pain during the TrP injection

Ten (71%) subjects indicated that they felt referred pain during the TrP injection. Based on these subjects' pain diagrams, in nine of the ten subjects (except subject 11), the referred pain regions felt during the TrP injection coincided with the referred pain regions felt during the investigator's palpation of the TrP (see Figure 19). Table 8 shows that eight out of ten subjects who experienced referred pain during the TrP injection reported a greater than 50% reduction in pain intensity following the injection.

Figure 19. Pain diagrams of subjects 1 to 14.

(a) subject indicates her area of pain at initial evaluation

(b) area of referred pain, if any, indicated by the subject during palpation of the TrP

(c) area of referred pain, if any, indicated by the subject during the TrP injection.

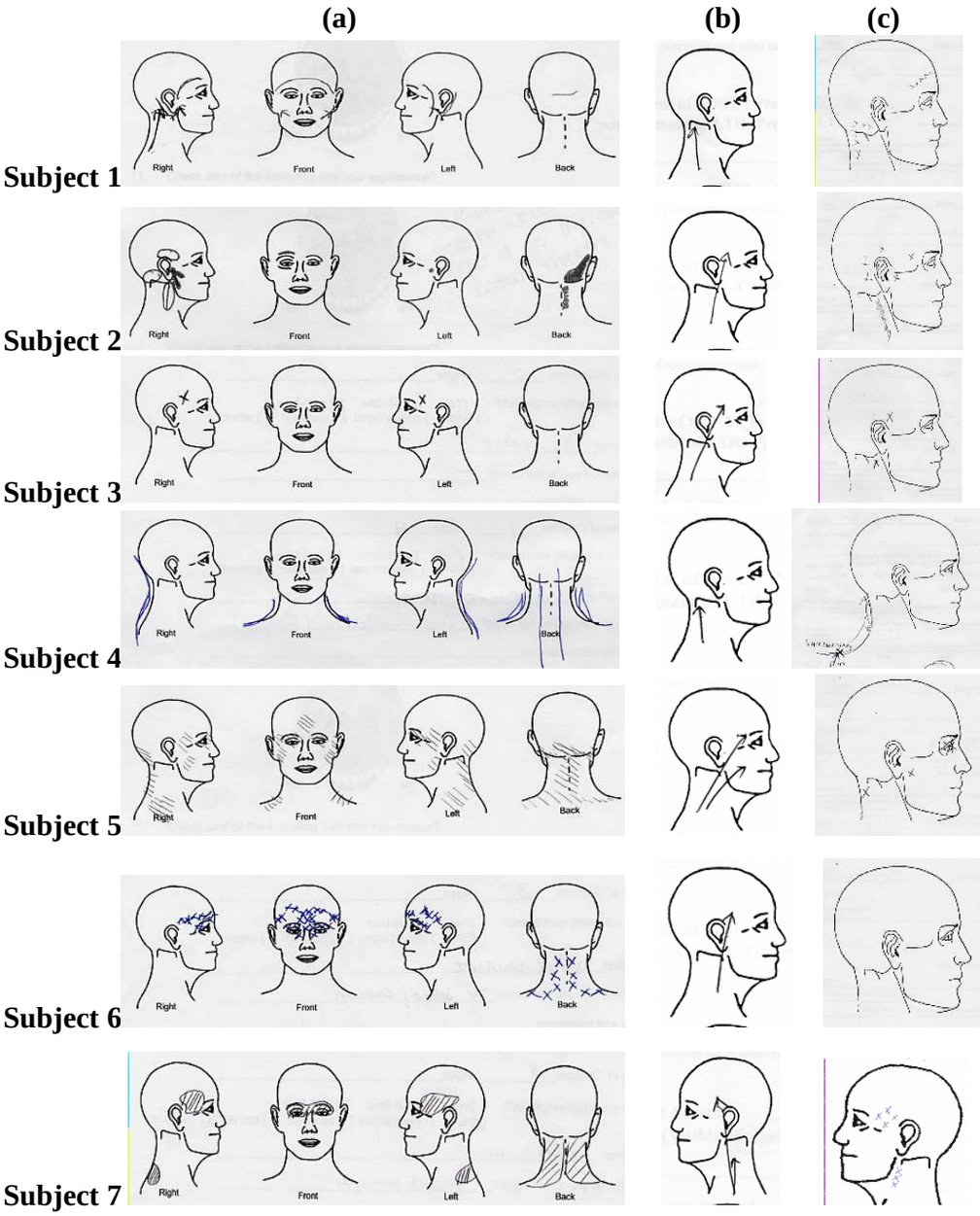
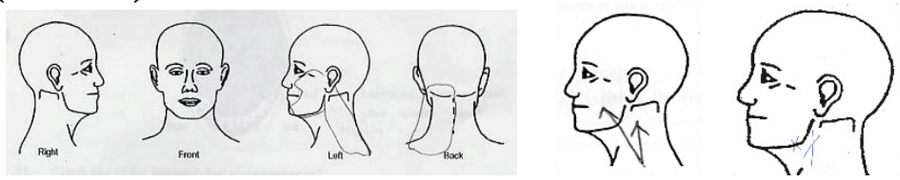
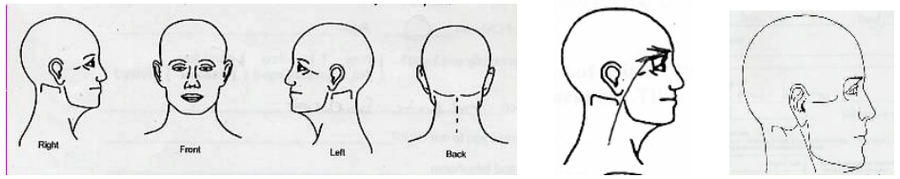


Figure 19 (continued)

Subject 8



Subject 9



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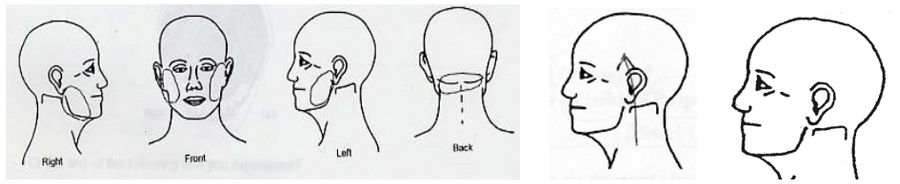

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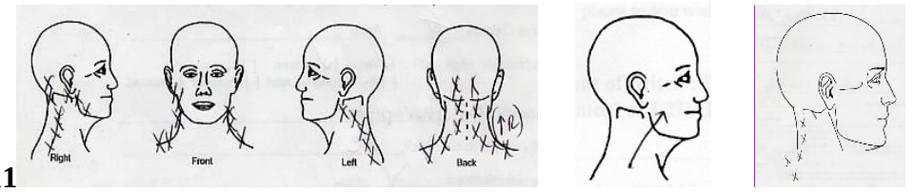
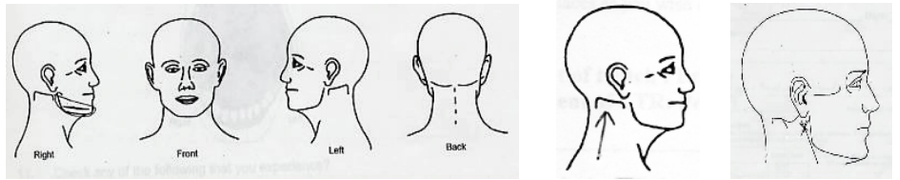
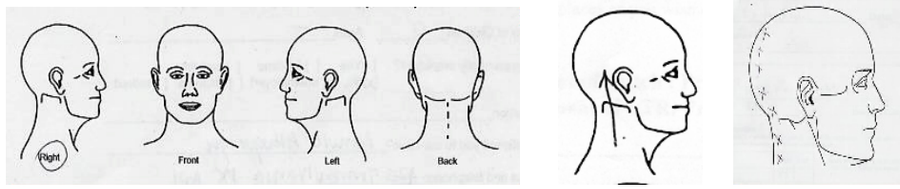


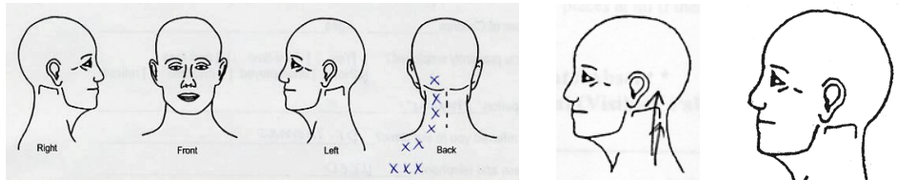
Subject 12



Subject 13



Subject 14



3.6. Final pain intensity

An immediate pain reduction of ≥50% was considered a positive response [94]. The mean final pain intensity was 2/10 (s.d. = 1.75), ranging between 0 – 5/10. Three (21%) subjects reported that they had no pain. Ten (71%) subjects reported ≥50% reduction in their pain. Four (29%) subjects had <50% reduction of their pain (44%, 38%, 25%, and 0%). Of the 14 subjects in this study only one (7%) subject reported no change in her pain. There was a statistically significant reduction in pain intensity (mean decrease = 3.57/10; s.d. = 1.74) (*t*-test; $p<0.001$) after the trigger point injection.

3.7. Complications or adverse reactions

No subject reported any complications or adverse reactions.

4. Discussion

4.1 Study design

This study was similar in design to several nEMG studies investigating the electrophysiological characteristics of myofascial TrPs [31] [32] [30] [35] [34] [36]. In these studies, a needle electrode was inserted into the active TrP and a second needle electrode (control) was inserted into the same muscle, but outside the taut band. In our study, one set of sEMG electrodes was placed around the TrP (sEMG 1) and another set placed around a control site on the same muscle but 2cm away from the taut band (sEMG 2). This is the first study, to date, which compared muscle activity at a TrP versus a control site on the same muscle using sEMG.

4.2. sEMG recording

During the five second voluntary contraction of the trapezius muscle, the mean sEMG activity at the control site (sEMG 2) showed a 3-fold increase, while that at the TrP site (sEMG 1) showed a 7.5-fold increase, compared to the respective mean baseline resting activity. This is in agreement with Donaldson et al [95] who, using dynamic sEMG techniques, found that muscles with TrPs generated more electrical activity than those without TrPs when performing the same movement.

Comparing the baseline resting muscle activity to the final resting muscle activity, only the control site (sEMG 2) exhibited a statistically significant decrease in the muscle activity ($p = 0.008$) (muscle activity at the TrP site decreased slightly, but did not reach a level of statistical significance). We speculate that this could be due to small numbers of subjects in our study (n=14). The decrease in activity agrees with Hendler et al [76] who reported a statistically significant decrease in trapezius muscle activity measured using sEMG following a TrP injection in the same muscle. Carlson et al [49] reported reduction in resting sEMG activity in masseter muscles (site of pain) after TrP injection in the trapezius muscle (source of pain). Perhaps this decrease in activity is a result of reduction in pain intensity. But Graff-Radford et al [96] investigated the effect of Transcutaneous Electrical Nerve Stimulation (TENS) in myofascial TrPs, and found that pain reduction was not associated with changes in TrP sensitivity measured by algometry. Also, in our study, subjects reporting higher initial pain intensity had statistically significantly lower mean sEMG activity at the TrP site, but not at the control site.

Snapping palpation of the TrP site resulted in a statistically significant increase in mean muscle activity from the baseline resting activity (p=0.013) at the TrP site, but not at the control site. These findings agree with Fricton et al [26] who, using nEMG in 16 subjects, found significantly higher muscle activity ($p \leq 0.001$) in the TrP than in normal muscle during snapping palpation of the TrP. They found no significant increase in nEMG activity during palpation of the control site. In addition, in our study, snapping palpation of the control site did not result in any statistically significant change in muscle activity both at the TrP (sEMG 1) and the control sites (sEMG 2).

No trend was observed in the sEMG 1 or sEMG 2 tracings during snapping palpation of the TrP and control sites, and during the TrP injection. This study is in agreement with previous studies that found that the LTR produced by snapping palpation [85] [27] and needling [27] could not be recorded by sEMG. However, the sample sizes in these studies were very small and these studies did not include a control sEMG electrode on the same muscle. We felt that an increase or burst of activity at the TrP site, but not the control site, would be representative of the LTR occurring at the TrP site. In this study, however no such differences were found. In fact, the LTR was recorded as clinically present by investigator and subject, and appeared to be positive on sEMG in only 1 subject (subject 6). In subjects 3, 8 and 10, in whom both the investigator and subject agreed that an LTR was present during the TrP injection, the sEMG tracing did not show a corresponding burst of activity at the electrode around the TrP (sEMG 1) relative to the control electrode (sEMG 2). Perhaps the nature of the twitch response is so small that sEMG is unable to register its occurrence. Our sEMG recording was accurate in recording muscle activity at rest, during tension and contraction, and yet unable to record this minute muscle response.

4.3 TrP injection and the LTR

Although the number of LTRs elicited during each TrP injection was not recorded in this study, LTRs observed by the investigator numbered no more than 1-2 twitches when they occurred. Chen et al [35] recorded a mean of 30.2 ± 4.7 LTRs elicited during rapid needling, and a mean of 8.4 ± 1.4 LTRs elicited during slow needle insertion into rabbit TrSs. Irnich et al [47] investigated the effect of dry needling in myofascial TrPs and upon needle insertion, manipulated the needle until at least one LTR was elicited. In Hong's study [17] investigating the effect of lidocaine injection (n=35) versus dry needling (n=23), needling was continued until no more LTR were elicited after 10-20 needle insertions. In subjects with the LTR, 5-30 LTRs were reportedly elicited from 20-60 needle insertions into each TrP. In cases where no LTR was elicited during the first injection or dry needling, further needling was carried on in an attempt to elicit LTRs "for ethical considerations" because Hong believed that failure to elicit the LTR would compromise the

treatment outcome. In our study, during the TrP injection, the investigator reported LTR in only 36% of the subjects, while 64% of the subjects reported that they felt the LTR, and the sEMG recorded only one LTR in one subject. The investigator was blinded to the number of LTRs perceived by each subject and the number of needle insertions was not counted. Subtle differences in the needling technique might account for the low numbers of LTRs elicited in our study compared with the high numbers of LTRs elicited in Hong's study despite the fact that both studies used the fast-in-fast-out technique described by Simons et al [2]. For example, the number of LTRs elicited may be influenced by the speed, force and number of needle penetration in and out of the TrP. The duration of needling might also be an important variable. Discrepancy between the LTRs reported by the investigator and that experienced by the subjects could be a result of some subjects misinterpreting the initial needle penetration or the needling as the LTR. Other subjects could be unfamiliar with what a LTR felt like or were eager to report a LTR simply because they were participating in a study that investigated the LTR and wanted to please the investigator with an affirmative response.

Hong [17] observed that there was little or no *immediate* treatment effect if no LTR was elicited (n=17). It would be interesting to look at the pain levels in those subjects (where LTR was absent) 2 weeks later. However, these data were not collected in the study. The results of our study shows that despite the low percentage of LTRs elicited clinically (36%), a large number of subjects (71%) reported more than 50% immediate reduction in pain intensity after the TrP injection. These data are in direct conflict with the literature reporting the importance of eliciting the LTR during TrP injections in reducing myofascial pain. Further research and longitudinal data are needed to establish whether or not a LTR is indeed a requirement for successful trigger point injections.

4.4. Referred pain

In this study, the area of referred pain on palpation of the TrP in the upper trapezius muscle was consistent with that described by Travell and Rinzler [97] and Wright [98]. Wright [98] investigated the referred pain pattern in 230 TMD patients and found that the trapezius muscle most commonly referred to the frontal, periorbital and occipital regions. Fricton et al [99] added that 63% of the subjects with jaw pain (n=104) had an active TrP in the trapezius muscle that referred pain to their jaw. They also found that TrPs in the trapezius muscle could refer pain to the teeth. Twenty-nine percent of the subjects in our study had jaw pain, but none reported referred pain to their teeth.

During the TrP injection, 10 subjects reported referred pain. Eight of them had more than 50% reduction in pain intensity following the TrP injection. In our study, self-reported referred pain was most predictive of immediate

reduction in pain intensity. The high success rate (≥50% reduction in pain intensity) in this cohort was a surprise considering that in our experience in clinical practice, patients usually have more than one active TrP responsible for the referred pain. We suspect this to be true for some of the subjects in this cohort.

4.5. Immediate pain reduction following the TrP injection

There was a statistically significant ($p<0.001$) reduction in reported mean pain intensity after the trigger point injection. Several factors could account for this pain reduction. Firstly, post injection hemostasis might be a form of acupressure. Garvey et al [46] reported that 66% of the subjects (n=16) who were randomized to a 10 second ethyl chloride spray followed by 20 seconds of acupressure on their lower back trigger points reported reduction in their lower back pain, compared to only 40% of the subjects (n=13) who received trigger point injections. Hou et al [100] also reported that ischemic compression of TrPs using either 90s of lower pressure or 30s of high pressure resulted in immediate pain relief. Secondly, resting for 5 minutes before and 5 minutes after the TrP injection, with the subjects' teeth apart and jaw relaxed, helped to promote muscle relaxation and the relaxation may have resulted in reduction of muscle pain. Thirdly, subjects may have felt a release from apprehension from having completed the injection as suggested by Carlson et al [49]. Fourthly, the pain reduction could be a placebo response. There was no control group in this study. Cummings and White [55] in a systematic review of 23 papers on needling therapies in myofascial pain, did not find any evidence that needling therapies have an effect beyond placebo. They concluded that "no technique is better than any other" and recommended that the method safest and most comfortable for the patient be used. Fifthly, regression to the mean [101] could play a role in the pain reduction. Subjects in this study presented with moderate initial pain intensity (mean of 5.6/10). Scicchitano et al [81] suggested that treatment is more likely to have an appreciable impact on the patient if the pain is severe enough for a clear reduction in pain to be experienced at the time of treatment.

Finally, psychological and other factors can affect treatment outcome. Scicchitano et al [81] studied the factors associated with excellent (n=23), partial (n=19) and poor (n=8) immediate response following TrP injections. They found that subjects reporting partial improvement had moderate-severe pain, acknowledged more stressors expressed as significantly lower scores on Denial scale of the Illness Behaviour Questionnaire (IBQ), and had greater difficulty expressing their feelings (significantly higher scores on Affective Inhibition scale of the IBQ), especially negative emotions, than the other two groups. They also reported that the greater the difficulty patients experienced in expressing their feelings, especially negative ones, the more likely their response to TrP injection would be short-

lived. Thus, patients who strongly denied current life stresses and had a tendency to focus on physical illness responded poorly to the TrP injections. Hopwood and Abram [78] investigated the factors associated with the failure of TrP injections in 193 subjects over a time period of at least 2 weeks. They found that an increased risk of treatment failure was associated with unemployment due to pain at the start of treatment, no relief from analgesics, constant pain, high levels of pain, prolonged pain, change in social activity, and lower levels of coping ability. On the contrary, Esenyel et al [50] reported that the presence of depression (in 22.9% of their subjects) or anxiety (in 89.3% of their subjects) did not limit the effect of physical modalities or TrP injection on pain reduction at 2 weeks and 3 months post treatment. These studies suggest that the patient's attitude towards his illness may have a greater influence on treatment outcome rather than the presence of psychopathology.

4.6. Limitations of this study

Several limitations deserve mention in this study. Firstly, this cohort consisted of only females. This was partly related to the ease of recruitment of females since there were more females than males seen in our clinic population. Secondly, this study sample was very small (n=14). Still, the fact that the sEMG could only record one out of five clinically observed and nine subjectively reported LTRs (during TrP injection) showed that the instrument has very poor ability to detect this muscle response. Thirdly, during the TrP injection, it is possible that the increase in activity at the TrP site resulted from the application of pressure by the investigator's fingers when trapping the taut band. To investigate this possibility, the same injection technique needs to be administered at the control site to determine if there was a similar increase in muscle activity at the control site. And in this case, to avoid bias, the investigator should be blinded to the location of the TrP and the control sites. Fourthly, this study involved only one investigator. Simons [12] stated that much skill was required to elicit the LTR by snapping palpation and needling. It is possible that the number of LTRs elicited may be different if the TrP injection was carried out by different investigators. Elicitation of the LTR is technique sensitive and inter-investigator reliability for detecting the LTR is low whether or not the examiner is trained [24 6 23]. This may explain the low numbers of LTRs elicited in this study compared with Hong's study [17]. Finally, subjects with other active TrP referring to the site of pain complaint were not excluded. The four subjects who reported less than 50% reduction in pain intensity following the TrP injection might have had sources of pain from other active TrPs or cervical structures (e.g. intervertebral discs [102]). The latter is possible because although subjects with cervical spine injury or surgery were excluded, we did not conduct a cervical spine examination to rule out objective findings of cervical spine disorders. Nevertheless, this study was designed to characterize the LTR and not to measure treatment outcome.

4.7. Recommendations

Future researchers should take into consideration the limitations raised in the discussion segment of this report (see *section 4.7. Limitations of this study*) in order to improve on study design for investigating the LTR. The results of this study need to be confirmed in a larger number of subjects. Our study suggests that sEMG is unable to capture the LTR. Yet objective measurement or record of the LTR is important in order to avoid observer bias. For now, nEMG might be the instrument of choice when it comes to studying the LTR phenomena. Further investigations into the techniques of eliciting the LTR (such as whether the rapidity of needling influence the ability to elicit LTRs), the factors which determine its presence, magnitude and frequency of occurrence, and its presence in asymptomatic muscles will contribute to the understanding of this unique muscle response. The therapeutic role of the LTR also remains to be determined.

5. Conclusion

This paper reported the preliminary findings on the inability of sEMG to register the LTR elicited by snapping palpation and TrP injection in a series of 14 female subjects with myofascial pain.

During the TrP injection, the investigator reported LTR in only 36% of the subjects, while 64% of the subjects reported that they felt the LTR, and the sEMG possibly only recorded one LTR in one subject. Despite the low percentage of LTRs elicited clinically (36%), a large number of subjects (71%) reported more than 50% immediate reduction in pain intensity after the TrP injection. Further research is needed to investigate the therapeutic effect of the LTR.

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Vita

Personal Data

Last Name	Lim
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Place of Birth	Singapore

Educational Background

Undergraduate

Dates	1991-1995
University	National University of Singapore
Degree	Bachelor of Dental Surgery (B.D.S)

Postgraduate

Dates	July 2001 – June 2002
University	University of Kentucky, USA
Course	International Fellowship Program in Orofacial Pain Management

Dates	July 2002 – June 2004
University	University of Kentucky, USA
Course	Certificate in Orofacial Pain

Scholastic and professional honors

1. Wrigley Company Book Prize 1992
   For best student in Biochemistry in the First B.D.S. Professional Examination
2. First Asia-Pacific Dental Student Research Competition 1993. First Prize Winner
   Research title : Replication of Surface Detail by Alginate Impression Material -- Two Techniques Compared
   Advisor : Alastair Stokes, BDS, MSc
   Team : Pei Feng Lim, Kok Hwee Neo, Ling Sitoh, Kai Loon Yeo
   Research presented at 17th Asia Pacific Dental Congress (Manila) 1994

Professional Positions held

1. Dental Officer, Ministry of Health, Singapore (1996--1998)
2. Associate Dental Surgeon, First Impressions Dental Surgery Private Limited (1998-2001)
3. Resident at the University of Kentucky Orofacial Pain Center (2001-2004)

Membership of Professional Bodies

1. Singapore Dental Association
2. International Association for the Study of Pain (IASP)
3. Guild of Dental Graduates (Singapore)
4. American Academy of Orofacial Pain
5. Diplomat of the American Board of Orofacial Pain (May 2004)

Lectures/Presentations

1. University of Kentucky, College of Dentistry, Grand Rounds, 24 October 2003
   Title: Is there a relationship between whiplash and temporomandibular disorders?
2. 16th Annual Orofacial Pain Alumni Symposium, 23 April 2004
   Title: I was rear-ended…… and got TMD.

Publications

1. PF Lim; KH Neo; L Sitoh; KL Yeo; A Stokes, BDS, MSc
   Adaptation of Finger-Smoothed Irreversible Hydrocolloid to Impression Surfaces
   Int J Prosthodont 1995;8:117-121

2. PF Lim, BDS, JP Okeson, DMD
   Prevalence of Thyroid Disorders in Orofacial Pain Patients.
   Abstract presented at the American Academy of Orofacial Pain 29th Scientific Meeting on Orofacial Pain and Temporomandibular Disorders. 25th – 28th March 2004. San Francisco, Ca.

Research Interests: Orofacial Pain

1. Endocrine disorders and orofacial pain.
2. Whiplash-Associated Disorders
3 Myofascial pain
4. Gene therapy
5. Psychoneuroimmunology – anxiety and pain